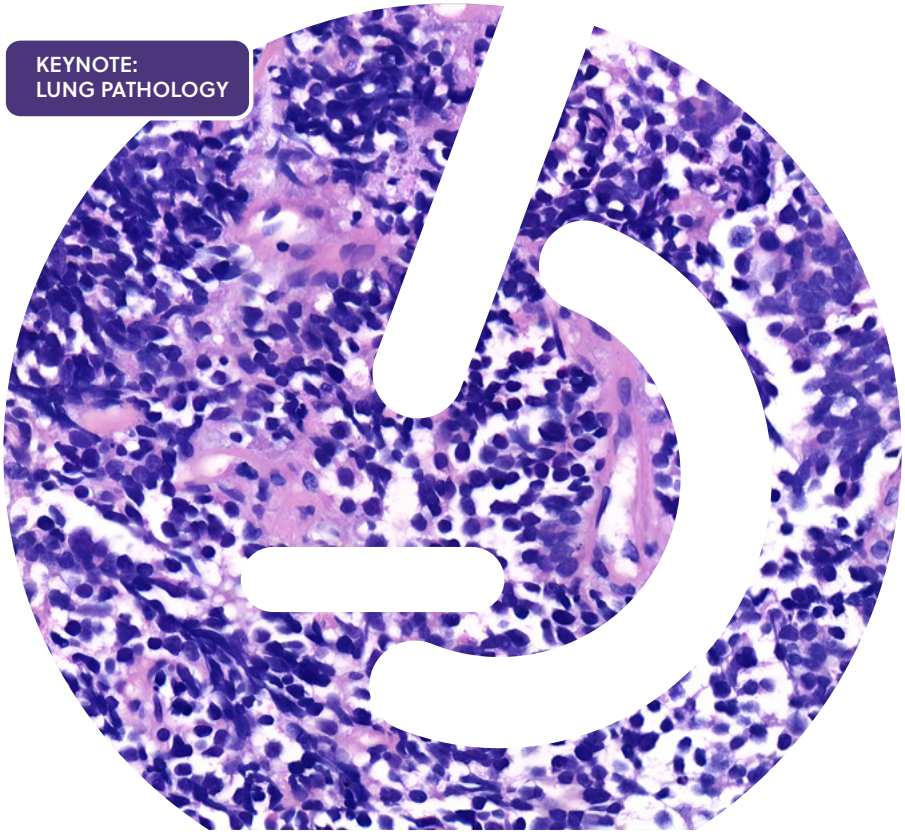
BWP Belgian Week of Pathology

15th BELGIAN WEEK OF PATHOLOGY

23.01 > 24.01.26

@ TANGLA HOTEL



With the courtesy of Philippe DELVENNE (ULiège)

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WELCOME

Welcome to BWP 2026: The 15th Belgian Week of Pathology

January 23–24, 2026 | Tangla Hotel, Brussels

After a vibrant joint meeting at Ghent Pathology in 2025, the BWP returns to its roots! We are delighted to welcome the Belgian pathology community back to our flagship annual congress—a premier environment for knowledge exchange, professional growth, and collaborative spirit.

Highlights of the 15th Edition:

- **Keynote Excellence**: Prof. Sylvie Lantuejoul (France) presents: «*Recent developments in Small Cell Lung Carcinoma.*»
- **A Global & Local Faculty**: We are honored to host an exceptional lineup of international experts from the USA, UK, Italy, Germany, The Netherlands, France, and Turkey. We are also proud to feature more than 20 distinguished national speakers, showcasing the high level of expertise within our own Belgian community.
- **Comprehensive Science**: A diverse program featuring nearly all BSP working groups and a dedicated session for our Young Pathologists.
- **Celebrating Research**: High-quality oral and poster presentations with prizes for both scientific research and challenging diagnostic cases.
- **Community & Connection**: Reunite with colleagues during the Friday Evening Congress Dinner, the social highlight of our calendar.

Finally, we extend our gratitude to our **industry partners** for their continued support, and to you—**our attendees**—for your dedication to advancing patient care through pathology.

Enjoy the Congress!

The organizing committee,

Vasiliki Siozopoulou and *Koen Van de Vijver*

BWP Belgian Week of Pathology

Accreditation

Accreditation has been requested with the INAMI/RIZIV for ethics and economy as well as anatomo-pathology.

Submission is done on the computers available in the exhibition area.

Submission is requested once a day on Friday, and only once on Saturday. For ethics and economy a physical signature will be additionally asked at the beginning of the session.

Language

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

Abstracts

Authors were invited to submit abstracts until November 30th 2025

The result of evaluation was sent to the first authors during the month of December 2025.

- 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 January 23 and Saturday January 24

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

The Belgian Week of Pathology and the Belgian Society of Pathology will award:

- Two best Oral Presentations: (500€ each)
- Two best Poster Presentations: (500€ each)

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 **or** the Q-Parc Esplanade
- along Avenue Mounier with time restriction,
  the Parking disc is mandatory.

Event Coordinator

Vivactis/Medisquare

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MDEON Visa

25/V2/13530/010663.

FRIDAY January 23
19.00-23.00

Cocktail Reception and Dinner

at Tangla Hotel

TANGLA

BRUSSELS

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www.belgian-society-pathology.eu

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VERSET Laurine	ULB

FRIDAY 23/01

■ ROYAL 2 & 3
■ ROYAL 1

08.00-09.00	WELCOME
09.00-10.30	■ **HEAD & NECK** Working Group ■ **DIGITIZATION** Working Group
10:30-11:15	COFFEE BREAK & POSTER TOUR
11:15-12:45	■ **GYNECOPATHOLOGY** Working Group ■ **NEUROPATHOLOGY** Working Group
12:45-14:00	LUNCH & POSTER TOUR
14:00-15:30	■ **UROPATHOLOGY** Working Group
15:30-16:15	COFFEE BREAK & POSTER TOUR
15:40-16:10	**BMS** Round table on PD-L1 scoring in advanced melanoma
16:15-17:30	■ **HAEMATOPATHOLOGY** Working Group ■ **JOINT MEETING DERMATO- & BREAST PATHOLOGY** Working Group
18:00-19:00	■ KEYNOTE LECTURE: **LUNG CANCER**
19:00-19:30	Cocktail Reception
19:30-23:00	Dinner

SATURDAY 24/01

■ ROYAL 2 & 3
■ ROYAL 1

08.00-09.00 WELCOME

09.00-10.30 ■ CYTOPATHOLOGY
■ JOINT MEETING
YOUNG PATHOLOGISTS & MOLECULAR
Working Group

10:30-11:15 COFFEE BREAK & POSTER TOUR

11:15-12:45 ■ CYTOPATHOLOGY
■ MOLECULAR PATHOLOGY Working Group

12:45-14:00 LUNCH & POSTER TOUR

14:00-16:00 ■ SURGICAL PATHOLOGY
■ DIGESTIVE PATHOLOGY Working Group

16:00-16:15 Awards Ceremony

Closing Ceremony BWP 2026

HOLOGIC®

08:00-09:00 WELCOME

ROYAL 2 & 3

09:00-10:30 **HEAD & NECK** Working Group
Moderators: Prof. Senada Koljenović, Prof. Esther Hauben

09:00-09:25 **Practical approach to salivary gland tumors**
Dr. Ignace Dalle (AZ Sint-Lucas Brugge)

09:25-09:50 **Role of RNA sequencing in salivary gland tumours**
Prof. Esther Hauben (UZ Leuven)

09:50-10:20 **Standardized reporting of salivary gland tumors (and other H&N sites) in Belgium**
Dr. Sarah Van Praet (Belgian Cancer Registry)

