BWP Belgian Week of Pathology

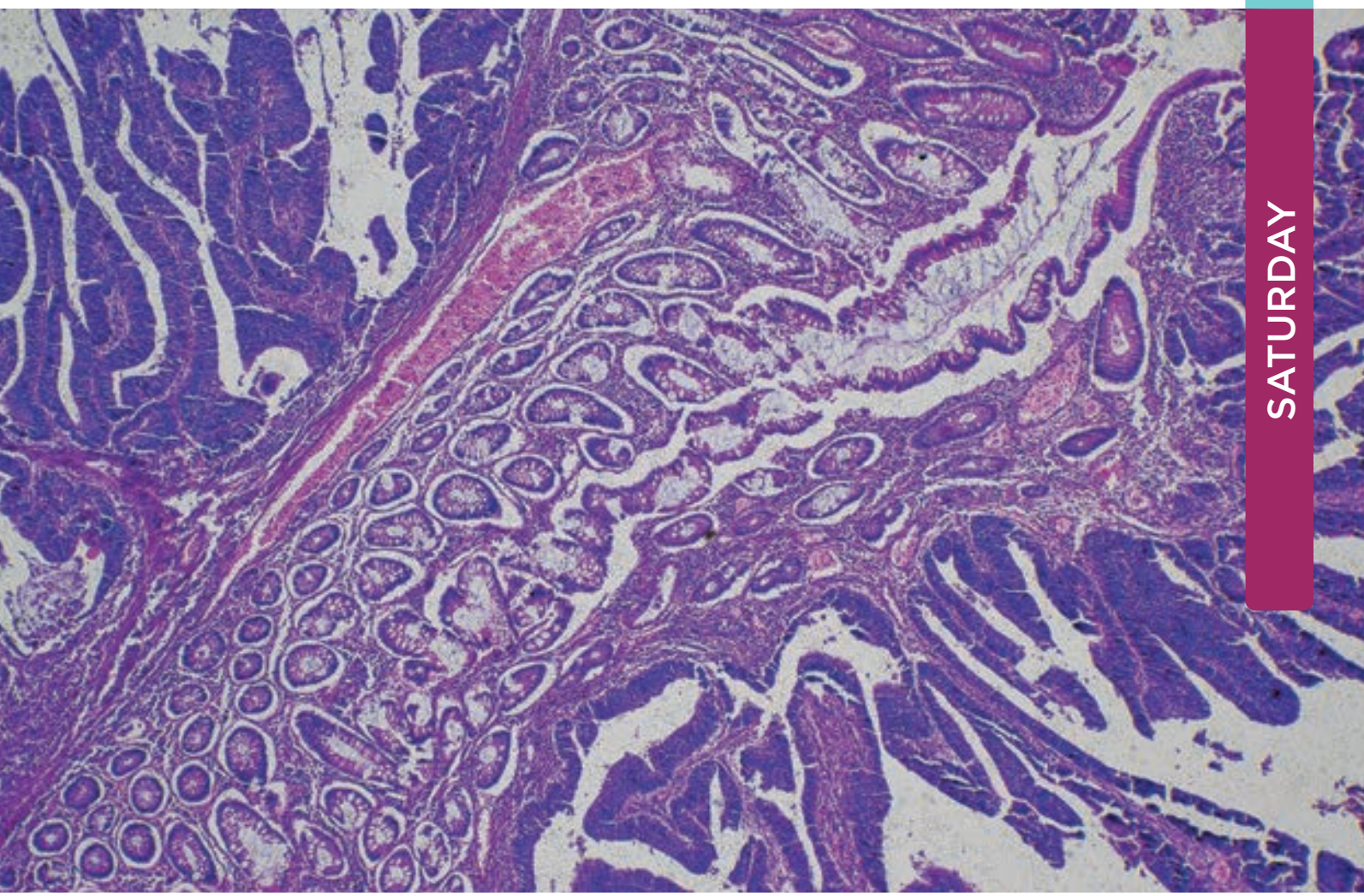
11th BELGIAN WEEK OF PATHOLOGY

22.10 > 23.10.21

@ TANGLA HOTEL

FRIDAY

SATURDAY



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WELCOME

Dear Colleagues and Friends,

The current COVID-19 crisis all over the world makes us very modest by reminding us how much we still need to learn about diseases. Small improvements in diagnostics and treatment can make a big difference. Nevertheless, we keep looking forward hopefully and on behalf of the Belgian Society of Pathology it is our pleasure to welcome you at the **11th edition of the Belgian Week of Pathology,** here in Brussels **in the Tangla Hotel**.

The **KEYNOTE** topic for this year's BWP2021 is Gastrointestinal Pathology. **Prof. dr. Fatima Carneiro, Prof. dr. Greg Lauwers** and **Prof. dr. Cord Langner** are present and they will give us state-of-the-art lectures.

In close collaboration with the different **Working Groups of the Belgian Society of Pathology** we are proud to offer you a splendid program reviewing the most recent advances of both the science underlying pathology and the targets needed for current and future clinical management. Participants will get an insight into exciting emerging novel technologies that are already being used, and others that are soon to be incorporated into different cutting edge pathological disciplines. Like every year, we encouraged **residents** to present their work by submitting abstracts, and again multiple prices will be awarded for excellent research and for presenting difficult and interesting disease entities.

And most of all, we thank our **sponsoring companies** for their continuous involvement and renewed support. Do not miss their Satellite Symposia, which have some excellent topics this year! Without their help, it would be impossible to bring together such an exceptional group of national and international experts in their fields.

This conference will provide a unique forum for pathologists, trainees and cytotechnologists to discuss our current understanding and future approaches to our most beautiful profession: **pathology**.

Enjoy BWP2021 at the Tangla in October !

Koen VAN DE VIJVER
President of the 11th Belgian Week of Pathology

GENERAL INFORMATION

Accreditation

Accreditation has been requested at the RIZIV/INAMI for anatomo-pathology and for ethics and economy.
Submission is done on the computers available in the exhibition area.
Submission is requested once a day. You will receive a confirmation e-mail after ending the procedure.
To obtain accreditation for ethics and economy, a physical signature of the participant is required (documents will be available at the entrance of the auditorium).

Language

The language of the congress is English (British spelling) for abstracts, slides and announcements.

Abstracts

Authors were invited to submit abstracts until August 31, 2021.
The result of evaluation was sent to the first authors on September 13, 2021.

- Oral presentations will be presented during the related sessions
- e-Poster presentations will take place during the morning and afternoon coffee breaks and lunch of Friday October 22 and Saturday 23.

e-Posters will be displayed during the congress on the assigned screens in the Exhibition Area.

The BWP / Belgian Society of Pathology will award:

- the Best Oral Presentation: Research (500€)
- the Best Oral Presentation: Case report (500€)
- Best e-Poster (500€).

Venue

TANGLA Hotel Brussels
5, Avenue Emmanuel Mounier
1200 Brussels

Parking available

Parking: Several possibilities during the 3 days

- the Parking of the Tangla Hotel is available, the cost per day will be 5€ per day: 120 spaces
- the Q-Parc of Hospital Saint-Luc
- along Avenue Mounier with time restriction, the Parking disc is mandatory.

Event Coordinator

DME Events
Anne-France De Meyer - 57, Av. G. Demey - 1160 Brussels - Belgium
Mobile: +32 477 27 00 45
E-mail: anne.france.de.meyer@dme-events.eu
Yuliya Sahitava
Mobile : 0492 34 48 19
E-mail : yuliya@bussol.com

Invitation

Lilly

SATELLITE SYMPOSIUM DURING THE BELGIAN WEEK OF PATHOLOGY

Friday October 22nd
13:20 - 13:50
Tangla Hotel Brussels

THE HUNT FOR RET ON OCTOBER 22nd

Program

FINDING RET FUSIONS
By Prof. Dr. Nicky D'haene, ULB Erasme

MUSTERING RET MUTATIONS
By Prof. Dr. Patrick Pauwels, UZ Antwerpen

RET ALTERATIONS ARE NOW ACTIONABLE
By Prof. Dr. Patrick Pauwels, UZ Antwerpen

Discover all the need-to-knows on RET-testing and RET-driven cancer treatment in only 30 minutes!

This meeting cannot receive accreditation from INAMI as Eli Lilly Benelux is not recognized by the comité paritaire of INAMI as a training organization.
This meeting is meant only for individuals allowed by law to prescribe or deliver medicines.
Please note that your personal data will only be used for this event and according to the EU general data protection regulation (GDPR).

October 22th 2021

INVITATION

satellite symposium

BIOMARKER TESTING IN NON-SMALL CELL LUNG CANCER:

A FAST PACED AND EVOLVING FIELD FOR MOLECULAR PATHOLOGY

This satellite symposium will be conducted during the 11th Belgian Week of Pathology at the Tangla Hotel in Brussels

Date: 22-10-2021

Room: ROYAL 1

15:10 – 15:40

The evolving biomarker testing landscape in NSCLC

dr. Glenn Broeckx

Moderator:

Prof. dr. Patrick Pauwels

NOVARTIS | Reimagining Medicine

Responsible editor : nv Novartis Pharma sa - Medialaan 40 - 1800 Vilvoorde - BE2109299277- 20/09/2021

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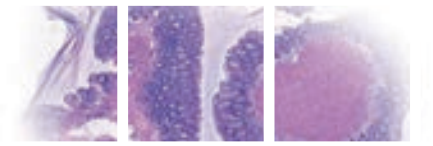
FRIDAY

SATURDAY



FRIDAY

SATURDAY

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www.belgian-society-pathology.eu

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Vasiliki SIOZOPOULOU

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Foreign faculty

BATTISTELLA Maxime	Paris, France	**JURMEISTER Philipp**	Munich, Germany
CARNEIRO Fatima	Porto, Portuga	**LANGNER Cord**	Graz, Austria
DEN BAKKER Michael A.	Rotterdam, The Netherlands	**LAUWERS Greg**	Tampa, Florida, USA
		OSZWALD André	Vienna, Austria
FLORQUIN Sandrine	Amsterdam, The Netherlands	**QUINN Cecily**	Dublin, Ireland
		SAVIC-PRINCE Spasenija	Basel, Switzerland
GOMES PINTO Daniel	Lisbon, Portugal	**TZANKOV Alexandar**	Basel, Switzerland

Belgian faculty

BALDEWIJNS Marcella	UZ Leuven	**KOLIVRAS Athanasios**	ULB Saint-Pierre
BEUSELINCK Benoit	UZ Leuven	**SAHEBALI Shaira**	UZ Brussels
BOSISIO Francesca	KU Leuven	**SASS Ursula**	ULB Saint-Pierre
BROECKX Glenn	UZ Antwerpen	**SCHOKKAERT Erik**	KULeuven
DENDOOVEN Amélie	UZ Gent	**SIOZOPOULOU Vasiliki**	UZ Antwerpen
DEOLET Ellen	UZ Gent	**TJALMA Wiebren**	UZ Antwerpen
DE SCHEPPER Sofie	UZ Gent	**VAN BEMPT Isabelle**	UZ Leuven
DESMEDT Christine	UZ Leuven	**VAN ROMPUY Anne - Sophie**	UZ Leuven
FONTANGES Quitterie	ULB Erasme	**VAN DER LEDEN Anneke**	ZNA Antwerpen

ROYAL 2 & 3 ROYAL 1

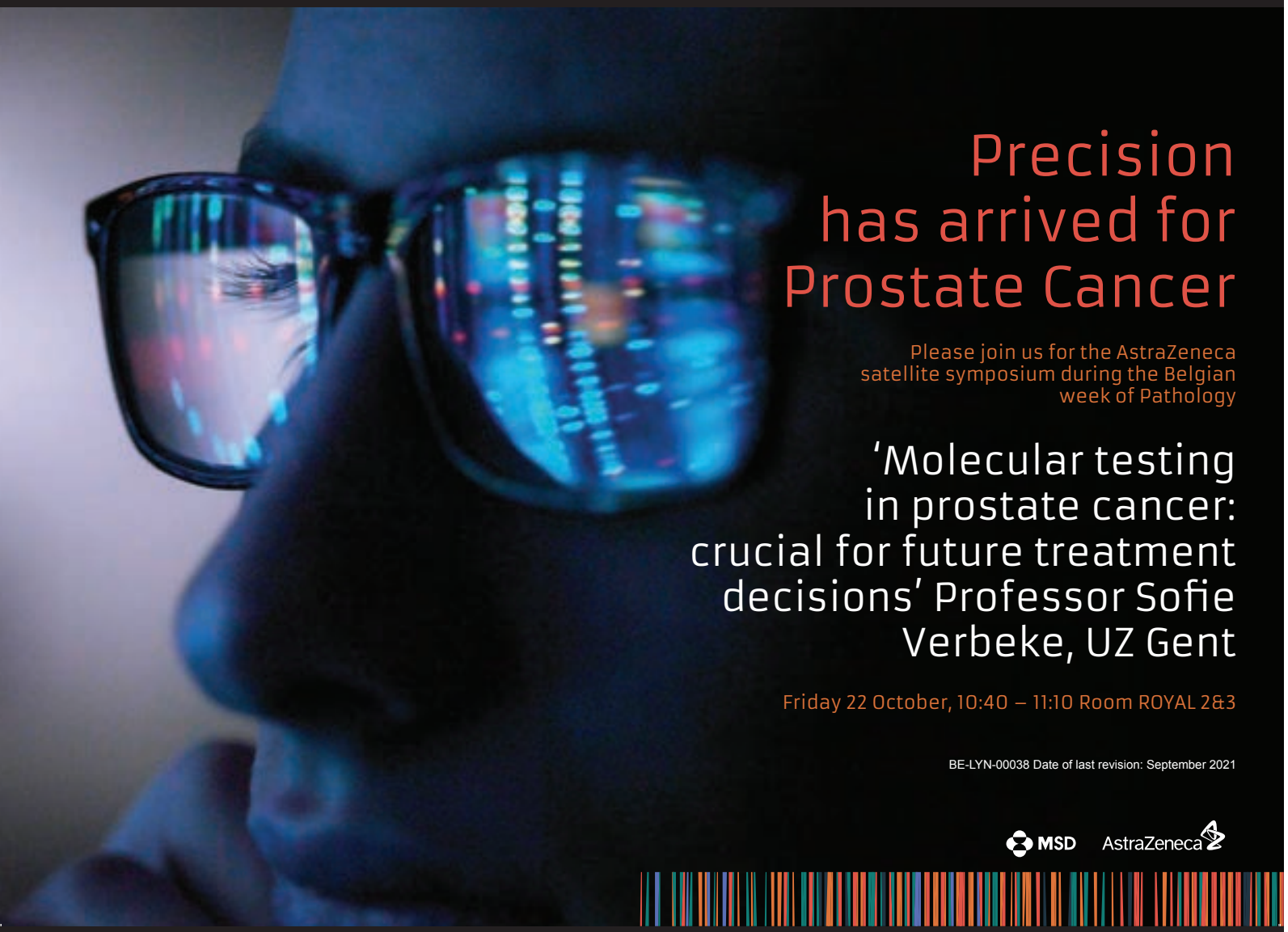
FRIDAY 22/10

08.00-09.00	WELCOME
09.00-10.30	Surgical Pathology Lung Pathology
10.30-11.15	COFFEE BREAK + POSTERS
10.40-11.10	SATELLITE SYMPOSIUM: **ASTRAZENECA**
11.15-12.45	Surgical Pathology Gynecological Pathology
12.45-14.00	LUNCH + POSTERS
13.20-13.50	SATELLITE SYMPOSIUM: **ELI LILLY**
14.00-15.00	EDUCATIONAL GRANT SYMPOSIUM: HEALTH CARE INNOVATIONS
15.00-15.45	COFFEE BREAK + POSTERS
15.10-15.40	SATELLITE SYMPOSIUM: **NOVARTIS**
15.45-17.45	Molecular Pathology Urological pathology
18.00-19.00	KEYNOTE lecture: Gastrointestinal Pathology
19.00-23.00	COCKTAIL RECEPTION AND DINNER AT THE TANGLA HOTEL

ROYAL 2 & 3 ROYAL 1

SATURDAY 23/10

08.00-09.00	WELCOME
09.00-10.30	Cytopathology Breast Pathology
10.30-11.15	COFFEE BREAK + POSTERS
10.40-11.10	SATELLITE SYMPOSIUM: **AIFORIA TECHNOLOGIES OY**
11.15-12.45	Cytopathology Dermatopathology
12.45-13.15	Gastrointestinal Pathology
13.15-14.30	LUNCH + POSTERS
13.45-14.05	GENERAL ASSEMBLY BELGIAN SOCIETY OF PATHOLOGY
14.30-16.00	Gastrointestinal Pathology
16.00-16.15	AWARDS CEREMONY + CLOSING OF THE CONGRESS



Precision has arrived for Prostate Cancer

Please join us for the AstraZeneca satellite symposium during the Belgian week of Pathology

'Molecular testing in prostate cancer: crucial for future treatment decisions' Professor Sofie Verbeke, UZ Gent

Friday 22 October, 10:40 – 11:10 Room ROYAL 2&3

BE-LYN-00038 Date of last revision: September 2021

MSD AstraZeneca

FRIDAY October 22 - Morning

FRIDAY

08.00-09.00 **WELCOME**

09.00-10.30

ROYAL 2&3

SURGICAL PATHOLOGY

Moderator: Philippe Delvenne (Liège)

- 09.00 • How to handle, process and interpret a renal biopsy
  DENDOOVEN Amélie (Ghent)
- 09.30 • Tubulo-interstitial pathology
  FLORQUIN Sandrine (Amsterdam, the Netherlands)
- 10.00 • Glomerulonephritis: the basics, with a focus on urgent diseases
  FLORQUIN Sandrine (Amsterdam, the Netherlands)

ROYAL 1

LUNG PATHOLOGY

Moderator: Karl Dhaene (Aalst)

- 09.00 • Interstitial lung disease, how (not) to diagnose these!
  DEN BAKKER Michael (Rotterdam, the Netherlands)
- 09.30 • Predictive immunocytochemistry in NSCLC
  SAVIC-PRINCE Spasenija (Basel, Switserland)
- 10.00 • *What do in situ observations on autopsy tissues tell us about the patho-mechanisms of lethal COVID-19?*
  TZANKOV Alexandar (Basel, Switserland)

10.30-11.15 **COFEE BREAK & POSTERS TOUR**

10.40-11.10 SATELLITE SYMPOSIUM BY **ASTRAZENECA (ROYAL 2&3)**

- Molecular testing in prostate cancer: crucial for future treatment decisions
  VERBEKE Sofie (Ghent)

11.15-12.45

ROYAL 2&3

SURGICAL PATHOLOGY

Moderator: Philippe Delvenne (Liège)

- 11.15 • Practical session: Applied nephropathology for the general pathologist: what not to miss in autopsy and biopsy/nephrectomy specimens, including COVID 19
  FLORQUIN Sandrine (Amsterdam,the Netherlands), DENDOOVEN Amélie (Ghent)
- 12.30 • Oral Presentation (by trainee): Pathological features of COVID-19 disease stages: a minimal invasive autopsy cohort study
  KEULEN L. (Antwerp)

ROYAL 1

GYNAECOLOGICAL PATHOLOGY

Moderators: Jean-Christophe Noël (Brussels), Cecile Colpaert (Leuven)

- 11.15 • Slide Seminar – Case presentations
  (VAN DER LEDEN A., VAN ROMPUY A-S., DEOLET E., FONTANGES Q., BROECKX G.)
- 12.30 • Oral Presentation (by trainee): Two cases of inflammatory myofibroblastic tumour
  REGINSTER M. (Gosselies)

FRIDAY October 22 - 19.00-23.00

COCKTAIL RECEPTION AND DINNER AT TANGLA HOTEL **(ROYAL 1)**

TANGLA
BRUSSELS

12.30-14.00 **LUNCH & POSTERS**

13.20-13.50 **SATELLITE SYMPOSIUM BY ELI LILLY (ROYAL 2&3)**

- The Hunt for RET on October 22nd
  Finding RET Fusions — *D'HAENE Nicky (Brussels)*
  Mustering RET Mutations — *PAUWELS Patrick (Antwerp)*
  RET Alterations are now actionable — *PAUWELS Patrick (Antwerp)*

14.00-15.00 **EDUCATIONAL GRANT SYMPOSIUM: (ROYAL 2&3)**

- Health Care Innovations: patient empowerment, scientific skepticism, and reimbursement decisions
  SCHOKKAERT Erik (Leuven)

15.00-15.45 **COFFEE BREAK & POSTERS**

15.10-15.40 **SATELLITE SYMPOSIUM BY NOVARTIS (ROYAL 2&3)**

- The evolving biomarker testing landscape in non-small cell lung cancer
  BROECKX Glenn (Antwerp)

15.45-17.45

ROYAL 2&3

MOLECULAR PATHOLOGY + ORAL PRESENTATIONS

Moderators: Nicky D'Haene (Brussels), Patrick Pauwels (Antwerp)

15.45 • Methylation-classifier in lung (and other) cancer diagnostics
JURMEISTER Philipp (Munich, Germany)

16.30 • RNA sequencing: when and why?
VAN BEMPT Isabelle (Leuven)

17.00 • MSI and Immunotherapy
BROECKX Glenn (Antwerp)

17.30 • Oral Presentation (by trainee): Correlation of Trop-2 expression with clinicopathological characteristics and outcome in TNBC
ICZI H. (Leuven)

ROYAL 1

UROLOGY PATHOLOGY

Moderators: Stephanie Verschuere (Roeselare), Lola Martinez (Gosselies)

15.45 • Mimics of conventional RCC subtypes
BALDEWIJNS Marcella (Leuven)

16.20 • Eosinophilic lesions in the kidney: benign versus malignant
OSZWALD Andre (Vienna, Austria)

16.55 • Therapeutic implications of histological subtyping and expression profiles in RCC
BEUSELINCK Benoit (Leuven)

17.30 • Oral Presentation (by trainee): Clinical and genomic indolence in lung-recurrent metastatic hormone-sensitive prostate cancer
VAN DER EECKEN K. (Ghent)

18.00-19.00 **KEYNOTE LECTURE: GASTROINTESTINAL PATHOLOGY (ROYAL 2&3)**

Moderator: Anne Hoorens (Ghent)

- Hereditary gastric cancer
  CARNEIRO Fatima (Porto, Portugal)

19.00-23.00 **Cocktail Reception and Dinner at Tangla Hotel (ROYAL 1)**

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During the coffee break session:
Saturday at 10.40am in room Royal 1.