10:20-10:30 • **OP: The Hidden Mass Behind the Drum: 2 cases of Middle Ear Neuroendocrine Tumor**
Dr. Amber Lievens (AZ Sint-Jan Brugge)

ROYAL 1

09:00-10:30 **DIGITIZATION** Working group
Moderator: Prof. Amélie Dendooven, Dr. Frederik Deman

9:00-9:10 **Welcome to the session and introduction on scope of the working group of digitization**
Dr. Frederik Deman (ZAS)

9:10-9:35 **What is SNOMED CT and what is the relevance for pathology?**
Dr. Stefan Dubois (UZ Antwerpen)

9:35-10:00 **BSP working group on digital pathology current projects and perspectives**
Prof. Amélie Dendooven (UZ Gent)

10:00-10:30 **Use case on workload measurement and assignment**
Dr. Franceska Dedeurwaerdere (AZ Delta)

10:30-11:15 COFFEE BREAK & **POSTER TOUR**


Roche

ROYAL 2 & 3

11:15-12:45 **GYNECOPATHOLOGY** Working Group
Moderators: Dr. Claire Bourgain, Prof. Cecile Colpaert

11:15-12:35 **Indication for placenta examination and legal aspects**
Prof. Drucilla Roberts (Cambridge, USA)

12:35-12:45 • **OP: Additional sectioning reveals underdiagnosis of serous (pre)malignancies in fallopian tube specimen**
Dr. A.B. Bouwmeester (Nijmegen, The Netherlands)

ROYAL 1

11:15-12:45 **NEUROPATHOLOGY** Working group
Moderators: Prof. Dietmar Thal, Prof. Laetitia Lebrun

11:15-11:45 **Diagnosis of neuroinflammatory conditions in brain biopsies**
Prof. Christine Stadelmann-Nessler (Göttingen, Germany)

11:45-12:15 **Current and future perspective on the role of molecular techniques in the neuropathological diagnosis of gliomas**
Prof. Philipp Sievers (Heidelberg, Germany)

12:15-12:45 **Case presentation, members of the working group**
Dr. Lukas Marcelis (UZ Leuven), Dr. Melek Ahmed (AZ Sint-Jan Brugge), Stefanie Brock (UZ Brussel)

12:45-14:00 LUNCH & **POSTER TOUR**

NOTES

ROYAL 2 & 3

14:00-15:30 **UROPATHOLOGY** Working Group
Moderators: Moderators: Dr. Maria-Dolores (Lola) Martin Martinez, Prof. Marcella Baldewijns

Testis lectures on GCT and SCT

14:00-14:25 Prof. Acosta, Andres Martin (Indianapolis, USA)

14:25-14:50 Prof. Berney, Daniel (London, UK)

14:50-15:20 **30 minutes practical cases**
Members of the working group

15:20-15:30 • **OP: Sertoli–Leydig Cell Tumor presenting with Ascites and Pleural Effusion: a diagnostic challenge**
Dr. Júlia Azevedo (IPO-Porto, Portugal)

15:30-16:15 COFFEE BREAK & **POSTER TOUR**

16:15-17:45 **HAEMATOPATHOLOGY** Working Group
Moderators: Dr. Kristof Cokelaere

16:15-17:35 **Beautiful lymphomas cases in the lung and mediastinum**
Presenters: Prof. T. Tousseyn (UZ Leuven), Dr. P. Collins (CHULiege), Dr. J. Somja (CHULiege), Dr. P. De Paepe (AZ Sint-Jan Brugge)

17:35-17:45 • **OP: Post-transplant lymphoproliferative disorders: clinico-pathological correlations**
Dr. Ahmad Chebbo (CUSL)

INVITATION

Your case, **our discussion**

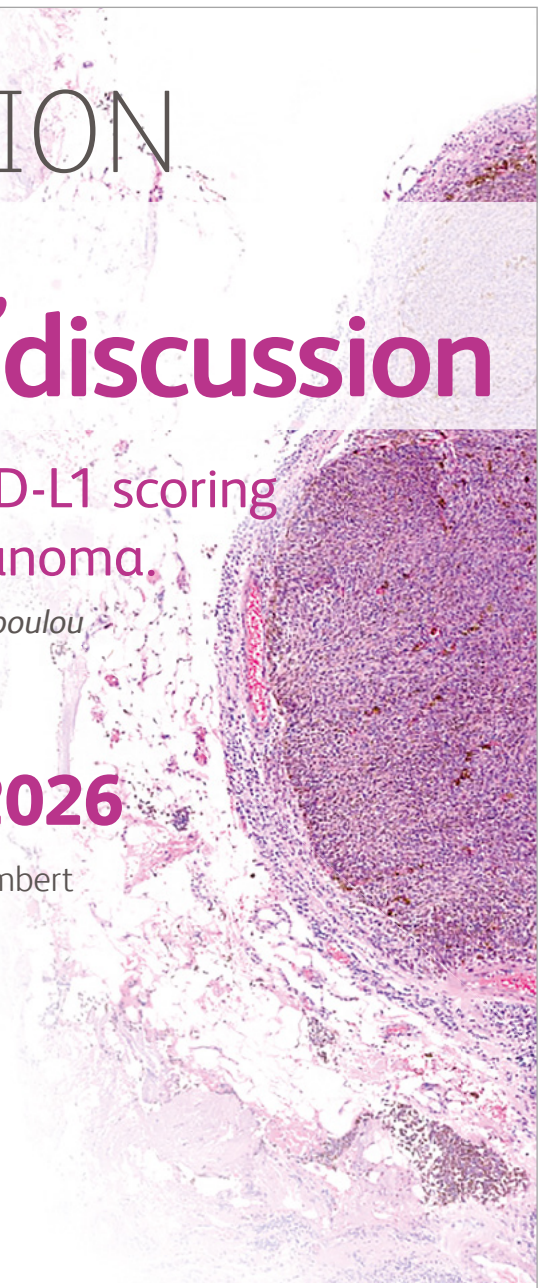
Round table on PD-L1 scoring in advanced melanoma.

Moderator: Prof. Vasiliki Siozopoulou

Friday 23 January 2026

Tangla Hotel - Woluwe St Lambert
15:40 - 16:10
Room: Royal 1

Coffee and refreshments will be served during the meeting



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ROYAL 1

15:40-16:10 **BMS**
Round table on PD-L1 scoring in advanced melanoma
Moderator: Prof. Vasiliki Siozopoulou

16:15-17:45 **JOINT MEETING DERMATO- & BREAST PATHOLOGY**
Working group
Moderators: Prof. Francesca Bosisio, Prof. Mieke Van Bockstal

16:15-17:05 **Update on nipple lesions of the breast**
Prof. Cristian Scatena (Pisa, Italy)

17:05-17:35 **Case presentation**
Dr. David Zylberberg (CUSL), Prof. Francesca Bosisio (UZ Leuven)

17:35-17:45 • **OP: Flowchart to reduce sentinel lymph node procedures after biopsy-diagnosed ductal carcinoma in situ**
Dr. Julia A.M. Riggi (CUSL)

ROYAL 2 & 3

18:00-19:00 **KEYNOTE LECTURE: LUNG CANCER**
Moderator: Prof. Philippe Delvenne

Recent developments in Small Cell Lung Carcinoma
Prof. Sylvie Lantuejoul (Lyon / Grenoble, France)

19:00-19:30 COCKTAIL RECEPTION

19:30-23:00 DINNER


Telemis

08:00-09:00 WELCOME

ROYAL 2 & 3

09:00-10:30 **CYTOPATHOLOGY**
Moderators: Dr. Shaira Sahebali, Dr. Claire Bourgain

09:00-09:45 **ASC-US study by the EFCS**
Dr. Claire Bourgain (Imelda Ziekenhuis)

09:45-10:30 **The New Cervical Screening Program: First Beginnings**
Dr. Koen Van Herck (Belgian Cancer Registry)

ROYAL 1

09:00-10:30 **JOINT MEETING YOUNG PATHOLOGISTS & MOLECULAR** Working group
Moderators: Prof. Nicky D'Haene, Dr. Loic Duchene

09:00-10:20 **Molecular insight of uterine carcinoma**
Prof. Koen Van de Vijver (UZ Ghent)

10:20-10:30 • **OP: Your sclerosing epithelioid fibrosarcoma refuses to stain for MUC4? Check for the YAP1-KMT2A fusion!**
Dr. Nathalie Mengeot (CUSL)

10:30-11:15 COFFEE BREAK & **POSTER TOUR**


AstraZeneca

ROYAL 2 & 3

11:15-12:45 **CYTOPATHOLOGY**
Moderators: Dr. Shaira Sahebali, Dr. Claire Bourgain

Focus on cytology exams by IAC

Case discussion (GYN)
Prof. Lia van Zuylen (Nijmegen, The Netherlands),
Dr. Anouk Bouwmeester (Nijmegen, The Netheands) and
Dr. Shaira Sahebali (UZ Brussels)

ROYAL 1

11:15-12:45 **MOLECULAR PATHOLOGY** Working group
Moderators: Prof. Nicky D'Haene, Dr. Franceska Dedeurwaerdere

11:15-11:55 **Liquid Biopsy in Thoracic Pathlogy, Today and Tomorrow**
Prof. Paul Hofman (Nice, France)

11:55-12:35 **Resistancy mechanisms in NSCLC**
Prof. Christophe Dooms (UZ Leuven)

12:35-12:45 • **OP: Identifying predictive biomarkers in Early Breast Cancer: A Focus on T-cell functional profiling**
Dr. Charlotte Birchall (UCLouvain / LTO, Gosselies)

12:45-14:00 LUNCH & **POSTER TOUR**

NOTES

ROYAL 2 & 3

14:00-16:00 **SURGICAL PATHOLOGY**
Moderators: Prof. Phillipe Delvenne, Prof. Ramses Forsyth

14:00-14:40 **Adult autopsy investigation of sudden death cases: a correct methodology is crucial for secondary prevention**
Prof. Giulia d'Amati (Rome, Italy)

14:40-15:20 **The role of molecular autopsy in sudden cardiovascular death cases**
Prof. Monica De Gaspari (Padova, Italy)

15:20-16:00 **Autopsy neuropathology across the lifespan: age-dependent disorders of the nervous system**
Dr. Stefanie Brock (UZ Brussel)

ROYAL 1

14:00-16:00 **DIGESTIVE PATHOLOGY** Working Group
Moderators: Prof. Anne Hoorens, Dr. Hélène Dano

14:00 – 14:25 **Non-IBD colitis**
Laurine Verset (ULB)

14:25 – 14:50 **IBD-colitis**
Pamela Baldin (CUSL)

14:50 – 15:20 **Nonconventional dysplasia in IBD**
Prof. Dr. Ensari (Ankara, Turkey)

15:20 – 15:50 **Colitis-associated cancer**
Prof. Dr. M Scvrek (Paris, France)

15:50-16:00 • **OP: SARIFA in colon cancer: prognostic limitations and biological perspectives**
Dr. Jennifer Fallas (ULB)

16:00-16:15 **AWARDS CEREMONY**

CLOSING CEREMONY BWP 2026

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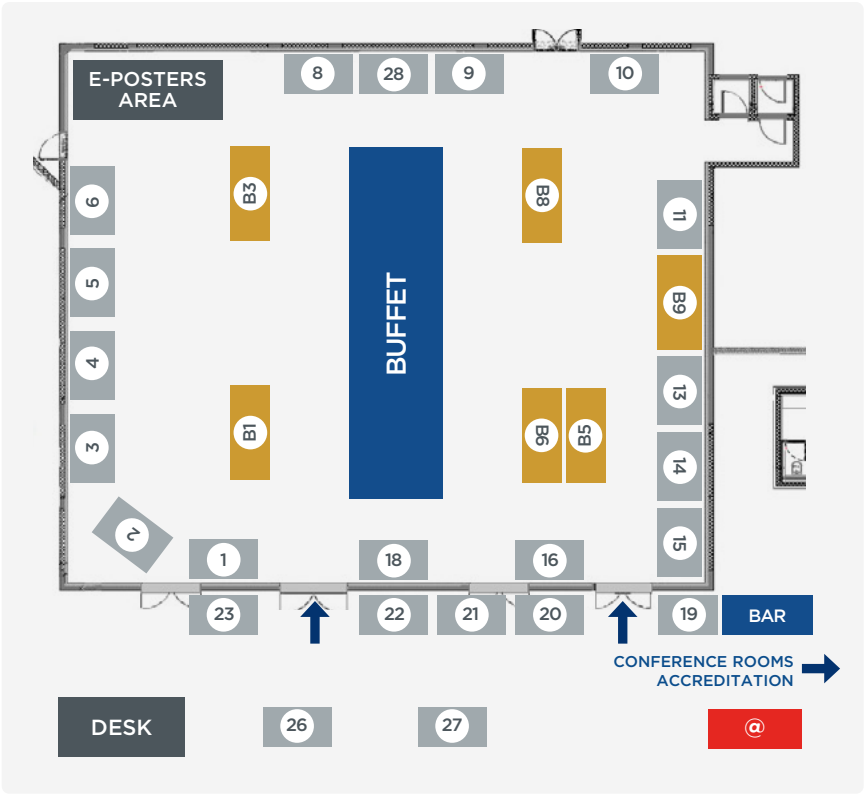

GOLD

B1	HOLOGIC
B3	TELEMIS
B5	BMS
B6	ROCHE Diagnostics
B8	ASTRA ZENECA

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ORAL FREE PAPERS

OF 01

SERTOLI–LEYDIG CELL TUMOR PRESENTING WITH ASCITES AND PLEURAL EFFUSION: A DIAGNOSTIC CHALLENGE

Júlia Azevedo[1], Sigi Hendrickx[2], Pieter De Visschere[2], Hannelore Denys[3], Rawand Sahili[4], Philippe Tummers[4], Jo Van Dorpe[5], Koen Van de Vijver[5]

1 Department of Pathology, Portuguese Oncology Institute of Porto (IPO-Porto), Portugal
2 Department of Radiology, Ghent University Hospital, Ghent, Belgium
3 Department of Oncology, Ghent University Hospital, Ghent, Belgium
4 Department of Gynecology, Ghent University Hospital, Ghent, Belgium
5 Department of Pathology, Ghent University Hospital, Ghent, Belgium

Background

Meigs' syndrome is a rare clinical condition defined by the triad of a benign gynecological tumor, ascites, and pleural effusion, resolving after tumor removal. Pseudo-Meigs' syndrome presents the same features, but is associated with other tumors, including malignant ovarian neoplasms, teratomas and uterine leiomyomas; its association with Sertoli-Leydig cell tumors is extremely uncommon, with few reported cases.

Materials and methods

We present the case of a 54-year-old woman with bilateral pleural effusion, prominent ascites, bilateral large solid–cystic ovarian lesions and multiple lymphadenopathies. Repeated ascitic fluid and lymph node analyses revealed scant reactive cells without malignancy. Progressive anasarca and persistent ascites with a weight gain of 15kg, prompted surgical intervention.