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SATURDAY October 23 - Morning

08.00-09.00 **WELCOME**

09.00-10.30

ROYAL 2&3

CYTOPATHOLOGY

Moderator: Shaira Sahebali (Brussels)

09.00 • Colposcopy in HPV positive lesions
TJALMA Wiebren (Antwerp)

09.45 • New international system for reporting Serous fluid cytopathology:
PINTO Daniel Gomes (Lisbon, Portugal)

ROYAL 1

BREAST PATHOLOGY

Moderator: Guiseppe Floris (Leuven)

09.00 • UPTIDER: the UZ/KU Leuven Post-mortem Tissue Donation Program
DESMEDT Christine (Leuven)

09.45 • Apocrine lesions of the breast.
QUINN Cecily (Dublin, Ireland)

10.30-11.15 **COFFEE BREAK & POSTERS TOUR**

10.40-11.10 SATELLITE SYMPOSIUM **AIFORIA TECHNOLOGIES OY (ROYAL 1)**

• Introducing an AI-diagnostic pathology workflow.

11.15-12.45

ROYAL 2&3

CYTOPATHOLOGY

Moderator: Shaira Sahebali (Brussels)

11.15 • HPV+/NILM in cervical screening
SAHEBALIShaira (Brussels)

11.35 • Slide seminar – case presentations

12.30 • Oral Presentation (by trainee): Implementation of automatic quantification of Ki-67 in well-differentiated gastro-entero-pancreatic neuroendocrine tumors: towards standardized evaluation into daily practice
LIFRAGNE F. (Liège-Brussels)

ROYAL 1

DERMATOPATHOLOGY

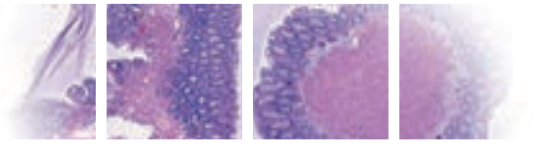
Moderator: Vasiliki Siozopoulou (Antwerp)

11.15 • Viral infections in dermatopathology
BATTISTELLA Maxime (Paris, France)

11.55 • Case presentations: Virus induced pathology of the skin
(by A. Kolivras, U. Sass, S. De Schepper, F. Bosisio, V. Siozopoulou)

12.30 • Oral Presentation (by trainee): Aleukemic leukemia cutis: a rare pathology challenging to diagnose
NOEL F. (Brussels)

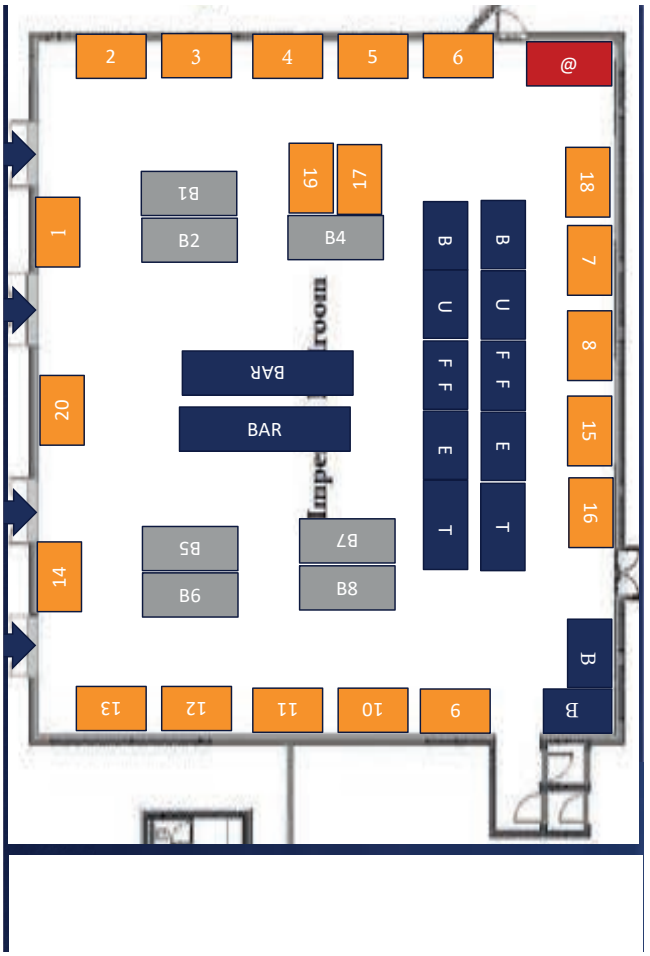
SATURDAY



EXHIBITION FLOOR

FRIDAY

SATURDAY



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SATURDAY October 23 - Afternoon

12.45-13.15 **ROYAL 2&3**

GASTROINTESTINAL PATHOLOGY

Moderator: Anne Hoorens (Ghent)

12.45 • **Premalignant and malignant lesions of the stomach**
CARNEIRO Fatima (Porto, Portugal)

13.15-14.30 **LUNCH & POSTERS**

13.45-14.05 GENERAL ASSEMBLY **BELGIAN SOCIETY OF PATHOLOGY (ROYAL 1)**

14.30-16.00 **ROYAL 2&3**

GASTROINTESTINAL PATHOLOGY

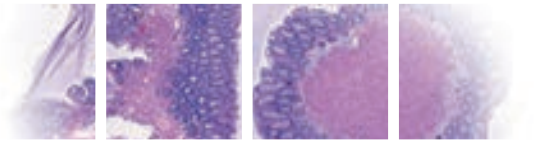
Moderator: Anne Hoorens (Ghent)

14.30 • **Recent advances in the diagnosis of dysplasia in IBD**
LAUWERS Greg (Tampa, Florida, USA)

15.15 • **Colorectal serrated lesions and polyps: distinguishing challenging cases and diagnosing dysplasia**
LANGNER Cord (Graz, Austria)

16.00-16.15 **AWARDS CEREMONY**
CLOSING CEREMONY BWP

BWP Belgian Week of Pathology



SAVE THE DATE

CONGRESS

12TH EDITION

BWP Belgian Week of Pathology

BELGIAN WEEK OF PATHOLOGY

21.10 > 22.10.22

VENUE:
@ TANGLA HOTEL

KEY NOTE TOPIC:
Urological Pathology

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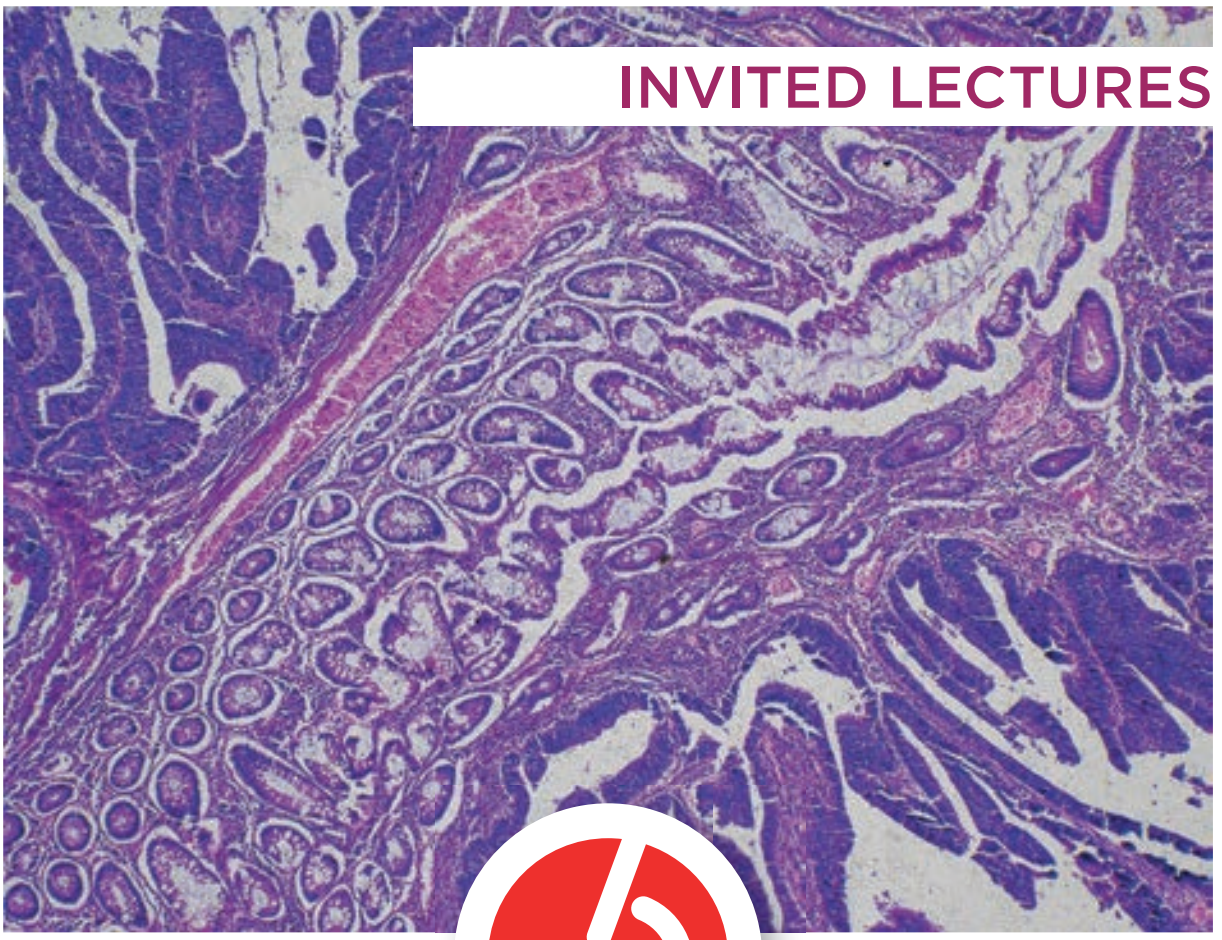


INVITED LECTURES

INDEX - Friday 22/10

UROLOGY PATHOLOGY

Moderators: Stephanie Verschuere (Roeselare), Lola Martinez (Gosselies)

Mimics of conventional RCC subtypes

BALDEWIJNS Marcella (Leuven)

Clear cell RCC (ccRCC) is the most common form of adult renal cancer, which accounts for over 60% of adult renal carcinomas and for most of the associated deaths. Of the most common RCC subtypes, ccRCC is an independent predictor of adverse outcome, with a significantly worse cancer-specific outcome than the other conventional subtypes such as papillary and chromophobe RCC.

The majority of ccRCC typically harbor alterations of the VHL gene, either in the form of mutation or promoter methylation followed by a "second hit" typically occuring as a large deletion that may include the majority of the p arm of chromosome 3. The latter serves as a potential diagnostic marker, as it can be detected by FISH or other copy number assessment techniques.

Recently, understanding of the genetics of clear cell renal cell carcinoma has been further enhanced by genomic analysis showing additional mutations in chromatin remodeling genes PBRM1, SETD2, and BRCA1-associated protein 1 (BAP1) contributing to the pathogenesis in some ccRCC cases. PBRM1 and BAP1 mutations are mutually exclusive. SETD2 often occur alone or together with PBRM1. BAP1 mutation is typically associated with high grade tumors and poor outcome.

The majority of ccRCC are sporadic, however, a subset occurs in the setting of von Hippel Lindau (VHL) syndrome and rare cancer predisposition syndromes, including the recently described BAP1 tumor predisposition syndrome.

In the last decades, some other tumor entities have been described with clear cell cytoplasm and showing morphological overlap with ccRCC. These entities include clear cell papillary RCC and Mit Family translocation RCC next to some novel entities such as RCC with leiomyomatous stroma and ELOC (formerly TCEB1) mutated RCC. However, these tumors do not share the same genetic aberrations and should be distinguished from the classical VHL-mutated ccRCC.

A correct classification of these tumors is relevant from a clinical prognostic point of view since some of these tumors have a more indolent behavior than conventional ccRCC.

UROLOGY PATHOLOGY

Moderators: Stephanie Verschuere (Roeselare), Lola Martinez (Gosselies)

Therapeutic implications of histological subtyping and expression profiles in RCC

BEUSELINCK Benoit (Leuven)

Renal cell carcinomas are currently treated with VEGFR-TKIs, immune checkpoint inhibitors and mTOR inhibitors, sequentially or in combination.

The use of this therapies depends on reimbursement conditions and histological subtype and features such as sarcomatoid dedifferentiation. During the last years, more molecular data have been published. Although these molecular data are not currently used in daily practice, they gave us a better insight into tumor heterogeneity within clear cell renal cell carcinomas.

GYNAECOLOGICAL PATHOLOGY

Modertors: Jean-Christophe Noël (Brussels), Cecile Colpaert (Leuven)

BCOR-associated endometrial stromal sarcoma

BROECKX Glenn (UZ Antwerpen)

Endometrial stromal sarcoma (ESS) is a less common malignant mesenchymal tumour of the uterus. According to the World Health Organization classification of tumours, ESS is categorized, based on morphology, clinical behaviour and genetic alterations, into low-grade ESS (LG-ESS), high-grade ESS (HG-ESS) and undifferentiated uterine sarcoma (UUS). The latter being an exclusion diagnosis.

Whereas 50% of the LG-ESS have a t(7;17) chromosomal translocation, resulting in gene fusions JAZF1-SUZ12, JAZF1-PHF1, EPC1-PHF1, MEAF6-PHF1 or MBTD1-CXorf67, USS at the other side of the spectrum has no defining genetic hallmarks. In 2014, the World Health Organization classification of tumours reintroduced HG-ESS based on the discovery of the translocation t(10;17) in these tumours with fusions in the YWHAE and NUTM2 genes. Since then, many researchers reported cases of HG-ESS lacking a YWHAE fusion. These cases usually harboured rearrangements or internal tandem duplications (ITD) in the BCOR gene, leading to inclusion in the most recent update on the World Health Organization classification of tumours (2020).

In this slide seminar, a few case of BCOR associated uterine lesions are presented, followed by a brief literature review on pathogenesis, histopathology, prognosis and therapy.

MOLECULAR PATHOLOGY

Modertors: Jean-Christophe Noël (Brussels), Cecile Colpaert (Leuven)

Microsatellite instability and immunotherapy

BROECKX Glenn (UZ Antwerpen)

Microsatellites are sequences of repetitive, (usually) non-coding DNA. Due to their repetitive nature, slips might occur, causing mismatch errors. Microsatellite instability (MSI) refers to variability in lengths of microsatellite foci due to the inability to repair these mismatches. In normal circumstances, the mismatch repair (MMR) enzymes recognize and initiate correction of mismatch errors. The major MMR proteins are MLH1, PMS2, MSH2 and MSH6. Inactivation of one of these enzymes might occur by a germ line mutation (Lynch syndrome), a sporadic mutation and epigenetic silencing by hypermethylation of the promotor region. The latter is most often seen in the MLH1 gene.

Testing for MSI is preferably done according to the ESMO guidelines (2019). Due to a good correlation between immunohistochemistry and the presence of MSI, testing for retained expression of the MMR enzymes on immunohistochemistry (IHC) is sufficient and should be the first test of choice. Quality measurements should be taken into consideration for correct evaluation. Furthermore, a 4 MMR panel is preferred over a 2 MMR panel. PCR testing is only mandatory in case of doubt of IHC. Next generation sequencing can be considered, but should only be executed in selected centres, devoted to the complex analysis of MSI on these molecular tests.

The prognostic value of MSI is widely known, but recent trials have also shown a positive predictive value towards immune checkpoint inhibitors. MSI causes frameshift mutations, leading to an increased translation of frameshift peptides. These peptides are presented on the surface of the tumour cells by MHC class I molecules. These neoantigens are recognized by cytotoxic T lymphocytes, initiating immune response. Tumour cells can evade this response by expressing PD-L1 or other immune checkpoints. This immune evasion can be targeted by immune checkpoint inhibitors.

LUNG PATHOLOGY

Moderator: Karl Dhaene (Aalst)

Interstitial lung disease, how (not) to diagnose these!

DEN BAKKER Michael (Rotterdam, the Netherlands)

Interstitial lung disease without a further qualifier is an imprecise term which encompasses a very large group of diseases with overlapping clinical, radiological and histopathological features. Moreover, interstitial lung diseases with a different etiology may share considerable histological and radiological overlap. From these simple statements alone it is apparent that a final diagnosis of an interstitial lung disease cannot usually be made based on a single diagnostic modality and that a multimodal approach should be adopted.

When the group of interstitial lung disease is narrowed down to those in which a definite etiology and pathogenesis has not been determined the prefix 'idiopathic' is added. This immediately reduces the number of entities but similarly requires correlation of clinical, radiological and in some cases histological features.

LUNG PATHOLOGY

Moderator: Karl Dhaene (Aalst)

Predictive immunocytochemistry in NSCLC

SAVIC-PRINCE Spasenija (Basel, Switzerland)

We witness a significant progress in treatment options for patients with advanced, inoperable stage non-small cell lung cancer (NSCLC), which represents one of the most common malignancies. Typically, these patients are diagnosed by small biopsies and cytology specimens and our diagnostic workup, including the subtype, the PD-L1 status and the results of targetable oncogenic driver alterations, are directly linked to the choice of a specific treatment (1).

The increasing number of treatment relevant targets is becoming a challenge and predictive immunohistochemistry (IHC) can provide time-, tissue- and cost-efficient results. Predictive IHC is well established for PD-L1, ALK and ROS1-tesing (2-3). Like for ALK, TRK is normally not expressed in adult lung tissue and has a high sensitivity and specificity as surrogate marker for NTRK1/2/3 gene rearrangements, which facilitates screening of patients for these rare alterations (4).

Cytology specimens as a source for predictive marker testing in NSCLC is very important as up to 40% of all lung cancers are diagnosed by cytology alone. Despite the established role of cytology in lung cancer diagnosis no commercial immunocytochemistry (ICC) assays were validated for cytology specimens, neither for cell blocks nor for conventional cytology.

Cell blocks (CB) are the most straightforward cytology preparation for immunochemistry, but not always available. Predictive ICC on cell blocks is valid using histology standardized protocols for formalin-fixed and paraffin-embedded (FFPE) specimens. For non-cell block cytology, most commonly performed on PAP-stained cytology specimens, ICC protocols need to be adjusted as the pre-analytic processing is significantly different from FFPE specimens. Predictive ICC on non-cell block cytology works well, but is less standardized, requires rigorous protocol optimization, validation and quality control (5-6).