Results

Both ovaries were hemorrhagic with grey-soft areas measuring up to 13 cm. Histologically, was seen a multilobulated neoplasm with alternating hypo-/hypercellular areas, composed of solid sheets and occasional "follicle-like spaces". Tumor cells were oval to spindle-shaped with vesicular nuclei, scant cytoplasm and moderate atypia with frequent mitoses, admixed with a second cell population of round, centrally located nuclei, prominent nucleoli, and vacuolated cells. Immunohistochemistry showed positivity for SF1, FOXL2, inhibin and calretinin. The second cell population stained positive for Melan-A. NGS analysis revealed no alterations, consistent with DICER1/FOXL2-wildtype subtype. The final diagnosis of bilateral, intermediately differentiated Sertoli-Leydig cell tumor was made.

Conclusions

The pathophysiology of effusions in Pseudo-Meigs' syndrome is multifactorial, involving neoplasm-related transudate, lymphatic/vascular obstruction, peritoneal irritation, and cytokine-induced vascular permeability. Clinical features often mimic advanced malignancy, posing diagnostic challenges, leading to misclassification and potentially inappropriate management. Early recognition is crucial for patients with hydrothorax, ascites and pelvic/ovarian mass, since surgical excision achieves resolution of effusions and excellent prognosis.

OF 02

IDENTIFYING PREDICTIVE BIOMARKERS IN EARLY BREAST CANCER: A FOCUS ON T-CELL FUNCTIONAL PROFILING

Charlotte Birchall[1-2], Caroline Bouzin[5], Aurélie Daumerie[5], Jérôme Ambroise[4], Isabelle Bar[2], Sandy Haussy[2], Déborah Petrone[2], Alix Devaux[3], Ahmad Merhi[2], Maxime Devos[2], Javier Carrasco[2-3], Francois P Duhoux[6]

1 IREC, Cliniques Universitaires Saint-Luc (UCLouvain Saint-Luc), Woluwe-Saint-Lambert, Belgium
2 Laboratory of Translational Oncology GHdC/IPG (LTO), Gosselies, Belgium
3 Medical Oncology Department, Grand Hopital de Charleroi (GHdC), Charleroi, Belgium
4 Centre de technologies moléculaires appliquées (CTMA), Woluwe-Saint-Lambert, Belgium
5 IREC, Imaging Platform (2IP), Woluwe-Saint-Lambert, Belgium
6 Medical Oncology department, Cliniques Universitaires Saint-Luc (UCLouvain Saint-Luc), Woluwe-Saint-Lambert, Belgium)

Background and objective

Adding immune checkpoint inhibitors (ICI) to neoadjuvant chemotherapy improves outcomes in triple-negative (TN) and luminal B HER2-negative (LumB) early breast cancers. However, ICIs induce substantial toxicity, and no validated predictive biomarkers exist. Within the B-IMMUNE trial (59 TN/LumB patients), tumor and blood samples were collected at baseline (W0), mid-treatment (W12) and surgery (W24) to investigate why only a subset of tumors responds to chemotherapy-immunotherapy.
We hypothesize that pre-existing or treatment-induced immunogenicity—especially adaptive T-cell activation—drives clinical benefit.
To address this, we evaluate CD4+ and CD8+ T-cell responses using multiplex immunofluorescence (mIF) to quantify T-cell functional profiling.

Materials and methods

Breast cancer and tonsil cryosections (5 μm) were mounted, fixed and paraffin-embedded. mIF was performed using 5–6 iterative staining cycles targeting T-cell infiltration, activation, proliferation and differentiation markers (CD3, CD8, CD69, Ki67, CD45RO, CD45RA, CD62L, CD103). Staining relied on primary antibodies, HRP-conjugated secondaries and tyramide amplification. Slides were counterstained with Hoechst and mounted. Pathological response was assessed using the RCB index: responders (RCB 0–I) vs non-responders (RCB II–III).

Results

Baseline tumor analysis showed that T-cell infiltration is associated with pathological response in LumB tumors but not in TNBC, and this was also true for both CD8+ and CD4+ T-cells. Early activation of T-cells (CD69+) was not associated with improved response, whereas a combination of activation (CD69+) and proliferation (Ki67+) appears to be associated with a pathological response for LumB tumors.

Conclusions

Preliminary data suggest that infiltration by activated and proliferating T cells is associated with improved pathological response in LumB tumors. Further analysis is needed to understand whether these T-cells contribute to an anti-tumor response.

OF 03

ADDITIONAL SECTIONING REVEALS UNDERDIAGNOSIS OF SEROUS (PRE)MALIGNANCIES IN FALLOPIAN TUBE SPECIMEN

A.B. Bouwmeester[1], T.A. Gootzen[2], M. Nobbenhuis[3], V. Rudaitis[4], C.B. van den Berg[5], H. van Beekhuizen[5], L.S.M. Alcalá[6], H.P.M. Smedts[7], N.Visser[8], V. Verhoef [9], G.N. Jonges[10], R.P. Zweemer[11], J.A. de Hullu[2], J.A.W.M. van der Laak[1], M.P. Steenbeek[2*], M. Simons[1*]

* Shared last author
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4 Department of Gynaecology and Obstetrics, Vilnius university hospital Santaros klinikos, Vilnius, Lithuania
5 Department of Gynaecology and Obstetrics, ErasmusMC, Rotterdam, The Netherlands
6 Department of Pathology, Amphia hospital, Breda, The Netherlands
7 Department of Gynaecology and Obstetrics, Amphia hospital, Breda, The Netherlands
8 Department of Pathology, PAMM, Eindhoven, The Netherlands
9 Department of Gynaecology and Obstetrics, Maxima medical center; Veldhoven, The Netherlands
10 Department of Pathology, University Medical Center Utrecht, Utrecht, The Netherlands
11 Department of Gynaecology, University Medical Center Utrecht, Utrecht, The Netherlands

Background

The pathophysiology of peritoneal high grade serous carcinoma (pHGSC) is still poorly understood. As the presumed precursor lesion, serous tubal intraepithelial carcinoma (STIC) has a mean size of 1.5 mm, sampling error could be a problem.

Materials and methods

Four groups of patients were included: 1) STIC without metachronous pHGSC (n =4); 2) STIC with metachronous pHGSC (n=4); 3) no STIC, without pHGSC (n=5); 4) without STIC, with metachronous pHGSC (n=5). Follow-up time was 10 years or until development of pHGSC. All fallopian tube tissue of the RRSO was serially sectioned at 150 μm levels and manually reviewed. Subsequently a deep learning model was used to support STIC detection.

Results

A focus of STIC or HGSC was found in the deeper sections in all patients who developed pHGSC after RRSO whilst not having STIC or HGSC in the original section. One of the women with STIC in the original sections displayed invasive carcinoma in the deeper sections, however this patient did not develop pHGSC. Four STICs transitioned towards serous tubal intraepithelial lesion (STIL) at one of the sides of the lesion based on Ki-67 positivity.

Conclusions

These findings underscore the premise that pHGSC originates in the fallopian tube, and they question invasiveness as part of disease progression from STIC towards pHGSC. Furthermore, they raise the question if the amount of ki-67 positivity should be decisive in STIC diagnosis and if deeper sections should be performed if a STIL is found.

OF 04

POST-TRANSPLANT LYMPHOPROLIFERATIVE DISORDERS: CLINICO-PATHOLOGICAL CORRELATIONS

Ahmad Chebbo[1], Pamela Baldin[1], Eric Van Den Neste[2], Alessandra Camboni[1]

1 Departement of Pathology, Cliniques universitaires de Saint-Luc/UCLouvain, Brussel, Belgium
2 Department of Hematology, Cliniques Universitaires Saint-Luc, Brussels, Belgium

Background

Post-transplant lymphoproliferative disorders (PTLDs) are rare but serious complications of solid organ transplantation. This study evaluates the prognostic impact of clinical and pathological factors, with a focus on EBV status, in a cohort of PTLD patients from our institution.

Materials and methods

Forty-six PTLD patients hospitalized at Saint-Luc University Hospital over the last ten years were included. Cases not meeting the WHO diagnostic criteria were excluded. All biopsies were reviewed by certified pathologists. Clinical data, EBV serostatus, EBER in situ hybridization, histological subtype, and disease distribution were collected. Patients were followed for outcomes, including overall survival, to assess the prognostic relevance of selected variables.

Results

No statistically significant difference in overall survival was observed between patients with EBER-positive and EBER-negative PTLD. Age at transplantation and time from transplantation to PTLD onset were significantly lower in EBER-positive patients ($p = 0.003$ and $p = 0.0005$, respectively). No significant associations were found for sex, DLBCL subtype, or nodal versus extranodal presentation. Using an alternative two-year threshold to distinguish early from late PTLD, EBER positivity was significantly associated with early-onset disease. No correlation was observed between EBV serostatus at transplantation and overall survival.

Conclusions

In this cohort of 46 PTLD patients, EBV status—both serological and EBER-based—did not influence overall survival. However, EBER positivity was associated with younger age at transplantation, shorter latency to PTLD onset, and earlier disease presentation. Other clinical and pathological variables showed no significant prognostic impact.

OF 05

SARIFA IN COLON CANCER: PROGNOSTIC LIMITATIONS AND BIOLOGICAL PERSPECTIVES

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Background

Colon cancer is highly prevalent, yet prognostic stratification remains challenging. Stroma AReactive Invasion Front Areas (SARIFA), defined by tumor-adipocyte contact without intervening stromal reaction, is a promising morphologic marker of adverse prognosis, recently reported by at least two centers.

Materials and methods

This retrospective single-center study included 414 patients with resected colon adenocarcinoma between 2010 and 2019. SARIFA was assessed histologically on H&E slides. Survival analyses used Kaplan-Meier curves and Cox regression models (univariable, multivariable), focusing on disease-free survival (DFS). Model performance was evaluated using AIC, concordance index, and likelihood ratio tests. We first analyzed the full cohort to mirror the original inclusion criteria, then restricted analyses to a more clinically relevant subgroup of fat-infiltrative but non-metastatic disease (pT3-4 M0).

Results

SARIFA assessment was simple, rapid, and highly reproducible. In the full cohort (n=414), SARIFA-positive tumors were significantly associated with adverse features, including lymphovascular and perineural invasion, high tumor grade, and shorter DFS. However, SARIFA did not retain independent prognostic value in multivariable models, suggesting overlap with established markers.

To better assess clinical utility, we examined a refined pT3-4 M0 subgroup (n=281), in which its prognostic impact was even less evident. Inclusion of early-stage (pT1-2) tumors proved misleading, as all cases with submucosal SARIFA areas also displayed subserosal SARIFA. SARIFA was overrepresented in metastatic (M+) tumors, where clinical decisions are typically straightforward. Inclusion of such extreme cases may inflate SARIFA's apparent prognostic power.

Conclusions

SARIFA is a simple and reproducible morphologic marker associated with adverse tumor features and shorter DFS. However, it lacks independent prognostic value beyond established histologic parameters, and its predictive power is highly dependent on cohort composition. SARIFA may nonetheless capture a distinct tumor phenotype, potentially reflecting metabolic or immune interactions with adipose tissue, and warrants further biological investigation.

OF 06

THE HIDDEN MASS BEHIND THE DRUM: 2 CASES OF MIDDLE EAR NEUROENDOCRINE TUMOR

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Background

Two patients presented with non-specific otological symptoms and a middle-ear mass on CT. Biopsies revealed a MeNET, a rare tumour that, due to its variable clinical presentation, requires histopathological and immunohistochemical assessment for definitive diagnosis

Materials and methods

Both biopsies underwent microscopic examination on H&E-stained sections. The performed immunohistochemical panel consisted of Ki67, cytokeratin AE1/AE3, chromogranin, synaptophysin, TTF1, S100, SatB2 and INSM1.

Results

Microscopic examination showed a highly cellular lesion with mixed morphology, including trabecular structures, solid areas, and glandular growth lined by cuboidal cells. The cells were monomorphic with round, eccentric, hyperchromatic nuclei and eosinophilic cytoplasm. No mitoses, necrosis, or atypia were identified. The surrounding stroma was densely fibrotic with focal hyalinisation.

The neoplastic cells were positive for cytokeratin AE1/AE3, chromogranin, and synaptophysin. TTF1 was negative in both cases, although occasional positivity has been reported. SATB2 showed weak expression, and INSM1 showed strong nuclear staining. Both lesions exhibited a low proliferation index (Ki-67 <1% and 3%). S100 highlighted no sustentacular cells, and in combination with cytokeratin positivity, this provides two arguments against a paraganglioma.

Conclusions

The combination of the morphological findings and the immunohistochemical profile supports the diagnosis of MeNET. Recognising this rare entity is important because, although generally characterised by indolent behaviour, it may follow an unpredictable clinical course. Therefore, when encountering a middle ear lesion with heterogeneous morphology and a strikingly monomorphic cell population, MeNET should be considered and ruled out using immunohistochemistry.

OF 07

YOUR SCLEROSING EPITHELIOID FIBROSARCOMA REFUSES TO STAIN FOR MUC4? CHECK FOR THE YAP1-KMT2A FUSION!

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Background

Sclerosing epithelioid sarcoma (SEF) is a rare MUC4-positive tumor with specific gene fusions. A newly identified SEF-like sarcoma lacks MUC4 but shows YAP1-KMT2A fusion. This report presents a case of this extremely rare variant.

Materials and methods

Routine hematoxylin and eosin (H&E) and extensive immunohistochemical (IHC) stainings and DNA and RNA NGS was performed on a 5 x 5 cm lesion in the knee of a 67 year-old male.

Results

Based on the morphological and immunohistochemical features on the diagnostic biopsy specimen, a SEF was suspected, but MUC4 was repeatedly negative, being performed on the two blocks of the biopsy specimen, with appropriate on-slide external controls.

Given these findings, a sarcoma with YAP1 and KMT2A fusion was considered, but RNA NGS, performed on both blocks, was twice unsuccessful. DNA NGS succeeded and showed no mutations. Luckily, NGS RNA on the resection specimen did succeed and indeed revealed the suspected fusion, with indirect evidence for a YAP1-KMT2A-YAP1 configuration. Methylation and copy number profiling results were consistent with data in the literature.

Conclusions

Our case of YAP1-KMT2A-YAP1 sarcoma illustrates the importance of careful and critical integration of morphological and immunohistochemical findings with molecular findings in soft tissue tumor diagnostics.

OF 08

FLOWCHART TO REDUCE SENTINEL LYMPH NODE PROCEDURES AFTER BIOPSY-DIAGNOSED DUCTAL CARCINOMA IN SITU

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Background

Mammary ductal carcinoma in situ (DCIS) is generally treated by surgery and radiotherapy, and frequently also by hormonal therapy if hormone receptor-positive, analogous to invasive breast cancer (IBC). According to ASCO/NCCN recommendations for DCIS, the sentinel lymph node biopsy (SLNB) should only be performed for mastectomy, but SLNB is frequently realized for lumpectomy. Paradoxically, de-escalation is already performed for early IBC, but not yet for DCIS. As proposed by the INSEMA trial for early IBC, we believe the SLNB could be omitted for some DCIS.