(1) NCCN guidelines version 5.2021
(2) IASLC Atlas of PD-L1 Testing in Lung Cancer, 2017 (free pdf download)
(3) IASLC Atlas of ALK and ROS1 testing in lung cancer, 2016 (free pdf download)
(4) Solomon SP et al., Mod Pathol., 2020; PMID: 31375766
(5) Jain D et al., Cancer Cytopathol., 2019; PMID: 31050216
(6) Savic Prince S et al., Virchows Arch., 2019; PMID: 30173280

Educational Grant Symposium

Health Care Innovations: patient empowerment, scientific skepticism, and reimbursement decisions?

SCHOKKAERT Erik (Leuven)

I will focus on innovation and the use of information in health care. I will first discuss the affordability issue raised by the increase in health care expenditures and argue that (a) this increase is mainly explained by innovation; and (2) poses a challenge for solidarity. Choices must be made. Citizen participation and patient empowerment are crucial elements in these developments. I will illustrate this for two specific topics: the rise of personalized medicine and the growing use of quality indicators. The connecting thread throughout my presentation will be the tensions between patient empowerment and scientific scepticism, and between solidarity and affordability.

GYNAECOLOGICAL PATHOLOGY

Moderators: Jean-Christophe Noël (Brussels), Cecile Colpaert (Leuven)

Slide Seminar – Case presentations

VAN DER LEDEN Anneke (ZNA Antwerp)

This is a case presentation of a 39 year old woman with a bilateral ovarian and endometrial carcinoma. This case illustrates changes in de new WHO 2020 in the section of seromucinous tumours of the ovary, where the diagnosis of sermucinous carcinoma is removed. These tumours are now considered to be a variant of endometrioid type carcinoma. A new comment in the WHO about synchronous endometrial and ovarian endometrioid carcinoma is also discussed.

GYNAECOLOGICAL PATHOLOGY

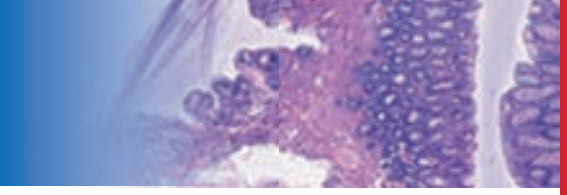
Modertors: Jean-Christophe Noël (Brussels), Cecile Colpaert (Leuven)

Slide Seminar – Case presentations

DEOLET Ellen (Ghent)

Mesonephric-like adenocarcinoma is a recently described rare neoplasm occurring in the uterine corpus and ovary. This under-recognized subtype of carcinoma can be very challenging to diagnose. In mesonephric adenocarcinoma a variety of growth patterns can be present within the same tumor, as a result of which they can be misinterpreted and diagnosed as low-grade endometrioid adenocarcinoma, clear cell carcinoma, or even serous carcinoma and carcinosarcoma. Advanced diagnostics, including improved morphologic, immunohistochemical and molecular knowledge can help develop new therapeutic strategies against this specific subtype of endometrial cancer with an aggressive clinical behavior.

NOTES



FRIDAY

SATURDAY

Educational Grant Symposium

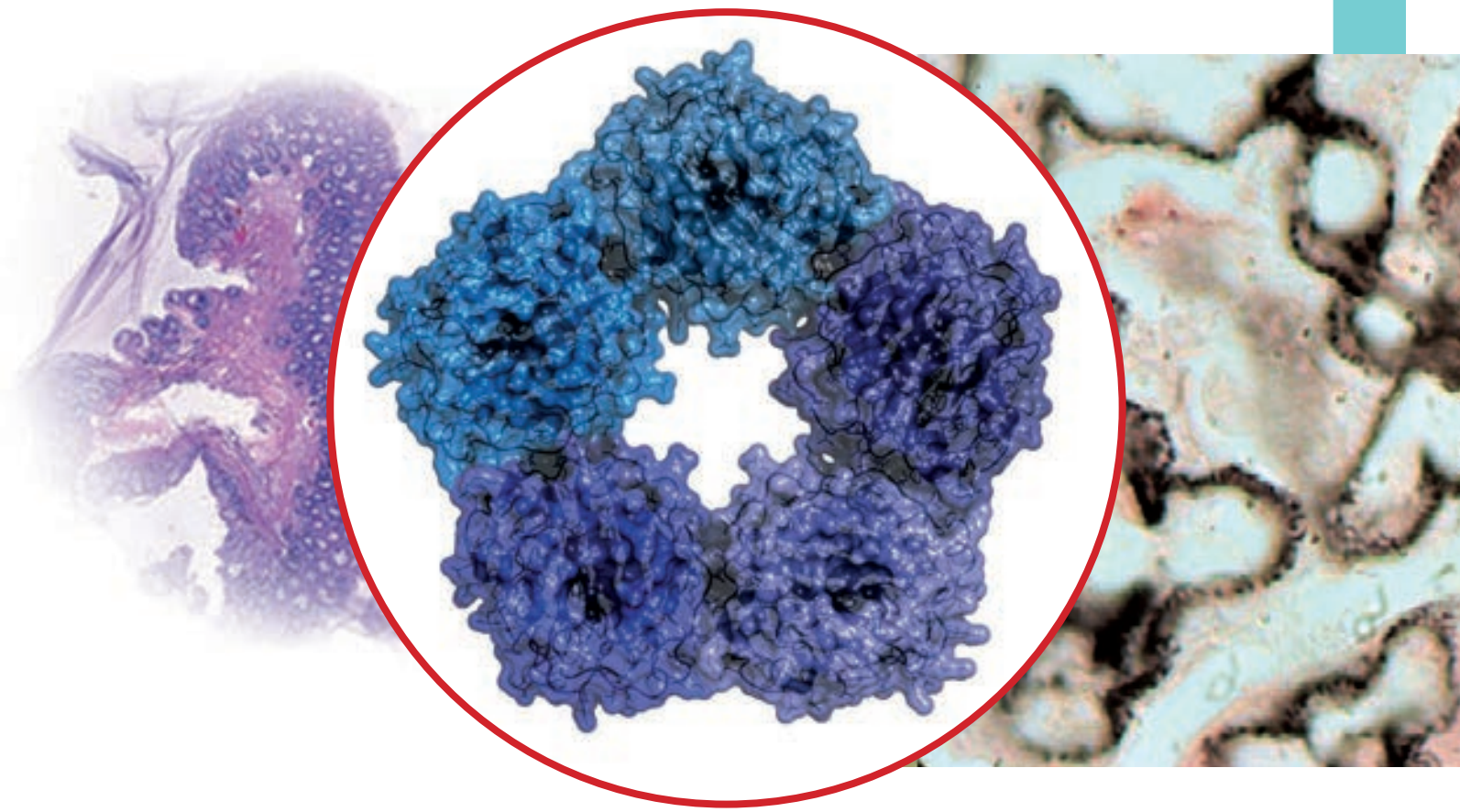
Royal 2&3

14.00-15.00

FRIDAY

Health Care Innovations: patient empowerment, scientific skepticism, and reimbursement decisions

SCHOKKAERT ERIK (Leuven)



Organised thanks to the Educational Grant

with the kind support

INDEX - Saturday 23/10

DERMATOPATHOLOGY

Moderator: Vasiliki Siozopoulou (Antwerp)

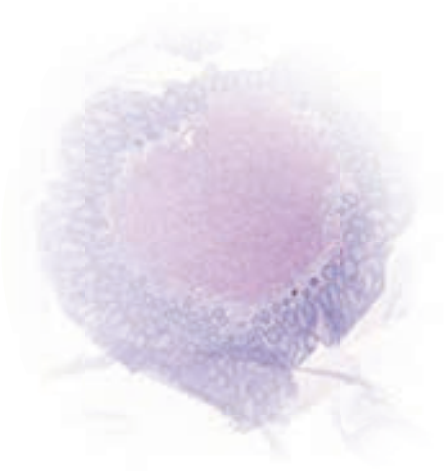
Viral infections in dermatopathology

BATTISTELLA Maxime (Paris, France)

The skin is a site of predilection for both viral infections and a constellation of virus-associated manifestations. The SARS-COV-2 pandemics has confirmed this, with a large number of dermatological-oriented publications in less than 2 years.

The pathologist/dermatopathologist is sometimes able to diagnose specifically a viral infection under the microscope thanks to peculiar viral cytopathogen effects, specific virus-induced changes, or virus detection by IHC or molecular techniques. In other situations, the pathologist will need a correlation to the clinical presentation to make a diagnosis of paraviral eruption.

We will discuss the main findings enabling specific viral infections diagnosis in the skin, pitfalls in the diagnosis of virus-related conditions, the recent entities linked to virus infections in the skin, and present the main clinico-pathological pictures in paraviral eruptions.



BREAST PATHOLOGY

Moderator: Guiseppe Floris (Leuven)

UPTIDER: the UZ/KU Leuven Postmortem Tissue Donation Program

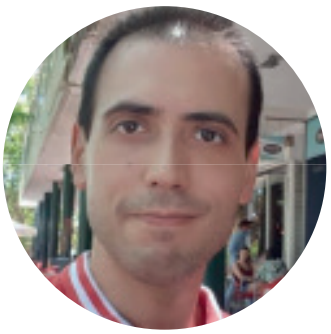
DESMEDT Christine (Leuven)

In the light of the still unfinished work of molecular characterisation of metastatic spread in breast cancer (BC), and with the underused potential of research autopsies bringing light at the horizon, we have established a unique tissue donation program at our institution called UPTIDER (UZ/KU Leuven Program for Post-mortem Tissue Donation to Enhance Research, NCT04531696). The overarching aim of the program is to unravel metastatic BC evolution, biology, heterogeneity and treatment resistance through multi-level and multiregional analysis of extensive samples rapidly taken in the post-mortem setting. Several substudies have been defined within the protocol, with a special focus on (i) rare histological subtypes, (ii) Inflammatory BC (IBC), (iii) male BC, (iv) hereditary cancer syndromes, (v) molecular heterogeneity and treatment response, (vi) the generation of new experimental models such as PDXs and/or PDOs, (vii) metabolomics, and, (viii) fluids (liquid biopsies).

CYTOPATHOLOGY

Moderator: Shaira Sahebali (Brussels)

NEW INTERNATIONAL SYSTEM FOR REPORTING SEROUS FLUID CYTOPATHOLOGY



PINTO Daniel Gomes (Lisbon, Portugal)

Effusion cytology is fundamental for patient management in the context of many diseases, both neoplastic and non-neoplastic. Effusions are easy to drain and provide ample material for diagnosis. Historically, the diagnostic efficacy of effusion cytology has not been uniform, however, with great variation between laboratories and even individual pathologists. With the aim of increasing standardization and following in the footsteps of previous classification systems, The International System for Reporting Serous Fluid Cytology (TIS) was developed by a workforce of specialists sponsored by the Academy of Cytology and American Society of Cytopathology. The system follows best international practices, the most up-to-date literature and expert consensus, consisting of five diagnostic categories: non-diagnostic (ND), negative for malignancy (NFM), atypia of undetermined significance (AUS), suspicious for malignancy (SFM) and malignant (MAL). By providing the framework for a standardized diagnostic approach, TIS enables better inter-observer agreement and well-defined risk of malignancy for each category, potentiating better patient management. In this talk we introduce and explore the new international system, its categories, clinical management and the role of ancillary diagnostic and theranostic testing in effusion samples.

BREAST PATHOLOGY

Moderator: Guiseppe Floris (Leuven)

Apocrine lesions of the breast

QUINN Cecily (Dublin, Ireland)

Apocrine change is recognised in benign, atypical and malignant lesions of the breast. Apocrine metaplasia is a frequent finding in the breast of women over the age of 25 years and is commonly seen in benign cysts with a simple or papillary configuration. Apocrine change is also recognised in other benign lesions including sclerosing adenosis, now known as apocrine adenosis, Apocrine atypia usually refers to cytological atypia in which there is at least three-fold variation in nuclear size but architectural atypia may also occur. The distinction between atypical apocrine hyperplasia and non-high grade apocrine ductal carcinoma in situ may be difficult due to the relative rarity of these entities and the lack of validated diagnostic criteria. Lobular carcinoma in situ (LCIS) with apocrine change is considered to be a variant of pleomorphic LCIS. An apocrine variant of encapsulated papillary carcinoma is also recognised. Apocrine change is described in invasive carcinoma, including no special type, lobular, micropapillary and mucinous variants. The recent WHO 2019 update recognises 'carcinoma with apocrine differentiation' as a special type breast carcinoma based on the presence of apocrine morphology in at least 90% of the tumour. Tumours with apocrine morphology are usually but not always hormone receptor negative. Human epidermal receptor type 2 (HER-2) status is variable. Molecular studies have identified breast tumours with apocrine features and high expression of androgen receptor mRNA including 'luminal androgen receptor tumours' and 'molecular apocrine tumours'. The term 'pure apocrine carcinoma' has been proposed to describe an invasive carcinoma with apocrine morphology that is oestrogen and progesterone receptor negative and androgen receptor positive. HER-2 status may be positive or negative. This talk reviews the pathology of benign, atypical and malignant apocrine lesions of the breast, with emphasis on a diagnostic approach and recent advances in our understanding of invasive apocrine carcinoma.

Discover all the need to know about *RET* testing during the **Lilly symposium** on 22/10 at 13h20

WHERE PRECISION and STRENGTH MEET

Retsevmo▼
selpercatinib

Test for *RET*[1]
Treat with Retsevmo*

Advanced *RET* Fusion-Positive NSCLC[1,2]

64%
ORR
(95% CI: 54, 73)

Previously received at least platinum-based chemotherapy (n=105)
17.5 months median DoR
16.5 months median PFS

Advanced *RET*-Mutant MTC[1,3]

69%
ORR
(95% CI: 55, 81)

Previously treated with cabozantinib and/or vandetanib (n=55)
Median DoR not reached
Median PFS not reached

Indications[1]: Retsevmo as monotherapy is indicated for the treatment of adults with:
- advanced *RET* fusion-positive non-small cell lung cancer (NSCLC) who require systemic therapy following prior treatment with immunotherapy and/or platinum-based chemotherapy.
- advanced *RET* fusion positive thyroid cancer who require systemic therapy following prior treatment with sorafenib and/or lenvatinib.

Retsevmo as monotherapy is indicated for the treatment of adults and adolescents 12 years and older with advanced *RET*-mutant medullary thyroid cancer (MTC) who require systemic therapy following prior treatment with cabozantinib and/or vandetanib.

* Retsevmo was designed to selectively target RET, the primary driver of certain RET-driven cancers[4]

Referenties: 1. RETSEVMO SmPC (current version). Available on www.fagg-afmps.be. **2.** Drilon A, et al. NEJM 2020; 383: 813-824. **3.** Wirth LJ, et al. NEJM 2020; 383: 825-835. **4.** Drilon A, et al. Nat Rev Clin Oncol. 2018; 15(3): 151-167.

▼ This medicinal product is subject to additional monitoring. This will allow quick identification of new safety information. Healthcare professionals are asked to report any suspected adverse reactions.

This material is meant only for individuals allowed by law to prescribe or deliver medicines.

METHOD OF DELIVERY: Medicinal product subject to restricted medical prescription.
Responsible publisher: ELB - Markiesstraat 1/4B Rue du Marquis - 1000 Brussels

Lilly

PP-SE-BE-0023 SEPT 2021

MINIMAL INFORMATIONS OF THE SPC ▼This medicinal product is subject to additional monitoring. This will allow quick identification of new safety information. Healthcare professionals are asked to report any suspected adverse reactions. See section 4.8 for how to report adverse reactions. **1. NAME OF THE MEDICINAL PRODUCT** Retsevmo 40 mg hard capsules Retsevmo 80 mg hard capsules **2. QUALITATIVE AND QUANTITATIVE COMPOSITION** Retsevmo 40 mg hard capsules Each hard capsule contains 40 mg selpercatinib. Retsevmo 80 mg hard capsules Each hard capsule contains 80 mg selpercatinib. For the full list of excipients, see section 6.1. **3. PHARMACEUTICAL FORM** Hard capsules. Retsevmo 40 mg hard capsules Grey opaque capsule, 6 x 18 mm (size 2), imprinted with "Lilly", "3977" and "40 mg" in black ink. Retsevmo 80 mg hard capsules Blue opaque capsule, 8 x 22 mm (size 0), imprinted with "Lilly", "2980" and "80 mg" in black ink. **4. CLINICAL PARTICULARS 4.1 Therapeutic indications** Retsevmo as monotherapy is indicated for the treatment of adults with: advanced *RET* fusion-positive non-small cell lung cancer (NSCLC) who require systemic therapy following prior treatment with immunotherapy and/or platinum-based chemotherapy advanced *RET* fusion-positive thyroid cancer who require systemic therapy following prior treatment with sorafenib and/or lenvatinib Retsevmo as monotherapy is indicated for the treatment of adults and adolescents 12 years and older with advanced *RET*-mutant medullary thyroid cancer (MTC) who require systemic therapy following prior treatment with cabozantinib and/or vandetanib. **4.2 Posology and method of administration** Retsevmo therapy should be initiated and supervised by physicians experienced in the use of anti-cancer therapies. RET testing The presence of a *RET* gene fusion (NSCLC and non-medullary thyroid cancer) or mutation (MTC) should be confirmed by a validated test prior to initiation of treatment with Retsevmo. Posology The recommended dose of Retsevmo based on body weight is: Less than 50 kg: 120 mg twice daily. 50 kg or greater: 160 mg twice daily. If a patient vomits or misses a dose, the patient should be instructed to take the next dose at its scheduled time; an additional dose should not be taken. Treatment should be continued until disease progression or unacceptable toxicity. The current selpercatinib dose should be reduced by 50% if co-administering with a strong CYP3A inhibitor. If the CYP3A inhibitor is discontinued, the selpercatinib dose should be increased (after 3-5 half-lives of the inhibitor) to the dose that was used before starting the inhibitor. *Dose adjustments* Management of some adverse reactions may require dose interruption and/or dose reduction. Retsevmo dose modifications are summarised in Table 1 and Table 2.