Methods

We reviewed 270 lesions of biopsy-diagnosed pure DCIS. We evaluated nuclear grade, DCIS architecture, stroma architecture, necrosis, calcification and tumour-infiltrating lymphocytes, and ER and HER2 protein expression. ER-positivity was defined by 1-100% of nuclear immunoreactivity, and HER2-positivity by a 3+ score, similar to IBC.

Results

We included 173 lumpectomies and 97 mastectomies. 51/270 (18.9%) lesions were upstaged to IBC after surgery. 152/270 (56.3%) DCIS were associated with an SLNB, of which 72/152 were combined with a mastectomy, and only 6/152 (3.9%) were positive. In univariate analysis, upstaging risk was significantly associated with solid architecture (p-value=0.013) and lack of calcification (p-value=0.005). In multivariate analyses, they were both independently associated to upstaging. We constructed a decisional flowchart according to the IHC profile (ER/HER2) and the type of surgery. In the arm of ER+/HER2- and mastectomy, we identified high risk DCIS, defined by solid architecture and/or lack of calcification. Thirty-seven DCIS were in the low-risk arm: SNLB omission is safe, as only three DCIS (8%) were upstaged and all SLNB were negative.

Conclusions

DCIS patients are overtreated as 80/270 (29.6%) patients undergo a lumpectomy with SLNB, despite international recommendations. Our decisional flowchart includes HER2 immunohistochemistry, and supports omission of SLNB in selected low-risk patients undergoing lumpectomy or mastectomy, in accordance with the INSEMA trial for IBC.

NOTES

POSTERS

P 01

UNDIFFERENTIATED UTERINE SARCOMA WITH HER2-AMPLIFICATION AND S100/SOX10-COEXPRESSION

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Background

Uterine sarcomas comprise a heterogeneous group of rare malignant mesenchymal tumors, with high-grade and undifferentiated categories posing significant diagnostic challenges. Molecular profiling is essential to improve diagnosis, identifying subgroups such as NTRK-rearranged spindle cell sarcoma and high-grade endometrial stromal sarcoma with YWHAE::NUTM2 fusion or BCOR rearrangements. A novel entity - undifferentiated uterine sarcoma with HER2-amplification and S100/SOX10-coexpression - was recently reported, emphasizing its distinct molecular and therapeutic relevance.

Materials and methods

We report the case of a 56-year-old woman with abdominal pressure, pain, and urinary frequency. Imaging revealed a markedly enlarged uterus with multiple nodular masses and extensive areas of diffusion restriction, compatible with sarcomatous transformation. Additional findings included ureteral dilatation from extrinsic compression and multiple lung, liver, pancreas and bone metastases. Biopsy of the left uterine horn was performed.

Results

Histology showed a highly cellular neoplasm composed of a monotonous population of cells arranged in solid, non-cohesive sheets. The neoplastic cells exhibited round to oval nuclei with vesicular chromatin, inconspicuous nucleoli, and scant eosinophilic cytoplasm. Mitotic activity was brisk, with atypical forms present. Immunohistochemistry revealed strong positivity for SOX10, S100, Cyclin D1, and HER2 (3+ with ISH amplification), focal positivity for PRAME and DOG1. Negative markers included ER, desmin, smooth muscle actin, cytokeratins, CD34, CD10, HMB45, MelanA, PAX8, WT-1 and NTRK. Molecular analysis confirmed the presence of a pathogenic ERBB2 V842I variant, with evidence of ERBB2 amplification and a suspected CDKN2A deletion.

Conclusions

Current evidence supports the existence of a high-grade uterine sarcoma defined by activating ERBB2 mutations and a neurogenic-like immunophenotype (S100/SOX10-positive). Debate persists on whether tumors with distinct genetic alterations should be grouped based on pathologic characteristics or recognized as separate molecularly defined entities. Nevertheless, routine NGS testing is essential for the assessment of potential therapeutic strategies, such as ERBB2/ERBB3 tyrosine kinase mutations.

P 02

PAPULAR ACANTHOLYTIC DYSKERATOSIS OF THE ANOGENITAL REGION: REPORT OF TWO CASES

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Background & objectives

Papular acantholytic dyskeratosis (PAD) is a rare entity of uncertain etiology, affecting both sexes, with no family history. Clinically, it presents in the anogenital and genito-crural areas as multiple, grouped, flesh-colored to whitish papules, asymptomatic or pruritic. Histopathological features are suprabasal clefting, acantholysis, dyskeratosis, “corps ronds” and “corps grains”, hyperkeratosis and parakeratosis. Here, we present two cases of PAD.

Results

The first case consisted of a single lesion in a 51-year-old woman without personal or familial history of dermatological disease. Microscopic examination suggested an acantholytic dermatosis (Hailey-Hailey disease, Darier’s disease, Grover’s disease, pemphigus). Given the vulvovaginal location, the lack of family history, and the seemingly solitary nature of the lesion, it was also considered consistent with papular acantholytic dyskeratosis.

The second case is a 81-year-old patient with long-lasting vulvar itching lesions (over 20 years), resistant to topical steroid treatment. Microscopic morphological features were compatible with a papular acantholytic dyskeratosis of the genitocrural area, sharing overlapping features with Hailey-Hailey disease and Darier disease. PAD diagnosis was favoured given the long-lasting duration of the lesions, lack of known family history, and assuming that pemphigus had been ruled out.

Conclusions

Papular acantholytic dyskeratosis (PAD) is a rare entity of uncertain etiology mostly affecting anogenital areas. Fewer than 50 cases have been described in the literature. The differential diagnosis is broad and requires clinical correlation, as microscopy alone is not sufficient to rule out other acantholytic dyskeratotic entities such as Hailey-Hailey disease, Darier disease and pemphigus.

P 03

NIFTP WITH RARE BRAF EXON 15 DUPLICATION: MOLECULAR AND CLINICOPATHOLOGICAL INSIGHTS

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Background

MRare BRAF exon 15 mutations are seldom identified in papillary thyroid neoplasms, and their clinical significance remains unclear. We report a case of Non-Invasive Follicular Thyroid Neoplasm with Papillary-like Nuclear Features (NIFTP) harboring a BRAF c.1745_1795dup alteration to clarify its pathological and clinical implications.

Materials and methods

A 28-year-old female patient, asymptomatic and referred for surgical evaluation of an incidentally discovered left thyroid nodule, underwent ultrasound, two fine-needle aspirations, and left lobectomy. Histopathology revealed a NIFTP. Tumoral DNA extracted from FFPE sections was analyzed through targeted next-generation sequencing using a Twist custom panel. Clinical, cytological, histological, and molecular findings were integrated to determine the significance and clinicopathological correlation of the detected BRAF c.1745_1795dup variant.

Results

Ultrasound showed a 28 mm EU-TIRADS 3 nodule. Two fine-needle aspirations yielded Bethesda V and later Bethesda IV results. Left lobectomy revealed a NIFTP. Sequencing identified a rare BRAF c.1745_1795dup (p.I582_A598dup) variant with 45.9% VAF. This alteration has been reported only once in the literature and is associated with encapsulated follicular variant papillary thyroid carcinoma displaying indolent behavior. Our findings align with prior evidence, as the tumor showed low-risk clinicopathological features consistent with NIFTP.

Conclusions

This case represents the second report of BRAF c.1745_1795dup in NIFTP and supports its association with low-risk features. Careful evaluation of rare and uncertain variants, even at low allele frequencies, may improve anatomo-clinical correlation and guide more conservative therapeutic decisions.

P 04

ANTI-ER SP1 CLONE ISSUES AND THE ROLE OF EXTERNAL QUALITY ASSESSMENT IN DETECTING INSUFFICIENT DESCRIPTIVE LOWER LIMIT OF DETECTION

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Background and objective

Estrogen receptor (ER) immunohistochemistry is a predictive biomarker guiding endocrine therapy in breast cancer. Variations in staining quality may potentially lead to misclassification with impact on therapy decisions. We aimed to highlight the clinical implications of analytical variability and the critical role of external quality assessment (EQA) in maintaining diagnostic accuracy.

Materials and methods

Recent safety alerts from Roche reported insufficient staining with specific lots of the SP1 anti-ER antibody, potentially reducing staining intensity and the percentage of positive tumor cells, in particular in breast carcinomas with low hormone receptor expression. However, no false-negative tumors were reported by the firm. Internal investigations revealed inconsistencies in antibody dilution protocols, contributing to performance variability. We analyzed publicly available data from EQA programmes, including Sciensano and NordiQC, to evaluate interlaboratory performance, and the potential impact on the descriptive lower limit of detection (dLLOD).

Results

We estimated that the affected lots were used from November 2024 till August 2025. The NordiQC 2025 evaluation showed a reduced pass rate, mainly due to low analytical sensitivity of the SP1 clone. A similar reduced pass rate due to SP1 clone issues was observed in the 2025 Sciensano evaluation, whereas the pass rate for other clones (such as EP1) remained unchanged. EQA data demonstrated superior reproducibility with ready-to-use systems compared to concentrated antibodies and confirmed the importance of sensitive control tissues such as tonsil for dLLOD evaluation.

Conclusions

Our analysis of publicly available NordiQC and Sciensano data revealed that the SP1 clone issues were reflected in the pass rates of EQA programmes for ER immunohistochemistry. Participation in EQA is very important to ensure technical standardization, optimize staining protocols and safeguard accurate treatment stratification (especially in low ER-expressing breast cancers), but it can also reveal, in retrospect, performance issues of a particular antibody clone.

P 05

LYMPH NODE DIAGNOSIS OF WHIPPLE DISEASE REVEALED DURING SYSTEMIC EOSINOPHILIC SYNDROME WORKUP

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Background

Whipple disease is a rare systemic infection caused by Tropheryma whipplei. Diagnosis usually relies on small-bowel biopsies, while primary lymph node involvement and associated eosinophilia are both uncommon. We report a case in which lymph node histopathology first suggested the diagnosis during evaluation of persistent eosinophilia.

Materials and methods

A 25-year-old woman presented with abdominal pain, mesenteric lymphadenopathy, cutaneous lesions, and persistent eosinophilia. PET imaging showed multiple hypermetabolic nodes. Extensive evaluation, including skin, gastrointestinal and bone marrow biopsies, was performed to exclude parasitic, vasculitic and clonal eosinophilic disorders. After an initial non-diagnostic lymph node biopsy, a second laparoscopic biopsy underwent routine histology and PAS staining. Molecular testing for T. whipplei is ongoing. The patient improved markedly after antibiotic therapy.

Results

Skin, gastrointestinal and bone marrow biopsies revealed eosinophilic infiltration without histiocytic involvement, and PAS staining was negative. Histological examination of the laparoscopic lymph node biopsy revealed preserved nodal architecture with marked sinusoidal expansion by foamy histiocytes forming confluent clusters, consistent with histiocytic lymphadenitis. These macrophages contained PAS-positive granular intracytoplasmic material. Despite the absence of histiocytic infiltration in previous biopsies, the lymph node findings were highly suggestive of Whipple disease. No lymphoproliferative disorder or alternative infection was identified. The morphological features, together with the patient's rapid clinical improvement after antibiotics, strongly supported Whipple disease as the underlying cause, pending molecular confirmation.

Conclusions

This case highlights an uncommon presentation of Whipple disease identified through lymph node biopsy during the evaluation of systemic eosinophilic involvement. It emphasizes the key role of pathology in recognizing this rare infection and the importance of adequate tissue sampling when initial biopsies are inconclusive.

P 06

A BENIGN MIMIC OF MUCINOUS NEOPLASM: PITFALLS IN ILEOCOLIC RESECTION

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Introduction

We present the case of a patient with Crohn's disease diagnosed with enteritis cystica profunda, emphasizing its pathogenesis, link with Crohn's disease, and the importance of distinguishing it from mucinous adenocarcinoma.

Case presentation

A 60-year-old man with Crohn's disease (Montreal A3L1B3) and primary sclerosing cholangitis underwent repeat ileocolonic resection for refractory ileitis. He had previously undergone ileocecal resection with end-to-side anastomosis in 2017. After years of lost follow-up, he resumed treatment for abdominal pain and diarrhoea. Despite clinical improvement, MRI in 2024 showed progression. Surgery was postponed due to his continued smoking. In 2025, he developed intestinal obstruction. Endoscopy revealed stenosis of the neoterminal ileum; colonoscopy was unremarkable. CT showed extensive ileitis with intramural abscesses, sinus tracts, and prestenotic dilatation. Surgery revealed dense adhesions and severe ileal inflammation. Gross examination showed firm nodularity with subserosal mucinous collections, suggesting neoplasm. Distal ileal stenosis was identified, along with proximal mucosal flattening and polypoid structures. Microscopic examination revealed active chronic ileitis with ulceration, focal stenosis, abscesses and fistulisation. Striking mucin collections lined by non-atypical, well-differentiated mucinous epithelium were present in the submucosa and muscularis propria, accompanied by acellular mucin lakes with a focal inflammatory reaction. These findings were consistent with enteritis cystica profunda.

Discussion

Enteritis cystica profunda is a rare benign condition characterised by misplaced mucosal glands and mucinous cysts located beneath the mucosa. It likely develops because of ulceration or inflammatory injury, as occurs in Crohn's disease, enabling epithelial migration into deeper layers. Histologically, it closely resembles invasive adenocarcinoma, which complicates diagnosis. Given the increased risk of small bowel adenocarcinoma in Crohn's disease, awareness is crucial for accurate differential diagnosis. Similar lesions occur in the colon and stomach and are known as colitis cystica profunda and gastritis cystica profunda, respectively.

P 07

THE UNDERRATED CULPRIT: FETAL NEUROPATHOLOGY OF IN UTERO HERPES SIMPLEX VIRUS INFECTION.

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Herpes simplex virus (HSV) is a widespread sexually transmitted infection, with both HSV type 1 and 2 potentially causing genital herpes. Primary infection during pregnancy can occur and is asymptomatic in approximately 80% of mothers. Fetal infection most commonly takes place during delivery from contact with the genital mucosa (85%), although, vertical transmission to the fetus or newborn can occur postnatally (10%), or through the placenta (5%) via hematogenous spread. We report the case of a 38-year-old woman referred at 23 weeks due to polyhydramnios and bilateral hydronephrosis on ultrasound. A follow up fetal MRI at 27 weeks gestation revealed diffuse brain damage with paraventricular bifrontal hyper- and hypointensities, an irregular cortical mantle suspicious for polymicrogyria, mild ventriculomegaly and abnormal cerebellar hemispheres. Given the progression to panventricular hydrocephaly on ultrasound, the pregnancy was electively terminated at 30 weeks. Fetal neuropathological examination showed severe bilateral ventriculomegaly with extensive necrosis of the cerebral cortex and white matter, and, to a lesser degree, the brainstem and cerebellum. Marked calcifications and inflammatory changes were also present. HSV infection was confirmed by PCR analysis of amniotic fluid and by immunohistochemical staining of fetal brain. The fetal visceral organs and the placenta showed no viral inclusions or positivity for HSV on immunohistochemistry, but hepatosplenomegaly was noted. Maternal serology indicated a primary infection with HSV2, with both IgG and IgM antibodies testing positive. This case illustrates the extensive neurologic damage that can result from congenital HSV infection and emphasizes the need for heightened clinical awareness.