Table 1 Recommended dose modifications for Retsevmo for adverse reactions based on body weight

Dose modification	Adults and adolescents ≥50 Kg	Adults and adolescents <50 Kg
Starting dose	160 mg orally twice daily	120 mg orally twice daily
First dose reduction	120 mg orally twice daily	80 mg orally twice daily
Second dose reduction	80 mg orally twice daily	40 mg orally twice daily
Third dose reduction	40 mg orally twice daily	Not applicable

Table 2 Recommended dose modifications for adverse reactions

Adverse drug reaction (ADR)		Dose modification
Increased ALT or AST	Grade 3 or Grade 4	• Suspend dose until toxicity resolves to baseline (see sections 4.4 and 4.8). Resume at a dose reduced by 2 levels. If after at least 2 weeks selpercatinib is tolerated without recurrent increased ALT or AST, increase dosing by 1 dose level. If selpercatinib is tolerated without recurrence for at least 4 weeks, increase to dose taken prior to the onset of Grade 3 or 4 increased AST or ALT. Permanently discontinue selpercatinib if Grade 3 or 4 ALT or AST increases recur despite dose modifications.
Hypersensitivity	All Grades	• Suspend dose until toxicity resolves and begin corticosteroids at a dose of 1 mg/kg (see sections 4.4 and 4.8). Resume selpercatinib at 40 mg twice daily while continuing steroid treatment. Discontinue selpercatinib for recurrent hypersensitivity. If after at least 7 days, selpercatinib is tolerated without recurrent hypersensitivity, incrementally increase the selpercatinib dose by 1 dose level each week, until the dose taken prior to the onset of hypersensitivity is reached. Taper steroid dose after selpercatinib has been tolerated for at least 7 days at the final dose.
QT interval prolongation	Grade 3	• Suspend dose for QTcF intervals >500 ms until the QTcF returns to <470 ms or baseline (see section 4.4). Resume selpercatinib treatment at the next lower dose level.
	Grade 4	• Permanently discontinue selpercatinib if QT prolongation remains uncontrolled after two dose reductions or if the patient has signs or symptoms of serious arrhythmia.
Hypertension	Grade 3	• Patient blood pressure should be controlled before starting treatment. Selpercatinib should be suspended temporarily for medically significant hypertension until controlled with antihypertensive therapy. Dosing should be resumed at the next lower dose if clinically indicated (see sections 4.4 and 4.8).
	Grade 4	• Selpercatinib should be discontinued permanently if medically significant hypertension cannot be controlled.
Haemorrhagic events	Grade 3 or Grade 4	• Selpercatinib should be suspended until recovery to baseline. Discontinue selpercatinib for severe or life-threatening haemorrhagic events.
Other adverse reactions	Grade 3 or Grade 4	• Selpercatinib should be suspended until recovery to baseline. Discontinue selpercatinib for severe or life-threatening events

Special populations Elderly No dose adjustment is required based on age (see section 5.2). No overall differences were observed in the treatment emergent adverse events or effectiveness of selpercatinib between patients who were ≥65 years of age and younger patients. Limited data are available in patients ≥75 years. *Renal impairment* Dose adjustment is not necessary in patients with mild, moderate or severe renal impairment. There are no data in patients with end stage renal disease, or in patients on dialysis (section 5.2). *Hepatic impairment* Close monitoring of patients with impaired hepatic function is important. No dose adjustment is required for patients with mild (Child-Pugh class A) or moderate (Child-Pugh class B) hepatic impairment. Patients with severe (Child-Pugh class C) hepatic impairment should be dosed with 80 mg selpercatinib twice daily (section 5.2). *Paediatric population* Retsevmo should not be used in children aged less than 12 years. There is no data in children or adolescents with RET fusion-positive NSCLC or thyroid cancer. Retsevmo is intended to be used from the age of 12 years for the treatment of patients with RET-mutant MTC (see section 5.1). In RET-mutant MTC, there are very limited data available in children or adolescents aged less than 18 years. Patients should be dosed according to body weight (see section 4.2). Method of administration Retsevmo is for oral use. The capsules should be swallowed whole (patients should not open, crush, or chew the capsule before swallowing) and can be taken with or without food. Patients should take the doses at approximately the same time every day. Retsevmo must be accompanied by a meal if used concomitantly with a proton pump inhibitor (see section 4.5). Retsevmo should be administered 2 hours before or 10 hours after H_2 receptor antagonists (see section 4.5). **4.3 Contraindications** Hypersensitivity to the active substance or to any of the excipients listed in section 6.1. **4.8 Undesirable effects** Summary of the safety profile –The most common serious adverse drug reactions (ADRs) are hypertension (0.9%), increased aspartate aminotransferase (AST) (1.6%) and increased alanine aminotransferase (ALT) (1.6%). Permanent discontinuation of Retsevmo for treatment emergent adverse events, regardless of attribution occurred in 6.0% of patients. ADRs resulting in permanent discontinuation (2 or more patients) included increased ALT (0.4%), increased AST (0.3%), hypersensitivity (0.4%), and thrombocytopenia (0.3%). Tabulated list of adverse drug reactions The ADRs reported in the 746 patients treated with selpercatinib are shown in Table 3. The ADRs are classified according to MedDRA the system organ class. Frequency groups are defined by the following convention: very common (≥ 1/10); common (≥ 1/100 to < 1/10); uncommon (≥ 1/1,000 to < 1/100); rare (≥ 1/10,000 to < 1/1,000); very rare (< 1/10,000), and not known (cannot be estimated from available data). Within each frequency group, undesirable effects are presented in order of decreasing seriousness. Median time on treatment with selpercatinib was 11.07 months. Table 3 Adverse drug reactions in patients receiving single agent selpercatinib (LIBRETTO-001)

System organ class	ADR	Selpercatinib (N=746)	
		All grades toxicity (%)	Grade 3, 4 toxicity (%)
Immune system disorders[a]	*Common*		
	Hypersensitivity[c]	5.2	1.7*
Metabolism and nutrition disorders	*Very common*		
	Decreased appetite	14.1	0.1*
Nervous system disorders	*Very common*		
	Headache[c]	24	1.5*
	Dizziness[c]	14.6	0.1*
Cardiac disorders	*Very common*		
	Electrocardiogram QT prolonged[c]	18.1	4.0
Vascular disorders	*Very common*		
	Hypertension[c]	37.4	19.4
Gastrointestinal disorders	*Very common*		
	Abdominal pain[c]	25.5	1.9*
	Diarrhoea[c]	39.0	3.5*
	Nausea	23.5	0.7*
	Vomiting	16.2	0.9*
	Constipation	27.1	0.5*
	Dry Mouth[c]	40.3	0
Skin and subcutaneous tissue disorders	*Very common*		
	Rash[c]	28.7	0.7*
General disorders and administration site conditions	*Very common*		
	Pyrexia	14.3	0.1*
	Fatigue[c]	38.2	2.3*
	Oedema[c]	38.7	0.5*
Investigations[b]	*Very common*		
	ALT increased	49.5	10.6
	AST increased	55.0	9
	Platelets decreased	34.5	3.0
	LymphocytecCount decreased	46.2	16.1
	Magnesium decreased	25.6	0.5
	Creatinine increased	39.1	1.2
Blood and lymphatic system	*Very common*		
	Haemorrhage[d]	16.6	2.4

[a] Hypersensitivity reactions were characterised by a maculopapular rash often preceded by a fever with associated arthralgias/myalgias during the patient's first cycle of treatment (typically between Days 7-21). [b] Based on laboratory assessments. Only patients with baseline and at least one post-baseline result are included. [c] Consolidated terms [d] See Description of selected adverse reactions for further characterisation. *Only includes a grade 3 adverse reaction. Description of selected adverse reactions *Aminotransferase elevations (AST / ALT increased)* Based on laboratory assessment, ALT and AST elevations were reported in 49.5% and 55% patients, respectively. Grade 3 or 4 ALT or AST elevations were reported in 10.6% and 9.0% patients respectively. The median time to first onset was: AST increase 4.1 weeks (range: 0.7, 108.1), ALT increase 4.1 weeks (range: 0.9, 111.1). Dose modification is recommended for patients who develop Grade 3 or 4 ALT or AST increase (see section 4.2). *QT interval prolongation* Review of ECG data showed 6.2% of patients had >500 msec maximum post-baseline QTcF value, and 17.5% of patients had a >60 msec maximum increase from baseline in QTcF intervals. At the time of the last post-baseline measurement, increase in QTc value >60 msec was reported in 2.6% of patients. There were no reports of *Torsade de pointes*, sudden death, ventricular tachycardia, ventricular fibrillation, or ventricular flutter. No patient discontinued treatment due to QT prolongation. Retsevmo may require dose interruption or modification (see sections 4.2 and 4.4). *Hypertension* In patients receiving selpercatinib, the median maximum increase from baseline systolic pressure was 29 mm Hg (range: –11, +96). Only 13% of patients retained their baseline grade during treatment, 45% had an increasing shift of 1 grade, 32.7% of 2 grades, and 8.3% of 3 grades. Hypertension was reported in 41.9% patients with history of hypertension (26.9% with grade 3) and 34.2% of patients without history of hypertension (14.1% with grade 3, 4). Overall, a total of 19.4% displayed treatment-emergent Grade 3 hypertension (defined as maximum systolic blood pressure greater than 160 mm Hg). Diastolic blood pressure results were similar, but the increases were of lesser magnitude. No patients were permanently discontinued due to hypertension. Dose modification is recommended in patients who develop hypertension (see section 4.2). Selpercatinib should be discontinued permanently if medically significant hypertension cannot be controlled with antihypertensive therapy (see section 4.4). *Hypersensitivity* Signs and symptoms of hypersensitivity included fever, rash and arthralgias or myalgias with concurrent decreased platelets or increased aminotransferase. In study LIBRETTO-001, 24.7% (184/746) of patients treated with selpercatinib had previously received anti-PD-1/PD-L1 immunotherapy. Hypersensitivity occurred in a total of 5.2% (39/746) of patients receiving selpercatinib, including Grade 3 hypersensitivity in 1.7% (13/746) of patients. Of the 39 patients with hypersensitivity, 64.1% (25/39) had NSCLC and had received prior anti-PD-1/PD-L1 immunotherapy. Grade 3 hypersensitivity occurred in 3.8% (7/184) of the patients previously treated with anti-PD-1/PD-L1 immunotherapy. The median time to onset was 1.9 weeks (range: 0.9 week to 77 weeks): 1.7 weeks in patients with previous anti-PD-1/PD-L1 immunotherapy and 8.9 weeks in patients who were immunotherapy naïve. Retsevmo may require dose interruption or modification (see section 4.2). Haemorrhages Grade ≥3 haemorrhagic events occurred in 2.4% of patients treated with selpercatinib, including 3 (0.4%) patients with fatal haemorrhagic events, one case each of cerebral haemorrhage, tracheostomy site haemorrhage, and haemoptysis. The median time to onset was 12.8 weeks (range: 0.1 week to 124.3 weeks). Selpercatinib should be discontinued permanently in patients with severe or life-threatening haemorrhage (see section 4.2). Additional information on special populations *Paediatric patients* There were 3 patients < 18 years (range: 15-17) of age in LIBRETTO-001. The safety of selpercatinib in children aged less than 18 years has not been established. Elderly In patients receiving selpercatinib, 24.5% were ≥65-74 years of age, 8.2% were 75-84 years of age, and 1.07% ≥ 85 years of age. The frequency of serious adverse events reported was higher in patients ≥65-74 years (43.2%), 75-84 years (50.8%), and ≥85 years (62.5%), than in patients <65 years (29.8%) of age. The frequency of AE leading to discontinuation of selpercatinib was higher in patients ≥65-74 years (6.0%), 75-84 years (13.1%), and ≥85 years (12.5%), than in patients <65 years of age (3.2%). Reporting of suspected adverse reactions Reporting suspected adverse reactions after authorisation of the medicinal product is important. It allows continued monitoring of the benefit/risk balance of the medicinal product. Healthcare professionals are asked to report any suspected adverse reactions via: Belgium: Agence fédérale des médicaments et des produits de santé, Division Vigilance, Boîte Postale 97, B- 1000 Bruxelles Madou, Site internet: www.notifieruneffetindesirable.be, e-mail: adr@afmps.be. Luxembourg: Centre Régional de Pharmacovigilance de Nancy, Bâtiment de Biologie Moléculaire et de Biopathologie (BBB), CHRU de Nancy – Hôpitaux de Brabois, Rue du Morvan, 54 511 VANDOEUVRE LES NANCY CEDEX, Tél : (+33) 3 83 65 60 85 / 87, E-mail : crpv@chru-nancy.fr ou Direction de la Santé, Division de la Pharmacie et des Médicaments, 20, rue de Bitbourg, L-1273 Luxembourg-Hamm, Tél. : (+352) 2478 5592, E-mail : pharmacovigilance@ms.etat.lu. Link pour le formulaire : https://guichet.public.lu/fr/entreprises/sectoriel/sante/medecins/notification-effets-indesirables-medicaments.html **7. MARKETING AUTHORISATION HOLDER** Eli Lilly Nederland B.V. Papendorpseweg 83 3528BJ Utrecht The Netherlands **8. MARKETING AUTHORISATION NUMBER(S)** EU/1/20/1527/001 EU/1/20/1527/002 EU/1/20/1527/003 EU/1/20/1527/004 EU/1/20/1527/005 EU/1/20/1527/006 EU/1/20/1527/007 EU/1/20/1527/008 EU/1/20/1527/009 EU/1/20/1527/010 EU/1/20/1527/011 **9. DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION** Date of first authorisation: 11 February 2021 **10. DATE OF REVISION OF THE TEXT** 22 June 2021 **METHOD OF DELIVERY** Medicinal product subject to restricted medical prescription. Detailed information on this medicinal product is available on the website of the European Medicines Agency http://www.ema.europa.eu.

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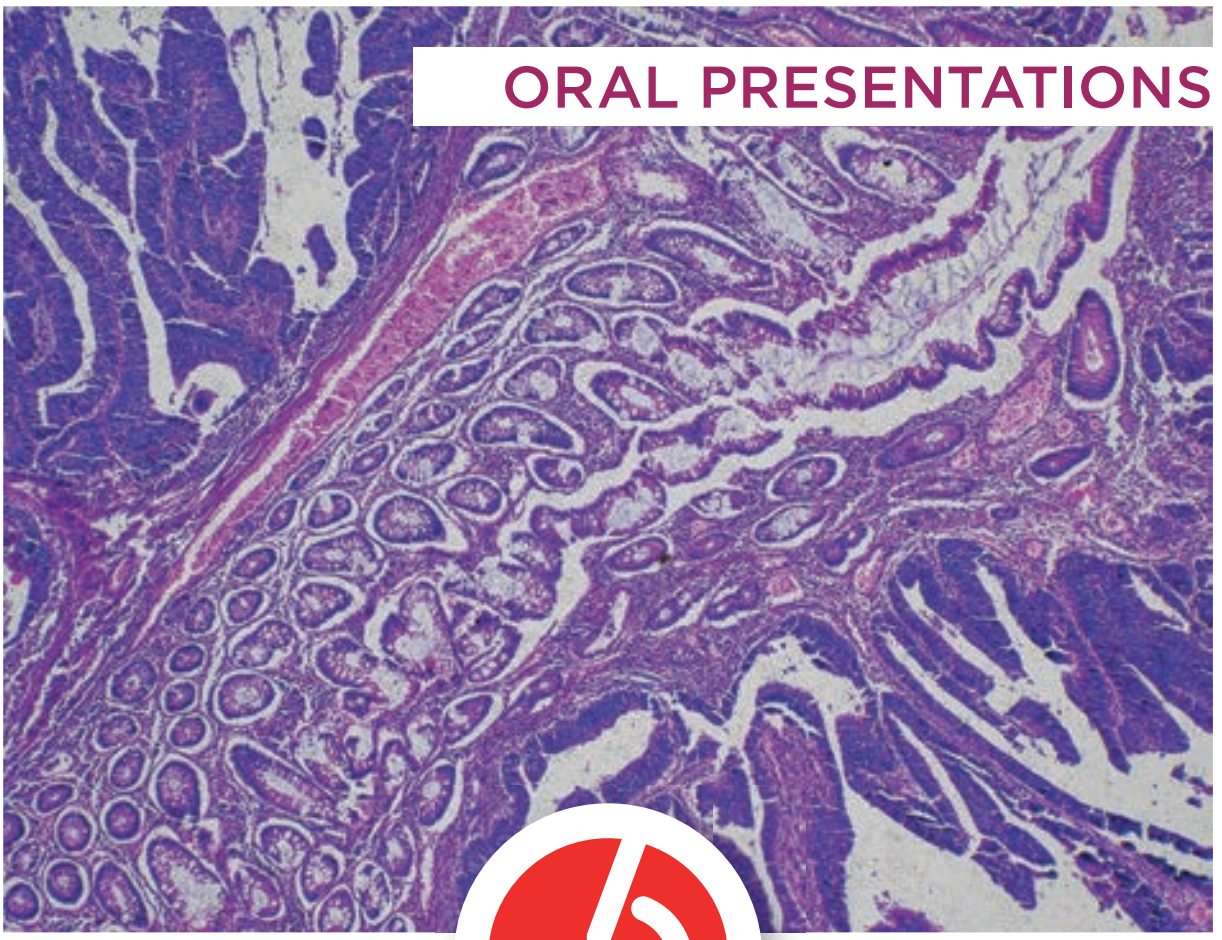

ORAL PRESENTATIONS

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O 01

PATHOLOGICAL FEATURES OF COVID-19 DISEASE STAGES: A MINIMAL INVASIVE AUTOPSY COHORT STUDY

L. Keulen[1], V. D'Onofrio[2,3], A. Vandendriessche[1], R. Achten[1,4], A. Dendooven[1,5,6], A. Driessen[1,5], J. Cox[2,3], M. Lammens[1,5]

1. Department of Pathology, Antwerp University Hospital, Antwerp, Belgium
2. Department of Immunology and Infection, Faculty of Medicine and Life Sciences, Hasselt University, Hasselt, Belgium
3. Department of Infectious Diseases and Immunity, Jessa Hospital, Hasselt, Belgium
4. Department of Pathology, Jessa Hospital, Hasselt, Belgium
5. Center for Oncological Research - CORE, University of Antwerp, Antwerp, Belgium
6. Department of Pathology, Ghent University Hospital, Ghent, Belgium

Background

The WHO classifies COVID-19 in different disease stages of which pathophysiological mechanisms differ. In this study we evaluate the histological characteristics of the disease stages.

Methods

Forty-eight COVID-19 patients, of which 44 PCR-confirmed, were included in a prospective minimal invasive autopsy cohort. Patients were classified according to WHO guidelines in mild-moderate disease, severe-critical disease and post-acute disease. Based on radiological findings needle biopsies were taken of normal lung, abnormal lung, liver, kidney , heart, spleen and abdominal fat. H&E stained slides were reviewed by 4 individual pathologists. Limited ancillary testing was performed on lung, liver and heart biopsies.

Results

The main abnormalities were found in the lungs. Diffuse alveolar damage (DAD) was absent in patients with mild-moderate disease but present in 93.8% and 87.5% of patients with severe-critical or post-acute COVID-19, respectively (p=.002). Hyaline membranes were absent in the patients with mild-moderate disease, while present in 81.3% of patients with severe-critical COVID-19 and 62.5% of patients with post-acute COVID-19 (p=0.019). Pneumocyte atypia was more prevalent in severe-critical disease (81.3%) and post-acute disease (75%) than in mild-moderate disease (25.0%). The typically described micro-thrombi were found in only 1 patient with severe-critical disease (2.3%). Vasculitis was present in 3 patients (6.8%), all suffering from severe-critical or post-acute COVID-19. Other organs with significant changes were the liver (sinusoidal dilatation) and heart (fibrosis and myocarditis).

Conclusions

Patients with mild-moderate disease had different histopathological characteristics compared to severe-critical disease, however differences between severe-critical disease and post-acute disease were limited. Minimally invasive autopsy can be a good alternative to conventional autopsies, but has its limitations due to 'sampling errors'.

O 02

TWO CASES OF INFLAMMATORY MYOFIBROBLASTIC TUMOUR

M. Reginster[1], S. Croce[2], Q. Fontanges[3], C. Koopmansch[1]

1. Institut de Pathologie et de Génétique, Gosselies, Belgium
2. Institut Bergonié, Bordeaux, France
3. Hôpital Civil Marie Curie, Lodelinsart, Belgium

Background

Inflammatory myofibroblastic tumor is a mesenchymal neoplasm of uncertain malignant potential. It has a predilection for the lung and abdominopelvic region, and infrequently occurs in the female gynecologic tract.

Cases presentation

The first case is a 32-year-old female with a four-centimeter lesion clinically described as a uterine fibroid. The second case is a 89-year-old female with a mass attached to the appendix and the right annexes.
Microscopically, these nodular and well delimited lesions consist of a proliferation of spindle-shaped mesenchymal cells associated with a dense inflammatory infiltrate. They both express the ALK protein by immunohistochemistry. FISH analyses revealed a rearrangement of the ALK gene in the first case.

Discussion

Inflammatory myofibroblastic tumors are mostly characterized by a rearrangement of the ALK gene with abnormal expression of the ALK protein.
This neoplasm of uncertain malignant potential has approximately one-fourth of cases experiencing recurrence and low rate of metastasis. This lesion may be underrecognized and, especially when it occurs in the female gynecologic tract, misdiagnosed as leiomyoma, endometrial stromal tumor or as a myxoid leiomyosarcoma.

Conclusions

Accurate diagnosis is important as patients with inflammatory myofibroblastic tumor should be followed up for the possibility of recurrence, and patients with recurrent or metastatic lesion can benefit from ALK inhibitor-based targeted therapy.

O 03

ALEUKEMIC LEUKEMIA CUTIS: A RARE PATHOLOGY CHALLENGING TO DIAGNOSE

F. Noel[1], V. Havelange[2], P. Saussoy[3], L. Marot[1,4], A. Camboni[1]

1. Pathology Department, Saint Luc University Hospital, Brussels, Belgium
2. Hematology Department, Saint Luc University Hospital, Brussels, Belgium
3. Clinical Biology, Saint Luc University Hospital, Brussels, Belgium
4. Dermatology, Saint Luc University Hospital, Brussels, Belgium

Background

Aleukemic leukemia cutis (ALC), an infiltration of leukemic cells involving the skin, may be the first manifestation of acute myeloid leukemia (AML), preceding the onset in marrow and blood by months and years. Incidence of ALC is low for AML and is often difficult to diagnose and is associated with a dismal prognosis.

Materials, methods and results

We report a case of a 2 years old girl with a small vulvar lesion, clinically described as a cyst. There was no medical history. The lesion was excised and sent for further histopathological investigation.