P 08

SOFTWARE TOOL FOR TEMPLATE-BASED STANDARDIZED STRUCTURED REPORTING IN PATHOLOGY: IS IT TIME-SAVING?

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Background

Reporting in pathology has changed throughout the years with increased use of synoptic reporting. This study aims to evaluate the time-efficiency of an online tool for template-based standardized structured reporting in skin pathology.

Materials and methods

Over one month, we implemented a software tool for template-based standardized structured reporting (from Tiro.health). Forms in the tool were designed, based on ICCR templates when available, or proprietary. For each finished real-world skin pathology report using one of the forms, time to completion was recorded. This was compared to time to completion for typing, and dictation. Afterwards, pathologists and clinicians were asked to fill out a questionnaire regarding the tool's usefulness, ease of use, ease of learning, and satisfaction.

Results

A total of 102 reports were analyzed for time to completion. Reports created using the tool were significantly faster (62.4s) compared to typing (108.8s) or dictating (84.5s). A statistically significant linear regression showed users improved in speed over time, indicating the tool is easy to learn, supported by questionnaire results. Moreover, both pathologists and clinicians rated the tool as useful and expressed high satisfaction.
Literature only holds limited information about improved turnaround times when using a software tool. To our knowledge, this research (though small scale) is the first to assess the time-efficiency of this type of structured reporting tool.

Conclusions

The use of a software tool for electronic standardized structured reporting shows promising results in terms of efficiency, thus enabling faster reporting. Feedback from both clinicians and pathologists indicated good user satisfaction.
To our knowledge, this is the first study to demonstrate the time-efficiency of software-based standardized structured reporting.

P 09

MYOEPITHELIAL-RICH ADENOSIS OF THE BREAST: A RARE BENIGN LESION MIMICKING MALIGNANCY

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Background

Breast adenosis is a benign lesion that may appear as architectural distortion, calcifications, or a mass. Its rare adenomyoepithelial variant can mimic malignancy, requiring selective immunohistochemistry to ensure accurate diagnosis and prevent misinterpretation, as illustrated in this case.

Materials and methods

Breast cancer screening was conducted in a 42-year-old patient undergoing treatment for a meningioma, with no specific breast-related complaints. Imaging revealed a BI-RADS 4a mass in the upper inner quadrant of the left breast, leading to an ultrasound-guided microbiopsy. Routine hematoxylin and eosin staining and immunohistochemistry (IHC) were performed on the biopsy samples. The IHC panel included p40, CK5, estrogen receptor (ER), progesterone receptor (PR), and E-cadherin.

Results

Careful evaluation revealed a dual population of epithelial and myoepithelial cells with distinctive staining patterns. The diagnosis of adenomyoepithelial adenosis was supported by strong peripheral lobular staining for p40 and CK5, while ER and PR expression mirrored that of adjacent normal luminal cells in surrounding lobules and ducts. Without immunohistochemical correlation, the lesion could easily be misinterpreted as lobular colonization by ductal carcinoma in situ (DCIS), as flat epithelial atypia or atypical ductal hyperplasia. Our findings are consistent with specific histological and immunophenotypic features reported in the literature, emphasizing the importance of integrating immunohistochemistry to achieve accurate diagnosis.

Conclusions

This case illustrates that adenosis, although benign, may present with prominent myoepithelial components mimicking B3 or B5a lesions such as limited DCIS. Accurate recognition of its histopathological architecture, combined with targeted immunohistochemical analysis, is crucial to prevent misinterpretation in breast pathology and to ensure subsequent appropriate clinical management.

P 10

SECRETORY BREAST CARCINOMA AND IMMUNOHISTOCHEMICAL PROFILE : A CASE REPORT

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Introduction

Secretory carcinoma is a rare histological type of invasive breast carcinoma with distinct histological features. This salivary gland-type tumor is characterized by a pathognomonic *ETV6-NTRK3* fusion.

Materials and methods

We report a female patient, aged 44, without history of disease, who had a 1cm nodule in her right breast. The biopsy underwent conventional histopathological, immunohistochemical and molecular analysis. A lumpectomy was also performed.

Results

Standard histology showed an infiltrative carcinomatous proliferation with a solid and cribriform architecture and colloidal intraluminal secretions. The cells were large with abundant eosinophilic cytoplasm, occasionally vacuolated. Nuclear atypia was moderate with rare mitoses. The fibrous stroma was sparse with mild chronic inflammation. Alcian Blue and Periodic Acid-Schiff stains were positive in the intraluminal secretions. Immunohistochemistry (IHC) showed a weak nuclear immunoreactivity for ER in 10 to 20% of tumor cells negative. The tumor was negative for PR and HER2, with a Ki67 index below 5%. The absent SMM-HC immunoreactivity within neoplastic cells confirmed the absence of myoepithelial cells and the infiltrating nature of the tumor. The antibodies CK5, CK7, S100, MUC-4, SOX10, and GATA3 were positive within the neoplastic cells, and there was focal weak p63 immunoreactivity. This profile was highly suggestive of secretory carcinoma. AR was negative, excluding an apocrine carcinoma. Pan-TRK IHC showed diffuse weak cytoplasmic staining of the tumor cells. RNAseq (Archer® FusionPlex® Sarcoma V2 panel) did not identify an ETV6-NTRK3 gene fusion. Confirmatory FISH analysis is currently ongoing.

Conclusion

Secretory breast carcinomas are often triple-negative (absent ER, PR and HER2 immunoreactivity). Despite the strong association with *ETV6-NTRK3* fusion, not all tumors show pan-TRK immunoreactivity. The tumor we describe here, did show diffuse weak positivity for pan-TRK, but so far, we were not able to demonstrate an *ETV6-NTRK3* fusion. Additional molecular analysis is currently ongoing to search for an alternative fusion.

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A CHALLENGING ADENOMYOEPITHELIAL TUMOUR WITH AN UNEXPEXTED SURPRISE

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Background and objective

Pleomorphic adenoma and benign adenomyoepithelioma of the breast are rare biphasic salivary gland-type tumors that may appear similar on limited sampling, especially when the latter is associated with extensive myxoid stroma. We report a case initially evaluated by cytology and core needle biopsy, ultimately diagnosed on the surgical specimen as a combined classic adenoid cystic carcinoma (ACC) and atypical adenomyoepithelioma (AME).

Case presentation

The patient initially underwent fine-needle aspiration of a 1cm nodule in breast. Cytological analysis showed non-atypical epithelial cells, suggestive of benign epithelial hyperplasia. Two years later, the patient complained about the nodule being painful.
The subsequent core needle biopsy revealed a biphasic epithelial–myoepithelial proliferation arranged in tubular structures within a myxoid stroma. The absence of cytologic atypia, mitotic activity or necrosis supported a differential diagnosis between pleomorphic adenoma and benign AME. Surgical excision of this B3 lesion was performed.
The excision specimen demonstrated two juxtaposed components. The first corresponded to a classic ACC, with cribriform and tubular patterns, a dual cell population, and abundant basement-membrane-like material in the lumina. The second component represented an AME, considered as an atypical AME, because of the focally infiltrative character and the presence of a slightly increased mitotic activity (3 mitoses/2mm^2).

Conclusions

This case highlights the inherent difficulties of diagnosing salivary gland-type breast lesions on cytology and core biopsy, where pleomorphic adenoma and AME may overlap.
The final identification of a classic ACC associated with the atypical AME underscores the importance of complete excision and thorough histopathologic evaluation.

NOTES

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