Results

A 55 year-old man consulted in another hospital for erythematous-purplish plaques on the trunk and tights. A diagnosis of “large-plaque” parapsoriasis was performed on a skin biopsy. A PUVA therapy was first initiated, but was ineffective. Few months later, a second skin biopsy revealed a dense dermal infiltrate of medium size cells positive for CD7, but negative for other T cell markers (CD3, CD5, CD2, CD4, CD8, TCR-Bêta, TCR-Delta) and negative for MPO, TDT, CD117, CD163, CD56 and Granzyme. Clonal T-cell receptor gene rearrangement was negative. A diagnosis of T cell lymphoma NOS was done. The case was reviewed in our institution. An immunohistochemistry for CD34 was performed and was positive in the skin infiltrate. CD123, CD303, CD57 and CD56 were negative. Epstein-Barr encoding region (EBER) in situ hybridization was negative. A diagnosis of leukemia cutis was proposed. Clinical examination and imaging revealed no other lesions. Bone marrow was subnormal without any blasts. A new skin biopsy was performed and revealed a dermic infiltrate by neoplastic cells with a similar morphology and a similar immunoprofile than the previous skin biopsy with the exception for a CD4 positivity. The next generation sequencing (NGS) demonstrated CEBPa, BCOR, IDH2 and NRAS mutations. A diagnosis of ALC was retained.

Conclusions

The primary extramedullary presentation of acute leukemia is extremely rare. Some aberrant markers, such as CD7, may be expressed by malignant ALC cells and could lead to misdiagnose the lesion. The early recognition of the lesions of ALC should be emphasized.

O 04

CLINICAL AND GENOMIC INDOLENCE IN LUNG-RECURRENT METASTATIC HORMONE-SENSITIVE PROSTATE CANCER

*N.M. Fonseca[†1], K. Van Der Eecken[†2,3], C. Herberts[†1], S. Verbeke[2], S.WS Ng[1], N. Lumen[3,4], E. Ritch[1], A.J. Murtha[1], C.Q. Bernales[1], E. Schönlau[1], L. Moris[5], J. Van Dorpe[2], M. Annala[6,1], A.W. Wyatt[*1,7], P. Ost[*3,8]*

*[†]co-first authors; *co-corresponding authors*
1. Vancouver Prostate Centre, Department of Urologic Sciences, University of British Columbia, British Columbia, Canada
2. Department of Pathology, Ghent University Hospital, Ghent, Belgium
3. Department of Human Structure and Repair, Ghent University, Belgium
4. Department of Urology, Ghent University Hospital, Ghent, Belgium
5. Department of Urology, University Hospitals Leuven, KU Leuven, Leuven, Belgium
6. Prostate Cancer Research Center, Faculty of Medicine and Life Sciences and BioMediTech Institute, University of Tampere, Tampere, Finland
7. Michael Smith Genome Sciences Centre, BC Cancer, Vancouver, British Columbia, Canada
8. Department of Radiation Oncology, Iridium Network, Antwerp, Belgium

Background and objective

Pulmonary involvement without synchronous bone involvement is rare in metastatic hormone-sensitive prostate cancer (mHSPC) that recurs after primary disease surgery or radiation. Guidelines recommend intensive systemic therapy, but case series suggest that patients with lung-recurrent mHSPC have relatively good outcomes. Therefore, we aimed to characterize outcomes and genomic alterations present within primary and metastatic tumours of lung-recurrent mHSPC. We hypothesized that a relatively indolent disease course would be reflected in genomic features.

Matherial and Methods

We performed a retrospective cohort study in 10 mHSPC patients with metastatic lung recurrences who underwent thoracic surgery (n=9) or fine-needle biopsy (n=1) in the years after curative-intent treatment for primary disease. After histopathological review, distinct primary tumour (n=46) and metastatic lesions (n=24) were selected. From each sample, genomic features were analysed using deep multi-gene targeted sequencing and whole exome sequencing.

Results

All patients remained alive despite a median follow-up of 139.3 months (range 97.5-170) following initial diagnosis and 55 months following lung-recurrence. Progression to metastatic hormone-resistance occurred in 2 patients. The lung-recurrent mHSPC driver landscape resembled localized prostate cancer with frequent PTEN, SPOP and chromosome 8p alterations; deleterious TP53 and DNA damage repair mutations were absent. Despite the long median time to recurrence (76.8 months), copy numbers and clonal mutations between metastases and matched primary tumours were highly similar, suggesting intra-patient homogeneity and that archival biopsy specimens are representative of late relapses.

Conclusions

Our results reveal the indolent genomic etiology underlying the relatively good clinical outcomes in this and prior cohorts of lung-recurrent mHSPC. We propose that treatment of lung-recurrent mHSPC with immediate or delayed androgen-deprivation therapy alone may be sufficient for long-term disease control. Prospective evaluation of lung-recurrent mHSPC as distinct from aggressive visceral disease, and inclusion in therapy de-intensification clinical trials is warranted.

O 05

CORRELATION OF TROP-2 EXPRESSION WITH CLINICOPATHOLOGICAL CHARACTERISTICS AND OUTCOME IN TNBC

*Hava Izci[*1], Kevin Punie[*1,2], Lise Waumans[*1,3], Annouschka Laenen[4], Hans Wildiers[1,2], Freija Verdoodt[5], Christine Desmedt[1], Jan Ardui[1,6], Ann Smeets[1,6], Sileny Han[1,6], Ines Nevelsteen[1,6], Patrick Neven[*1,6], Giuseppe Floris[*1,3]*

** Equal contribution*
1. KU Leuven - University of Leuven, Department of Oncology, B-3000 Leuven, Belgium
2. University Hospitals Leuven, Department of General Medical Oncology, B-3000 Leuven, Belgium
3. KU Leuven - University of Leuven, Department of Imaging and Pathology, Laboratory of Translational Cell & Tissue Research and University Hospitals Leuven, Department of Pathology, B-3000 Leuven, Belgium
4. Interuniversity Centre for Biostatistics and Statistical Bioinformatics, Leuven, Belgium
5. Belgian Cancer Registry, Research Department, Brussels, Belgium
6. University Hospitals Leuven, Department of Gynaecological Oncology, B-3000 Leuven, Belgium

Background

Trop-2 is a transmembrane calcium signal transducer highly expressed in multiple solid tumors including breast cancer, where overexpression has been associated with poor survival. Limited data exist about associations between Trop-2 protein expression, clinicopathological characteristics and outcome in triple-negative breast cancer (TNBC).

Methods

We performed a retrospective single-center cohort study of patients with TNBC, diagnosed in UZLeuven between 2000-2017 and stratified in 3 treatment groups (surgery + adjuvant chemotherapy(1); surgery alone(2) and neoadjuvant chemotherapy + surgery(3)). Trop-2-expression was determined semiquantitatively with IHC (ab227689, Abcam) on whole slide tumor sections from core needle-biopsy or resection specimens, and assessed as continuous and categorical variable (H-score high 200-300, medium 100-200 and low <100). Associations of Trop-2-expression with age, BMI, BRCA status, tumor grade/size, lymphovascular invasion (LVI), presence of ductal carcinoma in situ, nodal status, stromal tumor-infiltrating lymphocytes (sTILs), androgen receptor (AR), mitotic index, and outcome (distant recurrence-free interval (DRFI) and breast cancer-specific survival (BCSS)) were assessed.

Results

We included a total of 585 patients with a median age at diagnosis of 53y (range 22-85y) and 9.6y median follow-up. Trop-2 expression was high, medium and low in 96, 149 and 340 patients, respectively. In group 1 (n=406), older age at diagnosis, LVI and nodal involvement were correlated with higher Trop-2. In group 2 (n=115), AR expression, low grade and LVI were correlated with higher Trop-2. DRFI and BCSS were longer with increasing Trop-2 in the BMI-low (18-25) subgroup. In group 3 (n=64), higher BMI was correlated with higher Trop-2. Trop-2 was not associated with pCR, which was documented in 22/64 patients.

Conclusions

In this retrospective analysis of patients with TNBC in separate treatment settings, we found no consistent associations between standard clinicopathological characteristics and Trop-2 expression. Trop-2 expression was not associated with sTILs or outcome. Limited numbers of events warrant caution in interpretation.

O 06

IMPLEMENTATION OF AUTOMATIC QUANTIFICATION OF KI-67 IN WELL-DIFFERENTIATED GASTRO-ENTERO-PANCREATIC NEUROENDOCRINE TUMORS: TOWARDS STANDARDIZED EVALUATION INTO DAILY PRACTICE

Frédéric Lifrange[1,3], Nejla Gumus[2], Ligia Craciun[1], Maria Gomez Galdon[1], Pieter Demetter[1,2], Laurine Verset[1,2]

1. Department of Pathology, Institut Jules Bordet, Université Libre de Bruxelles (ULB), Brussels, Belgium
2. Université Libre de Bruxelles (ULB), Brussels, Belgium
3. Université de Liège (Uliège), Liège, Belgium

Background and objectives

Proliferation index (PI) estimated by Ki-67 immunostaining is an important prognostic factor for gastro-entero-pancreatic neuroendocrine tumors (GEP-NET). Several quantification methods exist, the current gold standard is represented by manual counting (MC).
The objective of our study was to evaluate the feasibility of implementing automatic quantification in routine for the evaluation of the proliferation index in well differentiated GEP-NETs.

Matherial and Methods

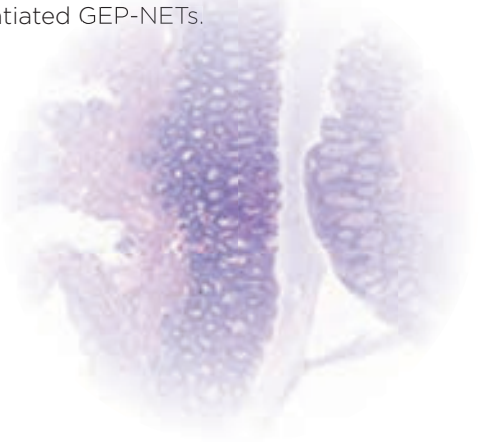
PI of 30 samples of well-differentiated GEP-NETs was assessed by 2 methods: MC using ImageJ software and automatic counting (AC) using QuPath software. MC was performed by a senior observer (confirmed pathologist) and a junior observer (previously trained medical student). Regions of interest (ROI) were previously selected by the senior observer to constitute the training panel and the validation panel. The junior observer's MC was first validated on the training panel. Validation panel was then evaluated manually by the 2 observers and by the AC. Inter-observer concordance and concordance between observers and QuPath software analyses were based on either the Lin's concordance correlation coefficient (CCC) for Ki-67 PI absolute values and weighted κ coefficient for grade.

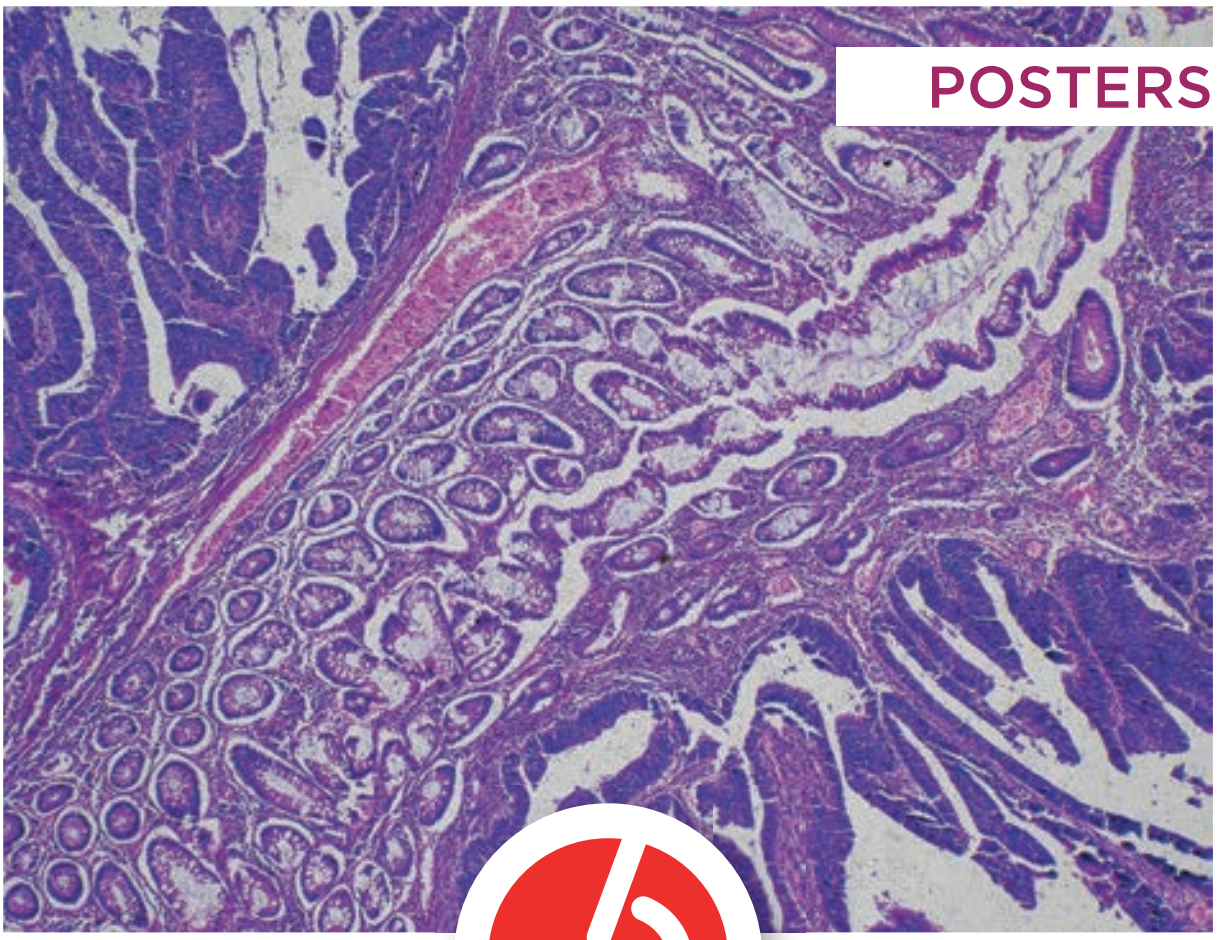
Results

137 ROIs from 30 samples were selected. Inter-observer CCC and Inter-observermean-Qupath CCC reached 0.91 (confidence interval 95%(CI): 0,89-0,93) and 0,90 (0,86-0,93), respectively, and we observed a substantial agreement with regard to grade between the two observers (k: 0,81 (0,71-0,91) and between the two observers and QuPath (k: 0,80 (0,69-0,91).
Senior observer evaluated PI faster than junior observer (mean timesenior / ROI: 11 min 21s; minimum: 2 min, maximum: 21 min 48s; mean timejunior / ROI: 16 min 33s; minimum: 8 min 23s, maximum: 37 min 43s). AC duration depended only on duration of slide scanning.

Conclusions

Automatic quantification using Qupath software can be easily implemented in routine for the evaluation of the proliferation index in well differentiated GEP-NETs.





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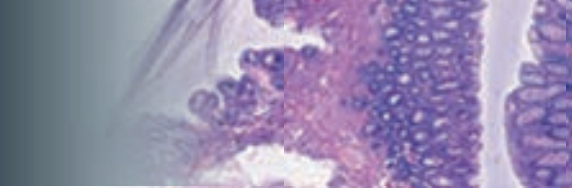

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P 01

THE BELGIAN VIRTUAL TUMOURBANK PROJECT: AVAILABILITY OF RARE CANCERS IN THE CATALOGUE

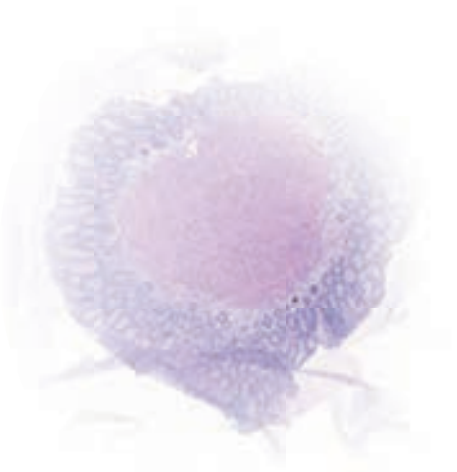
E. Van der Stock[1], K. Vande Loock[1], A. Debucquoy[1], K. Emmerechts[1], L. Van Eycken[1], E. Marbaix[2] on behalf of the Steering Committee of the Belgian Virtual Tumourbank

1. Belgian Cancer Registry, Koningsstraat 215 bus 7, 1210 Brussels, Belgium
2. Service d'Anatomie Pathologique, Université Catholique de Louvain, St-Luc University Hospital, Brussels, Belgium

Representing about 22% of all cancer cases diagnosed in Europe, rare cancers are often forgotten in the group of rare diseases. Availability of tumour samples in biobanks might be limited, especially for rare cancers. Within the Belgian Virtual Tumourbank (BVT) network, the search for tumour samples scattered among eleven Belgian university hospitals is facilitated by the central data collection of the residual human tumour samples that are stored locally. The data collected at sample level is made available for researchers via the online BVT catalogue. High quality of the data is guaranteed by automatic and manual controls performed by the BVT project team at the Belgian Cancer Registry. Currently, more than 111,000 registrations, including 86% primary tumour samples and 12% metastasis samples, are available in the catalogue for researchers in the oncology field.

The availability of rare cancers in the BVT catalogue was investigated. Based on the criteria proposed by the RARECARE project (http://www.rarecare.eu/), 33,131 registrations from 15,414 patients with rare cancers could be retrieved. The most common sample localisation of the rare cancers in the BVT catalogue is the central nervous system (17.7%). Digestive organs (like stomach, liver and pancreas but excluding colon and rectum) and lymph nodes complete the top three of sample localisations with 13.3% and 11.9% respectively. For 69.2% of the rare cancer registrations, only residual tumour tissue is stored. For some patients, also additional types of material are stored at the local biobanks and registered in the BVT catalogue. The most common type is corresponding normal tissue: 16.8% of the registrations. Blood (11.5%), plasma (10.9%) and serum (9.8%) are also available in some local biobanks.

Our data illustrate the great value of the BVT catalogue for cancer research, in particular for research on rare cancers.



P 02

SPECTRUM OF NEUROPATHOLOGICAL FINDINGS IN A SERIES OF 13 COVID-19 PATIENTS

Sieben A[1,2], Libbrecht S[1], Van Rafelghem B[3], Sirimsi S[1], Jacobs W[3,4], Van Dorpe J[5], Lammens M[1,2]

1. Department of Pathology, Antwerp University Hospital, Antwerp University, Edegem, Belgium
2. Institute Born-Bunge, Antwerp University, Edegem, Belgium
3. Department of Forensic Medicine, Antwerp University Hospital, Antwerp University, Edegem, Belgium
4. Military Hospital Queen Astrid, Crisis Unit, Belgian Defense, Brussels, Belgium
5. Department of Pathology, Gent University Hospital, Gent University, Gent, Belgium

COVID-19 has been associated with a broad spectrum of abnormalities in the central nervous system including inflammatory changes and coagulopathy.

We present the neuropathological findings in the brains of 13 COVID-19 patients. We included the clinical history with focus on immunological status. Our neuropathological workup included sampling of representative brain areas with olfactory bulb, meninges and choroid plexus. Histological and immunohistological stainings for inflammation including activated microglia, for cerebrovascular pathology as well as an immunohistochemical panel for neurodegenerative disorders were performed. Mean age of our cohort was 68 [33;90]. 4 patients had a previous diagnosis of a neurodegenerative disorder. Postmortem evaluation revealed severe meningoencephalitis with multiple hemorrhagic infarctions (n=1), multiple hemorrhagic infarctions (n=1), major hemorrhages (n=2) and hypoxic encephalopathy (n=1). Isolated bulbar and meningeal inflammation was found in 3 patients. 5 patients did not have any neuropathological changes. Our neurodegenerative analysis detected FTLD-TDP, Alzheimer's disease neuropathological changes and hippocampal sclerosis.

Our cohort confirms the variety of neuropathological findings in patients with COVID-19 with a range from isolated bulbitis/ meningitis to full blown meningo-encephalitis with hemorrhagic infarctions. The pleiad in symptomatology suggests that other factors should be taken into account, including concomitant cerebral and vascular disorders, as well as immunological status.

P 03

VULVAL INTESTINAL/ENTERIC HETEROTOPIA IN A PATIENT WITH CROHN'S DISEASE

S. Bouri[1,3], L. Verset[2], X. Catteau[1,3] J-C. Noël[1,3]

1. Department of Pathology, Erasme University Hospital, Université Libre de Bruxelles, Brussels, Belgium
2. Department of Pathology, Bordet Institue Université Libre de Bruxelles, Brussels, Belgium
3. Centre Universitaire Inter Regional d'Expertise en Anatomie Pathologique Hospitalière (CurePath), Jumet, Belgium

Intestinal/enteric heterotopia of the vulva is an extremely rare disease with only 3 cases described in the literature. We report here an unusual case of this disease occurring in a 26-year-old patient in a context of Crohn's disease. To the best of our knowledge, such type of association has not been previously described. The potential origins of these lesions including metaplastic transformation, dysontogenetic changes, or epithelial colonic displacement/ implantation are discussed.

Reference:
Case Rep Pathol , 2020 Mar 19;2020:6203826.
doi: 10.1155/2020/6203826.

P 04

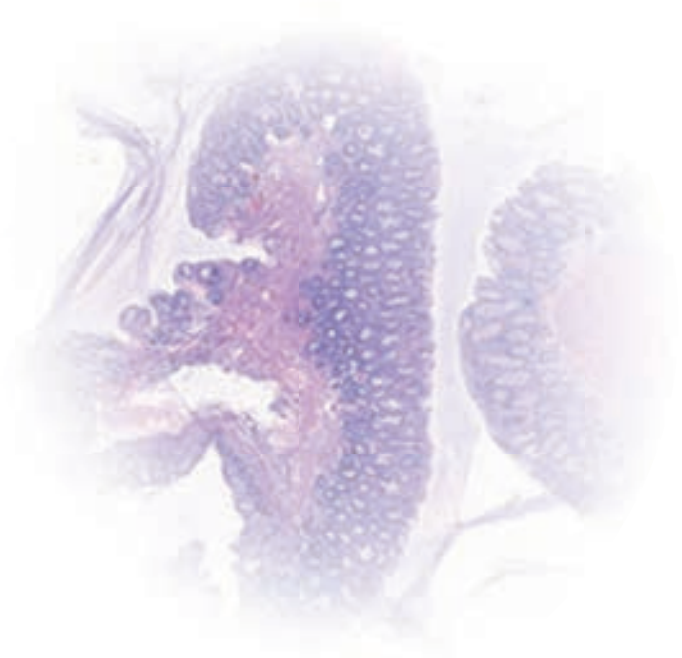
P53 AND PIK3CA MUTATIONS IN KRAS/HER2 NEGATIVE OVARIAN INTESTINAL-TYPE MUCINOUS CARCINOMA ASSOCIATED WITH MATURE TERATOMA

S. Bouri[1,3], P. Simon[2], N. D'Haene[1,3], X. Catteau[1,3] J-C. Noël[1,3]

1. Department of Pathology, Erasme University Hospital, Université Libre de Bruxelles, Brussels, Belgium
2. Department of Gynecology, Erasme University Hospital, Université Libre de Bruxelles, Brussels, Belgium
3. Centre Universitaire Inter Regional d'Expertise en Anatomie Pathologique Hospitalière (CurePath), Jumet, Belgium

Primary ovarian intestinal-type mucinous carcinomas associated with mature teratoma are rare and represent less than 3% of all primary ovarian neoplasms. The molecular profile of these tumors is still controversial. We report here the first case of mucinous ovarian tumor in which mutation of the PIK3CA and P53 genes could be demonstrated by next generation sequencing technique without KRAS mutation or HER2 amplification. Our data suggest that these mucinous carcinoma variants probably present an extremely complex molecular biology profile that should be known in the future to stratify therapeutic outcomes and potential targeted therapies, particularly in recurrent disease.

Reference:
Case Rep Obstet Gynecol, 2020 Jul 22;2020:8863610.
doi: 10.1155/2020/8863610.



P 05

ESTABLISHMENT OF A ZEBRAFISH DERIVED XENOGRAFT PLATFORM FOR PERSONALIZED TREATMENT OF OVARIAN CANCER

Charlotte Fieuws[1,2], Olivier De Wever[2,3], Hannelore Denys[2,4], Koen Van De Vijver[2,5], Kathleen Claes[1,2]

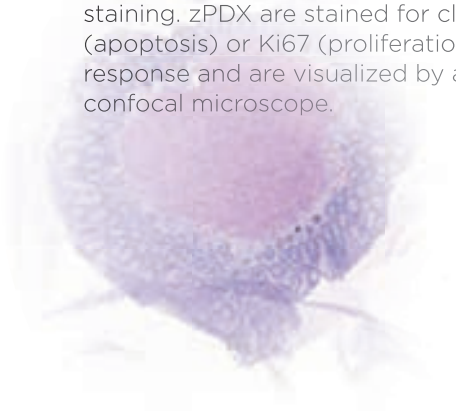
1. Center for Medical Genetics, Ghent University, Ghent, Belgium
2. Cancer Research Institute Ghent (CRIG), Ghent University, Ghent, Belgium
3. Laboratory of Experimental Cancer Research, Ghent University, Ghent, Belgium
4. Department of Medical Oncology, Ghent University Hospital, Ghent, Belgium
5. Department of Pathology, Ghent University Hospital, Ghent, Belgium

Background

Standard therapy is applied to all epithelial ovarian cancers, however specific subtypes do not respond. To improve treatment, an in vivo predictive test for treatment response is warranted. Very promising are zebrafish patient derived xenografts (zPDX) platforms which are fast, cost-effective and require limited donor material. The aim of this study is to optimize this zPDX platform starting from cancer cell lines.

Material and methods

Tumor cells are fluorescently labeled and injected into the perivitelline space of 2 day old zebrafish embryos. One day post injection, xenografts are scored for presence of tumor cells and size. Then they are randomly distributed into treatment groups. After 4 days of treatment, zPDX are euthanized and fixed for whole mount staining. zPDX are stained for cleaved-caspase 3 (apoptosis) or Ki67 (proliferation) to score tumor response and are visualized by a fluorescence confocal microscope.



Results

We have successfully engrafted 3 ovarian cancer cell lines with different characteristics in zebrafish embryos. M28/2, a LGSOC cell line derived from mice PDX shows a KRAS c.35 G > T (p.(Gly12Val) mutation and is sensitive to the MEK inhibitor, trametinib (De Thaye et al, 2020). A2780 and OVCAR-3 cell lines display in vitro sensitivity to PARPi and chemotherapy. For all 3 cell lines successful engraftment was observed by formation of compact tumors. Ki67 staining showed clear proliferation and upon treatment of zPDX with trametinib higher caspase activity and pyknotic nuclei were observed compared to control, providing evidence that the same sensitivity is obtained as in vitro and mouse PDX. Evaluation of the tumor response in zebrafish engrafted with A2780 and OVCAR-3 cell lines and treated with platinum and/or PARPi, is ongoing.

Discussion

The optimized protocols are recently introduced to establish zebrafish PDX models from ovarian cancer tumors. Conventional treatments will be applied to see if tumor response in zPDX is similar as in the patients. In parallel we will use targeted treatments to achieve higher tumor response, and thus a better outcome for the patient.

P 06
A REMARKABLE MESENCHYMAL TUMOR OF THE FINGER

Maaike Ramael[1], Raf Sciot[2], David Creytens[3], Walter Jacobs[4], Wim Develter[5], Marc Ramael[1,5]

1. Antwerp University , Belgium
2. Department of Pathology, University Hospital Leuven, Belgium
3. Department of Pathology, University Hospital Ghent, Belgium
4. Department of Plastic Surgery, Heilig Hartziekenhuis Lier, Belgium
5. Department of Pathology, Heilig Hartziekenhuis Lier, Belgium

Clinical history/Background

A 53 year old patient visited the outpatient clinic for a painless local swelling of digit 4 right. A nodular lesion was resected at the right digit 4 (DIP between extensor and collateral ligament) with clinical suspicion of a mucoid cyst.

Material & Methods/results

The resection specimen consisted of a solid nodulus with diameter of 0.7 cm. Classical H/E sections revealed a circumscribed multinodular mesenchymal tumour consisting of spindle cells to ovoid/epitheloid tumoural cells embedded in a fibrillar to myxoid matrix. The nucleus was regular round to oval and sometimes elongated. The chromatine pattern was fine with a rather inconspiciuos nucleolus. There were some sporadic mitoses. Atypia, hyperchromasia or necrosis were not observed. The cytoplasm was eosinophilic. Focally the matrix was hyaline thereby isolating epitheloid cells in small groups. Immunohistochemistry revealed immunoreactivity for ERG, SMARCB1(INI-1), SMARCA4. There was no aberrant immunoreactivity for pancytokeratine AE1/AE3, EMA, CD34, S100, SOX10, Calponine, Caldesmon, Desmine, MUC4 or panTRK.
FISH investigation showed no rearrangement for EWSR1 (22q12) and SS18(18q11.2) nor for TFE3.

Conclusion

The resected mesenchymal tumour with myxochondroid matrix can be considered as 'distinctive myxoid spindle and round cell neoplasm' according to Prof. Fletcher. The localisation (finger) is typical as well as the almost exclusive expression of ERG. He mentions a series of 60 hitherto unpublished cases with recidive in 2-3 cases but no metastasis.

P 07

ARTIFICIAL INTELLIGENCE HELPS IMPROVE ACCURACY AND EFFICIENCY IN PROSTATIC BIOPSY DIAGNOSIS

J.A. Retamero[1], P. Raciti[1], P. Hamilton[1], B. Rothrock[1], J. Sue[1], M. Horton[1], C. Kanan[1]

1. Paige.AI Inc., New York, USA

Background

Artificial intelligence (AI) applied to pathology promises increased diagnostic performance and efficiency for pathologists. However, given the novelty of the technology, relatively little evidence exists on the topic.

Objective

To test how the diagnostic performance and reading speed of pathologists varies between AI-aided diagnostic sessions and unaided readings. Also, we tested the performance of AI used as a post-diagnostic quality control tool.

Materials and Methods

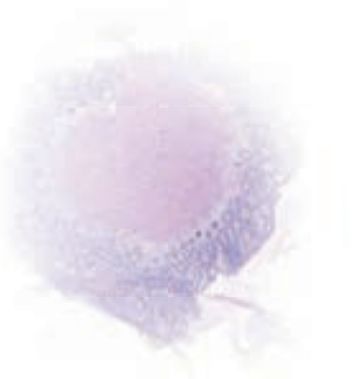
A timed multi reader, multi case study was conducted using 232 prostatic biopsy slides, read by three pathologists with and without AI assistance. In addition, an AI algorithm was applied retrospectively to prostatic biopsies from 100 patients as a quality control tool.

Results

When aided by AI, pathologists were more sensitive and more specific in detecting cancer foci in biopsy specimens, with a 60% reduction in diagnostic errors. In addition, the average reading time per slide was significantly shorter when pathologists were aided by AI. Also, AI used as a quality control tool identified 4 patients with a primary benign diagnosis that was subsequently reviewed and upgraded to ASAP/malignant.

Conclusion

AI assisted diagnosis helps pathologists be more accurate and more efficient, by reducing the number of diagnostic errors and reading times. In addition, AI can be used as a stand-alone post-diagnostic quality control tool to help identify potential errors.



P 08
AN UNUSUAL EXTRA-NODAL LOCATION OF ROSAI DORFMAN DISEASE

T. Yacoubi[1], A. Al Hada[2]

1. Department of Pathology and Laboratory Medicine, National Guard hospital, Al Ahsa, Saudi Arabia
2. Department of Neurosurgery, National Guard hospital, Al Ahsa, Saudi Arabia

Background

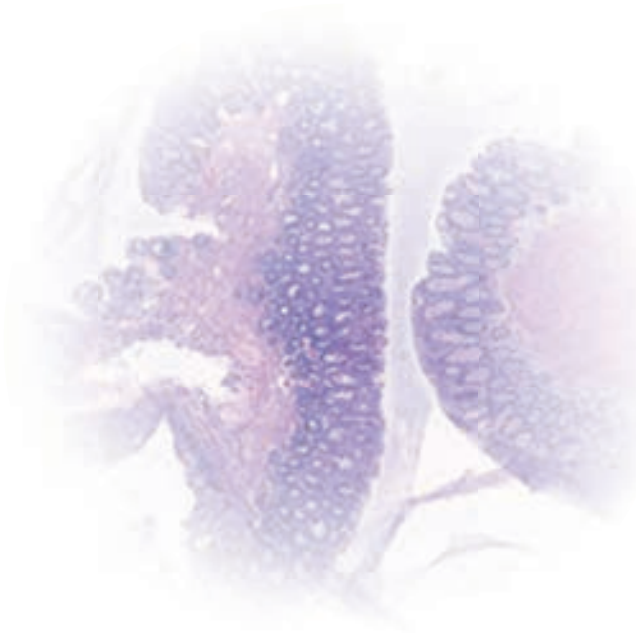
Rosai-Dorfman disease is a rare pathologic entity characterized by massively enlarged painless cervical lymph nodes, showing histologically a lymphadenopathy with massive histiocytosis. Extra Nodal location of this disease is uncommon, specially in the skull and the Nervous system.

Case presentation

A 31-year-old lady known patient for Hypertension, idiopathic anemia a with hemoglobin ranging between 9-7 over the last 3 years. Presented with right frontal scalp swelling for one year duration, increasing gradually in size. The imaging showed an extra-axial mass, involving the skull and located closely to the cortex and meninges, suspecting meningioma. The lesion was excised and the underlying brain is biopsied. Histologically the lesion comprised multiple histiocytes, many are foamy associated to lymphocytes and plasma cells, emperipolesis is also identified, the immunostaining by CD68 and s100 is positive in these histocytes, the plasma cells are polyclonal and CD1a is negative. Thus, the diagnosis of Rosai-Dorfman disease is proposed.

Conclusion

Extra-nodal Rosai Dorfman disease is benign lesion, with rare location in the skull. this diagnosis should not be confused with other aggressive lesions like plasmacytoma. The immunostaining plays a major role in the diagnosis.
We review here the criteria of diagnosis, the differential diagnosis and the underlying patient status.



P 09

RADIOGENIC ANGIOSARCOMA OF THE BREAST: CASE REPORT AND REVIEW OF THE LITERATURE

T. Yacoubi[1], T.i Al Qurashi[2]

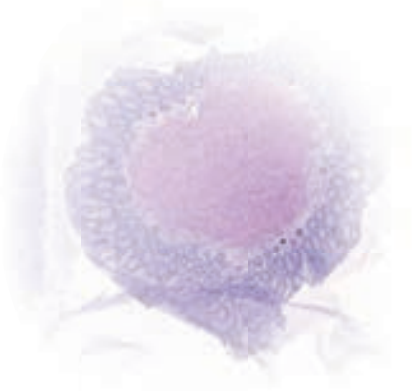
1. Department of Pathology and Laboratory Medicine, National Guard Hospital , Al Ahsa, Saudi Arabia
2. Department of Breast Surgery, National Guard Hospital , Al Ahsa, Saudi Arabia

Background

Angiosarcoma of the breast can occur primarily or can be secondary to radiation for breast cancer. Radiogenic angiosarcoma is rare late sequela of local irradiation of the breast or chest wall skin after breast cancer.

Case presentation

A 79-year lady, underwent a total mastectomy with chemotherapy and radiation for invasive carcinoma of left breast. The patient was free for disease for 4 years, since she presented with a blueish-brownish nodule of the skin of the thoracic wall in the site of the mastectomy. The lesion was excised with surrounding skin. histologically the lesion was epithelioid angiosarcoma. Due the corona pandemic the patient was lost and came back in 2021 for appearance of multiple lesions, some are like pt over the skin and some others are deeply located in the subcutaneous tissue with grossly free surgical margins. These lesions are similar to the previous lesions, and are consistent with epithelioid angiosarcoma, qualified as radiogenic angiosarcoma.



Conclusions

Through this case, we emphasize the diagnostic criteria of the radiogenic and primarily developed angiosarcoma of the breast and their prognosis ; as well as the differential diagnosis.

P 10
SPECIAL CASE OF A TRICHILEMMAL CYST WITH ECCRINE DIFFERENTIATION

M. Reginster[1], J-L. Dargent[1], M. Hérin[1]

1. Institut de Pathologie et de Génétique, Gosselies, Belgium

Background

Trichilemmal cysts or pilar cyst are common lesions. However, foci of glandular differentiation within these cysts have rarely been described.

Case presentation

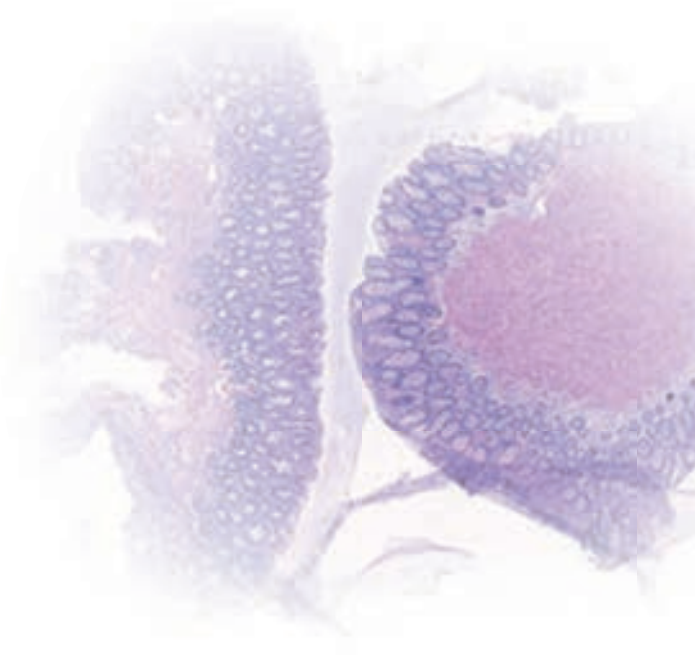
We report a case of a 63-year-old man with a cutaneous lesion of 0.5 cm on the outside of the right leg. Microscopic examination showed a cystic formation coated with a squamous epithelium containing small tubular structures. The diagnosis of trichilemmal cyst was confirmed by positivity for immunohistochemical markers CK17, p63, CK 5/6 and CK14. The tubular structures expressed CEA, EMA and CK7 and showed PAS positive deposits. There was no significant expression of GCDFP-15 and podoplanin (D2-40).
Morphology and immunohistochemical profile were suggestive of an hybrid cyst of the trichilemmal type with small foci of eccrine differentiation.

Discussion

A wide variety of adnexal differentiations are observed in hybrid trichilemnal cysts. Two publications concerned apocrine and sebaceous differentiation, and two (including this one) showed eccrine differentiation.
These observations suggest the existence of pluripotent cells within the epidermis and the appendages that can give rise to follicular, sebaceous, apocrine and eccrine lines.
The clinical signification of these rare and underrecognized lesions is not known.

Conclusions

Trichilemmal cyst with eccrine differentiation has only been described once in the literature. Its clinical significance is unknown and needs further obseervations.



P 11

MYOEPITHELIAL CARCINOMA IN CERVICAL BONES OF AN 8-MONTH INFANT. A MIMICKER OF ROUND CELL SARCOMA. CASE REPORT

J. Liu[1], K. Norga[2], M. De Praeter[3], P. Pauwels[1,4]

1. Department of Pathology, University Hospital of Antwerp, Belgium
2. Department of Paediatric Oncology, University Hospital of Antwerp, Belgium
3. Department of Neurosurgery, University Hospital of Antwerp, Belgium
4. Centrum For Oncological Research, University of Antwerp, Belgium

Introduction

Myoepithelial neoplasms, including the myoepithelial carcinoma, are frequently seen in the salivary glands. However, in the last two decades there are series of cases of myoepithelial tumors reported in the soft tissue. While having many similarities to the salivary gland neoplasms, the myoepithelial neoplasms of the soft tissue are distinct in several ways, including the histologic criteria for malignancy and their characteristic genetic aberrations. Myoepithelial tumors of the soft tissue occur also in pediatric patients and are frequently malignant.

Material and Methods

We describe a case of a 8-month-old infant with paralysis of the upper right extremity. The performed computed tomography (CT-) scan and X-ray of the right shoulder and neck don´t show any signs of lesion. Then a magnetic resonance imaging (MRI) is done and show a mass in the lateral part of the cervical bones C2-C4 with probably invasion in the neuroforamen. He has underwent a surgical excision of the cervical mass and the bulky mass is sent for further histopathological investigation.

Results

In the haematoxylin-eosin (HE) section the tumor show necrosis and sheets of rounded cells with moderate to severe cytologic atypia. These cells have limited amphophilic cytoplasm and frequently a vesicular, irregular nuclei with a prominent nucleoli. There are scattered mitotic figures. The immunohistochemically staining of these lesional cells show multifocal CD99 positivity. Also there are expression for pan-keratin and EMA, while S100 and GFAP are negative. These neoplastic cells show lost of nuclear INI-1 expression and don´t harbored the EWR1-gene rearrangement. Furthermore, the blood samples result a slightly elevated level of NSE (neuron specific enolase) with a normal catecholamine level in urine. Based on the morphology and immunohistochemistry the diagnosis of myoepithelial carcinoma is suggested. After the surgery, the patient regains its function of his right upper extremity.

Conclusions

Myoepithelial tumors in soft tissue are very uncommon and have increasingly characterized over the last two decades. They most commonly arise as a subcutaneous nodules on the extremities and limb girdles. These neoplasms occur approximately 20% in pediatric patient, in whom they are frequently malignant with an aggressive course. The subcategory myoepithelial carcinoma of the soft tissue, in contrast to those of the salivary gland, are graded as low, intermediate and high based on the cytologic atypia. Although the EWSR1 gene rearrangement is in up to 45% found in soft tissue myoepithelioma and myoepithelial carcinoma, this patient show no INI-1 expression indicating that the gene product on chromosome 22 is lost or deleted.

P 12

POLYARTERITIS NODOSA IN THE APPENDIX OF A 14-YEAR-OLD FEMALE: A CLINICAL MIMICKER OF APPENDICITIS. CASE REPORT

J. Liu[1], K. Norga[2], M. De Maat[3], P. Pauwels[1,4]

1. Department of Pathology, University Hospital of Antwerp
2. Department of Paediatric Oncology, University Hospital of Antwerp
3. Department of Abdominal Surgery, University Hospital of Antwerp
4. Centrum For Oncological Research, University of Antwerp

Introduction

Polyarteritis nodosa (PAN) is a vasculitis of the small to medium-sized muscular blood vessels which frequently affect the kidneys, skin etc. There are only a few cases of polyarteritis nodosa reported in the appendix.

Material and Methods

Here we describe a case of a 14-year-old female with chronic intermittent pain in the lower right abdomen in the past 2 years. The patient has underwent multiple radiological investigation such as ultrasonography, computed tomography (CT-) scan. However, the clinical examination and radiological images don´t show any sign of a lesion and still there is no explanation for the unclarified intermittent abdominal pain in the right fossa region. Then a diagnostic laparoscopic resection of the appendix is performed for this patient and the specimen is send for further histopathological examination.

Results

The haematoxylin-eosin (HE) sections of the whole-embedded appendix show neither mucosal inflammation nor signs of infection. But in the deeper part of the muscular layer and the (sub-)serosal tissue of the appendix, there is an dense, concentric infiltrate around and in the muscular wall of the small to medium blood vessels. On higher magnification, this perivascular infiltrate, so called cuffing, consist of small non-atypical lymphocytes, some eosinophil granulocytes, plasma cells and histiocytes. Some of these affected blood vessels have fibrinoid necrosis. Therefore, the diagnosis of a systemic vasculitis is made, with a strong suggestion for the polyarteritis nodosa type.

Conclusions

Polyarteritis nodosa is a systemic vasculitis of the small to medium-sized arteries and is a subcategory of the Kawasaki disease which is often seen in patients with clinical presentation of the affected kidneys or skin. So far to our knowledge, there are only a few cases described of the appendix in the English literature. This disease could be fatal for the patient if it´s not treated properly. Our patient has received additional investigation such as blood samples for antibodies ANCA, ANA etc., and administration of corticosteroid is started.

P 13

FURTHER EVIDENCE THAT LOW-GRADE RENAL ONCOCYTIC TUMOR IS AN ENTITY DESERVING SEPARATE CLASSIFICATION

L. Libbrecht[1,2], G. Pairet[3], M-A. Van Caillie[4], H. Dano[2]

1. Department of Pathology, AZ Groeninge, Kortrijk, Belgium
2. Department of Pathology, Cliniques Universitaires Saint-Luc, Brussels, Belgium
3. Department of Pathology, Centre Hospitalier Jolimont, La Louvière, Belgium
4. Department of Pathology, AZ Sint-Lucas Brugge, Brugge, Belgium

Background and objective

Renal low-grade oncocytic tumor (LOT) is an emerging entity within the oncocytic renal tumors spectrum and several recently published series indicate it probably deserves separate classification. However, more comprehensive phenotyping is warranted before classifying it as a specific WHO-recognised entity. Therefore, we planned to probe our kidney tumor registry for LOT cases and aimed to identify additional markers supporting the idea this tumor should be considered a distinct entity.

Materials and methods

The renal tumor archives in the departments of pathology of the different authors were searched for LOT cases. Identified cases were diagnosed in consensus. Immunohistochemistry profiling was performed, with a focus on identification of possible new markers.

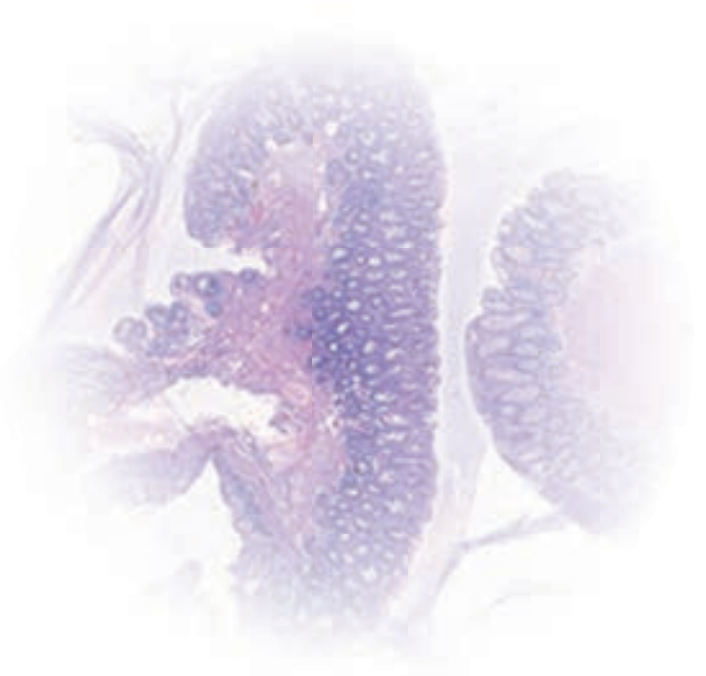
Results

We identified 3 tumors with a macroscopical, microscopical and immunohistochemical phenotype corresponding to that described for LOT in the series in the literature. In these series, the extent of AMACR-positivity was variable and in our 3 cases it appeared to be rather at the high-end of the described spectrum. All 3 cases showed a diffuse GATA3 expression, which has been proposed to reflect a distal nephron origin.

We evaluated the literature for GATA3 expression throughout the spectrum of renal tumors and found that only clear cell papillary renal cell carcinoma and reversed polarity renal papillary neoplasm have been described as showing diffuse GATA3-positivity. Since these tumors have a different morphological profiles than LOT, diffuse GATA3 expression might not only serve as argument that LOT is indeed a distinct entity, but it could possibly also be used as a diagnostic marker.

Conclusion

Our findings support the idea that renal LOT is a distinct entity and our observation of diffuse GATA3-expression in this small number of cases should be evaluated in larger series, to further investigate whether GATA3 can be used as a diagnostic and entity-defining marker for LOT.



P 14

WARTHIN-LIKE MUCOEPIDERMOID CARCINOMA : PRESENTATION OF 3 CASES, EMPHASIZING ON THE IMPACT OF MOLECULAR TECHNIQUES IN THE DIFFERENTIAL DIAGNOSIS OF SALIVARY GLAND CARCINOMAS

F. Lifrange[1,2], P. Demetter[1], N. De Saint Aubain[1]

1. Department of Pathology, Institut Jules Bordet, Université Libre de Bruxelles (ULB), Brussels, Belgium
2. Department of Pathology, Université de Liège (Uliège), Liège, Belgium

Background and objective

Mucoepidermoid carcinoma (MEC) is the most common salivary gland malignancy. Several morphological variants of MEC have been described. Some, including the ciliated and Warthin-like variants, are easily confused with benign lesions.
Over the last years, an increasing number of specific genetic alterations have been reported in salivary gland tumors, including MYB rearrangements in adenoid cystic carcinoma, ETV6 rearrangements in mammary-analogue secretory carcinoma, EWSR1-ATF1 fusion in hyalinizing clear cell carcinoma, HER2 amplification in salivary duct carcinoma, PRKD rearrangements in cribriform adenocarcinoma of minor salivary glands.
Recent studies have shown that most MEC carry a CRTC1 or CRCT3-MAML2 fusion transcript, which can be detected by fluorescence in situ hybridization (FISH), with a sensitivity of 82% and a specificity of 100%.
We present 3 cases of the Warthin-like MEC variant and discuss the role of molecular biology in the differential diagnosis of salivary gland carcinomas.

Materials and methods

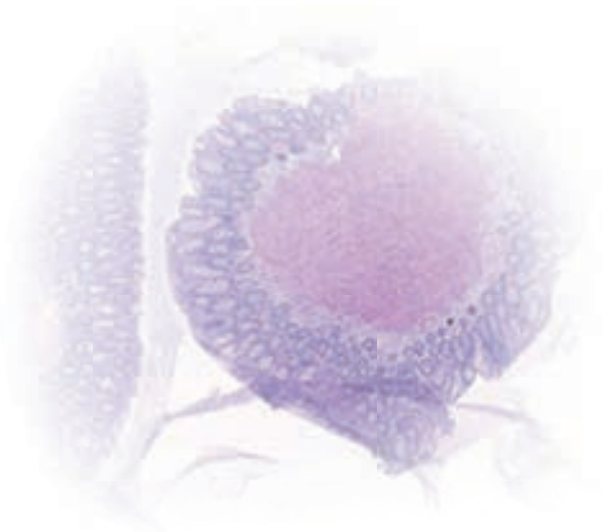
3 cases of Warthin-like MEC were retrieved in our archives and were reviewed by 2 pathologists. FISH for MAML2 was performed in all 3 cases.

Results

MAML-2 rearrangements were demonstrated by FISH in our 3 cases, confirming the morphological diagnosis of Warthin-like mucoepidermoid carcinoma.

Conclusions

We present 3 cases of a recently described variant of MEC and discuss the impact of molecular techniques in the diagnosis of salivary gland malignancies.



P 15

THE PROGNOSTIC ROLE OF TUMOR-INFILTRATING LYMPHOCYTES AFTER NEOADJUVANT TREATMENT IN INFLAMMATORY BREAST CANCER

G. Broeckx[1,2], C. van Berckelaer[2,3,4], C. Colpaert[3,5], L. Vercauteren[6], I. Vermeiren[6], S. Van Laere[2,3], L. Dirix[3,7], P. Van Dam[2,3,4]

1. Department of Pathology, Antwerp University Hospital (UZA), Edegem, Belgium
2. Centre of Oncological Research (CORE), Antwerp University (UA), Wilrijk, Belgium
3. Translational Cancer Research Unit, Gasthuiszusters Ziekenhuizen Antwerpen (GZA), Antwerp, Belgium
4. Multidisciplinary Breast Clinic, Unit Gynecologic Oncology, Antwerp University Hospital (UZA), Edegem, Belgium
5. Department of Pathology, AZ Turnhout, Belgium
6. Faculty of Medicine, Antwerp University (UA), Wilrijk, Belgium
7. Department of medical oncology, Gasthuiszusters Ziekenhuizen Antwerpen (GZA), Campus SintAugustinus, Antwerp, Belgium

Introduction

Increasing evidence indicates the importance of stromal Tumor Infiltrating Lymphocytes (sTIL) in inflammatory breast cancer (IBC) , a rare, but aggressive type of breast cancer. Infiltration with sTIL is associated with a better response to neo-adjuvant chemotherapy (NACT) and longer overall survival (OS) in IBC patients. However, the prognostic role of sTIL in patients without a complete pathological response (pCR) after NACT remains unclear. In this study we evaluated the effect of NACT on sTIL in IBC and locally advanced non-inflammatory breast cancer (LABC) and the prognostic impact of sTIL in IBC.

Methodology

In this retrospective case-control study we evaluated sTIL according to the recommendations by the International TILs Working Group in patients with IBC (n=60) and LABC (n=134), before (diagnostic biopsy) and after (resection specimen) NACT. sTIL difference between pre-treatment and post-treatment specimen was called δsTIL.

Results

25% of the IBC patients had pCR after NACT, all showing less than 1% sTIL in the tumor bed area. There was no significant difference in the pre-treatment median sTIL score between IBC (12.5%, range: 1-80%) and LABC (10%, range: 1-85%). Both in IBC (median δsTIL: -4.5%, P=0.01) and in LABC (median δsTIL: -1%, P=0.06) the number of sTIL was lower after NACT. This decrease was significantly greater in the IBC cohort (P= 0.04) and was independent of molecular subtype, grade or nodal status.
Higher sTIL before NACT was associated with better OS (P= 0.006) in IBC. IBC patients without pCR, but with a stronger decrease (> 4.5 %) of sTIL after NACT had a better OS (P= 0.04) and disease-free survival DFS (P=0.04). There was a significant correlation between δsTIL and both higher sTIL before NACT (P<.001) and lower residual cancer cellularity (P=0.02). However, in a multivariate model, both a strong decrease of sTIL (HR: 0.32; 95% CI 0.10-1.00; P=0.05) and a low residual cancer cellularity (HR: 0.22; 95% CI 0.07-0.68; P=0.009) were independent predictors of a better DFS.

Conclusion

The IBC phenotype was associated with a stronger decrease of sTIL after NACT; this decrease was an independent predictor of better prognosis in IBC.

P 16

MALIGNANT PLEURAL MESOTHELIOMA WITH AN EML4-ALK FUSION: EXPECT THE UNEXPECTED!

F. Cordier[1], J. Van der Meulen[2,3,4], N. van Roy[3,4,5], H. van Dijck[6], F. Vanhoenacker[7], M. Lambrechts[8], V. Noyez[9], L. Ferdinande[1,3], A. Dendooven[1,3,10], J. Van Dorpe[1,3], D. Creytens[1,3]

1. Department of Pathology, Ghent University Hospital, Ghent University, Ghent, Belgium
2. Molecular Diagnostics Ghent University Hospital (MDG), Ghent University Hospital, Ghent University, Ghent, Belgium
3. CRIG, Cancer Research Institute Ghent, Ghent University Hospital, Ghent University, Ghent, Belgium
4. Centre for Medical Genetics, Ghent University Hospital, Ghent University, Ghent, Belgium
5. Department of Biomolecular Medicine, Ghent University, Ghent, Belgium
6. Department of Pathology, AZ St. Maarten, Mechelen, Belgium
7. Department of Radiology, General Hospital Sint-Maarten Mechelen, Antwerp University Hospital, Antwerp University, Belgium
8. Department of Pneumology, General Hospital Sint-Maarten Mechelen, Antwerp University Hospital, Antwerp University, Belgium
9. Department of Thoracic and Vascular Surgery, AZ Sint-Maarten Hospital, Mechelen, Belgium
10. Faculty of Medicine and Health Sciences, Antwerp University, Wilrijk, Belgium

Malignant mesothelioma is a rare aggressive neoplasm, mostly arising from the pleura in patients with a chronic exposure to asbestosis. The genomic features of malignant pleural mesothelioma (MPM) include mutations in BAP1, NF2, TP53, SETD2, along with deletions of CDKN2A and NF2. We present an exceptional case of a MPM with loss of BAP1, presence of a TP53 pathogenic variant, a homozygous deletion of CDKN2A and deletions of NF2, LATS2 and SETD2. An ALK gene rearrangement was detected by fluorescence in situ hybridization and confirmed by RNA-based next-generation sequencing. The co-occurrence of ALK gene fusions with the more common genetic alterations in CDKN2A, NF2 and BAP1 has, to our best knowledge, not yet been described in malignant mesothelioma. Furthermore, the finding of an ALK gene fusion could suggest a potential target for therapy in this subset of malignant mesotheliomas.

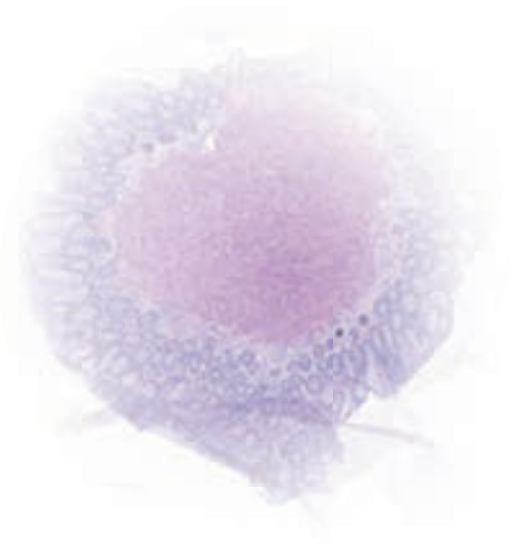
P 17

AN UNDIFFERENTIATED SARCOMA OF BONE WITH A ROUND TO EPITHELIOID CELL PHENOTYPE HARBORING A NOVEL EWSR1-SSX2 FUSION IDENTIFIED BY RNA-BASED NEXT-GENERATION SEQUENCING

F. Cordier[1], J. Van der Meulen[2,3], B. Van Gaever[1], L. Lapeire[3,4], G. Sys[3,5], J. Van Dorpe[1,3], D. Creytens[1,3]

1. Department of Pathology, Ghent University Hospital, Ghent University, Ghent, Belgium
2. Molecular Diagnostics Ghent University Hospital (MDG), Ghent University Hospital, Ghent University, Ghent, Belgium
3. CRIG, Cancer Research Institute Ghent, Ghent University Hospital, Ghent University, Ghent, Belgium
4. Department of Medical Oncology, Ghent University Hospital, Ghent University, Ghent, Belgium
5. Department of Traumatology and Orthopaedics, Ghent University Hospital, Ghent University, Ghent, Belgium

Due to the increased application of RNA-based next-generation sequencing techniques on bone and soft tissue round cell sarcomas new fusions are frequently found, leading to the introduction of new sarcoma entities such as CIC-rearranged sarcomas, sarcomas with BCOR genetic alterations and round cell sarcomas with EWSR1-non-ETS (in particular NFATC2 and PATZ1) fusions. These entities are now incorporated in the recent 2020 5th edition World Health Organization (WHO) Classification. Despite the significant advancement in the molecular classification of small round cell sarcomas of bone and soft tissue, pathologists are still facing cases with an undifferentiated phenotype that do not fit within the current WHO classification and remain 'unclassified'. In this case report we describe and discuss the finding of an undifferentiated sarcoma of the bone with a round to epithelioid cell phenotype harboring a novel EWSR1-SSX2 fusion. We discuss the histological and molecular findings. Treatment of this new bone tumor entity according to the Euro Ewing 2012 protocol led to complete pathologic response.



P 18
VASCULAR NEOPLASM IN A YOUNG GIRL: A WOLF IN SHEEP'S CLOTHING

A. Vandendriessche[1], P. Pauwels[1,2], V. Siozopoulou[1,2]

1. Department of Pathology, University Hospital of Antwerp, Belgium
2. Centrum for Oncological Research, University of Antwerp, Belgium

Background and objective

Angiosarcoma, a soft tissue tumor of endothelial cell origin, is a rare entity in adults and ever rarer in the paediatric population. Prognosis is poor and depends on the patient's age, tumor location, size, histological grade and extent of tumor progression.

Materials and methods

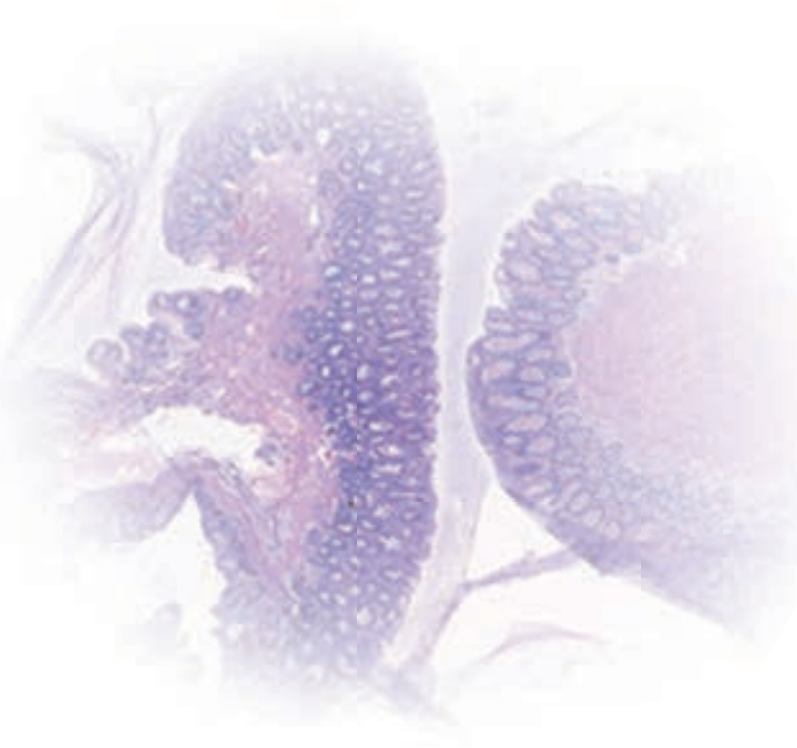
We present a rare case of metastatic angiosarcoma in a young girl. By the age of one, she presented with an osteolytic lesion on the scalp. The lesion was resected and examined histologically. Two years later, after presenting with severe iliac crest pain, a bilateral bone marrow biopsy was conducted. The unique histopathologic features of these two lesions were compared and analyzed. Molecular work-up (next generation sequencing) is in progress. Whole-body PET scan showed no other metastatic locations.

Results

Histology of the first lesion on the scalp showed a vascular tumor with no overt features of malignancy. However, no precise diagnosis could be made due to the odd morphology and immunohistochemical characteristics, and the descriptive diagnosis of cellular epithelioid vascular neoplasm was favored. For the second lesion, bilateral bone marrow biopsy shows complete replacement of marrow spaces by a highly malignant proliferation consistent with epithelioid angiosarcoma.

Conclusions

Although vascular neoplasms of bone are known to be multifocal, multifocality lies mainly in the same anatomic region. The combination of scalp and iliac crest is uncommon in that context, if not unseen. As a result, the tumor in the bone marrow is considered a metastatic site. Few reports in the English literature describe angiosarcoma in childhood. With this we describe an unusual case and peculiar initial presentation of epithelioid angiosarcoma, that may in the feature help in the differential diagnostic work-up of vascular neoplasms in children.



P 19

EX VIVO DERMOSCOPICAL IMAGE CLASSIFICATION WITH DEEP LEARNING FOR SKIN LESION-SPECIFIC PROCESSING

V. Clauwaert[1,2], M. Haspeslagh[1,2], E. Verhaeghe[2,3], B. Vankeirsbilck[3], T. Verbelen[3], L. Brochez[2,3,4]

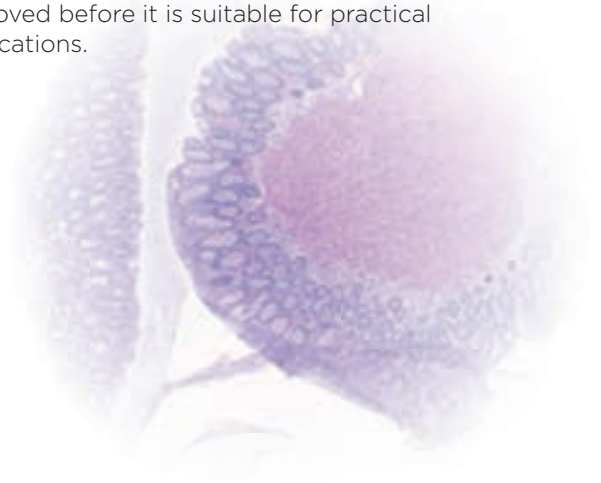
1. Dermpat, Ghent, Belgium
2. University Hospital of Ghent, Ghent, Belgium
3. University of Ghent, Ghent, Belgium
4. Cancer Research Institute Ghent, Ghent, Belgium

Skin cancer is rapidly increasing worldwide. Diagnostic accuracy and efficiency can be augmented by using ex vivo dermoscopy (EVD) with derm dotting (marking of diagnostical zones) to process skin biopsies in a lesion-specific, targeted way. Automating EVD skin tumour classification can make the methodology accessible, cost-efficient and standardized for lab technicians and pathologists. Therefore, the feasibility is evaluated of automating EVD image classification of skin tumours with convolutional neural networks (CNN).

Classification was evaluated on a dataset of 13729 images divided into 7 commonly accepted skin tumour classes. Highest sensitivities were obtained with a ResNet CNN architecture, optimization through stochastic gradient descent, a learning rate of 0.001, a batch size of 32, pretraining and fine-tuning of all layers. In this case, overall sensitivity was 74% and class-specific sensitivities ranged from 50% to 88% depending on sample size and intra-class homogeneity. Classification of these lesions into their respective processing strategy reached a sensitivity of 84%. Furthermore, the network separated malignant tumours from benign tumours with a sensitivity of 87% and an AUROC of 0.9.

Weighted sampling and data augmentation of minority classes through flipping (an adjustment to address class imbalance) increased overall sensitivity with 4% and precision with 5%. Fine-graining training classes into visually more distinct subtypes did not improve overall performance, but increased sensitivity for (possibly) malignant classes; with 2% for epitheliomas and with 4% for melanocytic lesions. The most important causes of misclassification were a lack of dermoscopical cues or incorrect/incomplete ground truth labelling. Thus, performance can be enhanced through data cleaning/standardizing efforts and by handling non-informative images as a separate class.

Our results indicate that EVD image classification of skin tumours is feasible, but should be further improved before it is suitable for practical applications.



P 20
RESULTS OF A SECOND BELGIAN PANTRK IHC RING TRIAL

K. De Winne[1], Karen Zwaenepoel[1], Laure Sorber[2], Suzan Lambin[1], Patrick Pauwels[1,2]

1. Laboratory of Pathological Anatomy, Antwerp University Hospital (UZA), Edegem, Belgium
2. Center for Oncological Research Antwerp (CORE), University of Antwerp (UAntwerp), Wilrijk, Belgium

Background

In 2019, a first ring trial was organised for a small number of labs to help them optimise and validate their own panTRK IHC test. However, general awareness and experience still remain insufficient. To make testing even more accessible, this second pan-TRK IHC ring trial aimed to involve more labs and hospitals.

Methods

Six selected cases were stained for TRK using the VENTANA pan-TRK Assay on Benchmark Ultra. Each of the 22 participants received two unstained slides and were asked to return one TRK–stained slide per case and to report the protocol used and their interpretation. Two sites participated with two different protocols. The stained slides were evaluated by two designated pathologists based on staining intensity, percentage of positive tumour cells and background staining.

Results

Compared to the Ventana pan-TRK reference method, 17 protocols achieved a sufficient mark. Seven labs were advised to further optimise the protocol. Two clones were used during the trial: A7H6R (Cell Signaling) and EPR17341 (concentrated form Abcam / ready-to-use assay Ventana). Eleven (46%) labs used the RTU clone, of which eight followed the manufacturer's instructions and achieved the optimal mark. Most labs used the Ventana platform (71%). The remaining labs used the Dako Omnis platform. About half of the protocols on both platforms received an optimal score.

Conclusions

TRK clone EPR17341, used by more than 90% of the participating labs, demonstrated high sensitivity and specificity for the detection of NTRK fusions. Only two labs used the A7H6R clone which, in contrast to the first ring trial, didn't score well: suboptimal staining intensity or a seemingly different specificity resulted in other cells to stain. Labs using the Ventana ready-to-use system based on the EPR17341 clone in combination with the recommended protocol settings scored best. Finally, Interpretation of panTRK IHC remains challenging, expecially in cases with physiological TRK expression.

P 21

APOPTOSIS AND PROLIFERATION IN UNINFECTED AND INFECTED STAPHYLOCOCCUS AUREUS RADIATION SKIN ULCER

M.S. Myroshnychenko[1], M.V. Krasnoselskyi[2], E.S. Pushkar[2], L.I. Simonova[2]

1. Kharkiv National Medical University, Kharkiv, Ukraine
2. State Organization «Grygoriev Institute for Medical Radiology and Oncology of the National Academy of Medical Sciences of Ukraine», Kharkiv, Ukraine

Background

Radiation skin ulcer (RSU) is often radiotherapy complications in malignant tumors patients. The objective is to identify the cells apoptosis, proliferation in uninfected, infected Staphylococcus aureus (SA) RSU.

Materials and methods

Rats were divided into 3 groups: 1 (G 1), included 5 unaffected rats; 2 (G 2), involved 25 rats with RSU in the hip area; 3 (G 3), consisted of 25 rats with infected SA RSU. Rats were withdrawn from the experiment on days 14, 30. Skin was the study material. Monoclonal antibodies to p53, Ki-67 were used.

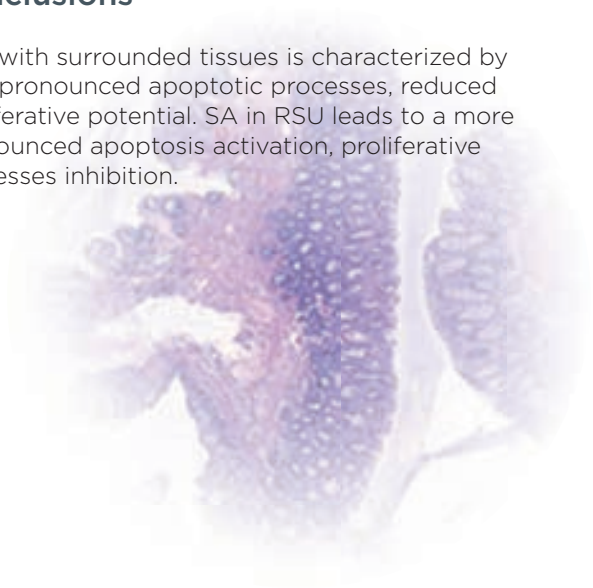
Results

In G 1 p53, Ki-67 were expressed by epidermis epithelial cells; skin appendages cells; immune cells, fibroblasts located in underlying tissues (dermis, hypodermis, muscle tissue); vascular endotheliocytes; muscle layer myocytes. In G 2, 3 on days 14, 30 p53, Ki-67 expressed similar to G 1 cells located in RSU marginal areas; on day 30 – immune cells, fibroblasts, vascular endotheliocytes of RSU bottom granulation tissue.

On day 14 in G 1, 2, 3 the number of p53-, Ki-67-positive cells in epidermis was 2.1±0.3 and 5.6±0.5, 5.9±0.4 and 3.2±0.5, 7.2±0.3 and 2.2±0.3; in underlying tissues – 0.9±0.1 and 3.1±0.4, 6.7±0.5 and 4.6±0.4, 9.8±0.5 and 3.4±0.2. On day 30 in G 2, 3 the number of p53-, Ki-67-positive cells in epidermis was 8.2±0.5 and 6.2±0.4, 12.8±0.6 and 3.1±0.2; in underlying tissues – 9.1±0.4 and 7.0±0.5, 14.6±0.5 and 4.3±0.4; in granulation tissue – 10.2±0.8 and 8.1±0.6, 15.7±0.6 and 5.5±0.7. On day 14 in G 2 and especially in G 3, compared with G 1 apoptotic activity increased, proliferative processes decreased. On day 30 in G 3 compared with G 2, it was more pronounced apoptosis, less pronounced proliferation.

Conclusions

RSU with surrounded tissues is characterized by cells pronounced apoptotic processes, reduced proliferative potential. SA in RSU leads to a more pronounced apoptosis activation, proliferative processes inhibition.



P 22

MACROPHAGE ACTIVITY IN THE KIDNEYS OF NEWBORNS, DEVELOPED UNDER MATERNAL PREECLAMPSIA CONDITIONS

M.S. Myroshnychenko[1], N.V. Kapustnyk[2], Yu.Ya. Fedulenkova[1], I.I. Torianyk[3], N.G. Popova[1], G.Ye. Khrystian[3], T.M. Moiseyenko[1]

1. Kharkiv National Medical University, Kharkiv, Ukraine
2. Public nonprofit organization of the Kharkiv District Council «Regional Clinical Perinatal Centre», Kharkiv, Ukraine
3. Mechnikov Institute of Microbiology and Immunology of the National Academy of Medical Sciences of Ukraine, Kharkiv, Ukraine

Background

Preeclampsia (PE) is a common pregnancy complication, negatively affecting the health status of a woman and her baby.
The objective is to identify the features of macrophage activity in the kidneys of newborns from mothers whose pregnancy was complicated by PE.

Materials and methods

The studied material was the kidneys of full-term newborns. There were 4 groups formed. Group 1 included 15 newborns from mothers with physiological pregnancy. Group 2 was represented by 13 newborns from mothers whose pregnancy was complicated by mild PE. Group 3 consisted of 14 newborns, developed in conditions of moderate severity maternal PE. Group 4 included 13 newborns, developed in conditions of severe maternal PE. Microspecimens were stained with hematoxylin and eosin. Immunohistochemical study was performed with a monoclonal antibody to CD 68.

Results

In group 1 a few CD 68-cells were identified in the fat capsule, in the stroma of the cortical and medulla in the intertubular, peritubular, perivascular and periglomerular areas. The absolute number of CD 68-cells was 6.77±0.20. In groups 2-4 CD 68-cells were located not only in similar sites of group 1 but also in areas of sclerosis, around immature glomeruli and tubules, glomerular and tubular cysts. The absolute number of CD 68-cells increased ($p<0.05$) in the direction from group 2 to group 4 and amounted to 10.63±0.25 in group 2, 11.67±0.23 in group 3, and in group 4 15.46±0.27. In groups 2-4, the number of CD 68-cells increased ($p<0.05$) compared to group 1.

Conclusions

Maternal PE leads to activation of the macrophage system in the kidneys of newborns, indicating both increased needs for these cells for damaged structures phagocytosis, and their possible participation in the morphogenesis of sclerosis, cyst formation, delayed processes of glomerulogenesis and tubulogenesis.

P 23

THE IMPACT OF THE COVID-19 PANDEMIC AND THE ASSOCIATED GOVERNMENTAL MEASURES ON A BELGIAN ACADEMIC LABORATORY FOR SURGICAL PATHOLOGY AND CYTOPATHOLOGY

Marie-Caroline de Pelsemaeker[1], Yves Guiot[1], Jonathan Vanderveken[1], Christine Galant[1,2], Mieke R. Van Bockstal[1,2*].*

** Both authors contributed equally*
1. Department of Pathology, Cliniques universitaires Saint-Luc Bruxelles, Avenue Hippocrate 10, 1200 Brussels, Belgium
2. Institut de Recherche Expérimentale et Clinique, Université Catholique de Louvain, Avenue Hippocrate 10, 1200 Brussels, Belgium

Aims

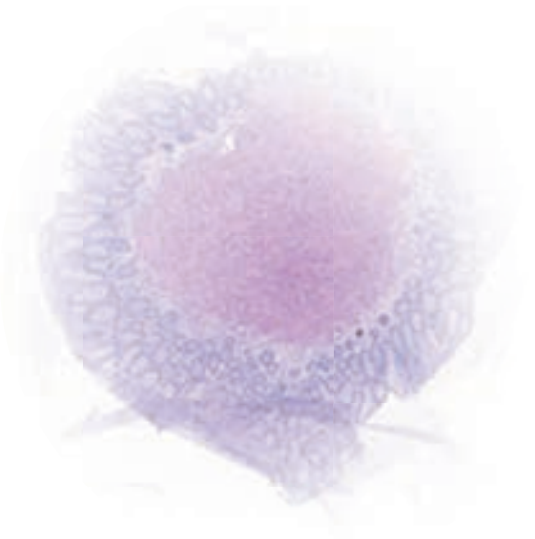
In cancer Epithelial-to-Mesenchymal Transition (EMT) is associated with tumorigenesis, stemness, invasion, metastasis, and resistance to therapy. Recent data suggest that EMT should be a stepwise process with distinct intermediate hybrid states characterized by co-expression of epithelial and mesenchymal markers. These data come mainly from in vitro cell cultures and some animal models. In contrast, the present study aims to evaluate the prognostic value of hybrid EMT states for patients with urothelial carcinomas.

Methods and results

Data on monthly numbers of histological and cytological samples, as well as immunohistochemistry and molecular tests, were extracted from the laboratory information management system. We observed a substantial reduction in the total number of histopathological and cytological samples, accompanied by a less pronounced but yet significant decrease of the number of tissue blocks and glass slides. The impact on oncology-related surgical procedures was rather limited. However, the anti-Covid-19 measures significantly diminished all screening-related samples, such as colon biopsies, breast biopsies and cervical cytology, and strongly reduced the number of samples related to 'functional' pathology, such as thyroidectomies and gastric biopsies.

Conclusions

Since many different health care interventions are reflected in the workload of a pathology laboratory, this study enabled us to identify areas for 'deconfinement' health care actions. Our findings indicate that all areas in medicine were affected, but the impact seemed largest for cancer screening programmes. Health care professionals should assure that consultations related to cancer screening are postponed instead of cancelled.



P 24

PROSTATIC METAPLASIA IN FEMALE-TO-MALE TRANSGENDER INDIVIDUALS

H. Van Beveren[1], G. T'Sjoen[2], S. Weyers[3], J. Van Dorpe[1], K. Van de Vijver[1]

1. Department of pathology, Ghent University Hospital, Ghent, Belgium
2. Department of Endocrinology and Center for Sexology and Gender, Ghent University Hospital, Ghent, Belgium
3. Department of Gynaecology and Obstetrics, Ghent University Hospital, Ghent, Belgium

Background

Histological changes in the genital tract of female-to-male transgenders have been described in the past. However, not much research is done on histological changes of vaginal tissue and the amount of patients in previous studies was generally low. Anderson et al. recently described a new phenomenon in vaginal and cervical tissue which they stated as "prostatic metaplasia". The aim of this study is to provide data on a larger scale on the description and prevalence rate of histological changes and lesions in the vagina and ectocervix of female-to-male transgenders with special attention to "prostatic metaplasia", including an immunohistochemical profile of this lesion and its differentials.

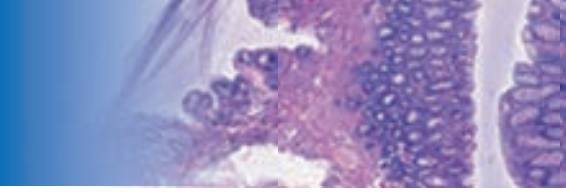
Methods and Results

All H&E slides and immunohistochemically stained slides of 140 vulvovaginectomies with 72 accompanying uteri were reviewed. Transitional cell metaplasia was seen in vaginal (96.5%) and ectocervical (85%) tissue. Prostatic metaplasia was observed in vaginal (92%) and ectocervical (82%) tissue. Prostatic metaplasia depicts a spectrum with increasing maturation ranging from a) intracellular changes in the basal layer of the epithelium, b) prostatic type glands located within the epithelium or at the basal layer of the epithelium, c) glands with a luminal and basal layer that have invaginated into the subepithelial lamina propria and/or d) fully developed glands in the lamina propria resembling (ectopic) prostate tissue. The basal layer of the metaplastic epithelium stained with NKX3.1. The glands stained with NKX3.1, prostate-specific antigen (PSA), prostate specific acid phosphatase (PSAP), androgen receptor (AR), cytokeratin 7 (CK7), cytokeratin 8-18 (CK8-18), cytokeratin 17 (CK17), cytokeratin 19 (CK19), EpCAM and also CD10 for glands in the lamina propria resembling (ectopic) prostate tissue. Additionally other structures such as Bartholin glands (6%), ectopic breast tissue (4%), mucous cysts (4%) and mesonephric duct remnants (4%) were encountered. Bartholin glands showed positive staining with NKX3.1, but not with PSA and PSAP. Ectopic breast tissue, mucous cysts and mesonephric duct remnants were negative with NKX3.1, PSA and PSAP.

Discussion

We believe that this metaplastic process is caused under the influence of androgens. We also believe that, since prostatic metaplasia is frequently encountered, it was often overlooked in the past. The mechanism and clinical impact of prostatic metaplasia are not yet clear. This study will raise awareness towards this metaplastic phenomenon among pathologists which will be fundamental for future research on further insights and clinical correlation of this lesion.

NOTES



FRIDAY

SATURDAY

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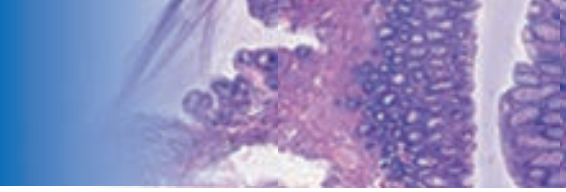

FRIDAY

SATURDAY

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