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Malignant Ascites Mimicking Meigs and Pseudo-Meigs Syndromes: A Case of Cytology-Proven Metastatic Adenocarcinoma with Peritoneal Carcinomatosis

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Abstract

Meigs' syndrome is classically defined as the triad of a benign ovarian fibroma (or fibroma-like tumour), ascites, and pleural effusion, with complete resolution of the fluid accumulations following surgical resection of the tumour. Pseudo-Meigs' syndrome broadens this presentation to other pelvic or ovarian tumours (benign or malignant) but is strictly characterised by the absence of malignant cells in the ascitic or pleural fluid. We report a 79-year-old woman who presented with progressive abdominal distension and imaging features suggestive of ovarian malignancy, initially raising suspicion for a Meigs'-like physiology. However, cytological examination of the ascitic fluid revealed metastatic adenocarcinoma with immunohistochemical features consistent with epithelial origin, confirming peritoneal carcinomatosis and malignant ascites. This finding definitively excluded both Meigs' and pseudo-Meigs' syndromes. The case underscores the indispensable role of ascitic fluid cytology in differentiating benign transudative processes from true malignant dissemination, with major implications for accurate staging, therapeutic planning, and prognosis in suspected gynaecological malignancies.

Keywords: Meigs' syndrome; Pseudo-Meigs' syndrome; Cytological

Introduction

Ascites in postmenopausal women with suspected pelvic pathology poses a significant diagnostic challenge. The differential includes benign entities such as Meigs' syndrome, inflammatory or infectious peritonitis, and advanced malignancies with peritoneal spread. Meigs' syndrome, first systematically described in 1937 by Joe Vincent Meigs and John W. Cass, involves a benign solid ovarian tumour (most commonly fibroma), ascites, and often right-sided pleural effusion that resolves after tumour removal. The pathophysiology is thought to involve transudation of fluid from the tumour surface due to mechanical irritation, lymphatic obstruction, or cytokine release, without direct malignant involvement. Pseudo-Meigs' syndrome extends the triad to other ovarian or pelvic tumours, including non-fibromatous benign lesions (e.g., struma ovarii, mucinous cystadenoma, mature teratoma) or even malignant neoplasms and metastatic deposits to the ovary, provided the effusions remain cytologically negative for malignant cells. In contrast, malignant ascites—typically associated with peritoneal carcinomatosis—features positive cytology, low serum-ascites albumin gradient (SAAG), high protein content, and persistent fluid accumulation driven by increased vascular permeability (mediated by factors such as vascular endothelial growth factor, VEGF), lymphatic obstruction, and direct tumour infiltration. Distinguishing these entities is critical: Meigs' and pseudo-Meigs' syndromes are potentially curable with surgery alone, whereas malignant ascites signals advanced-stage disease (often FIGO stage III or IV

ovarian or primary peritoneal carcinoma) with poorer prognosis and the need for systemic therapy. Elevated serum CA-125, while sensitive for ovarian/peritoneal malignancy, lacks specificity and can occur in both benign and malignant conditions with peritoneal irritation. This case illustrates how imaging and tumour markers may mimic a Meigs'-like syndrome, only for cytology to reveal the malignant nature [1-13].

Case Presentation

A 79-year-old woman was admitted to hospital with diarrhoea and abdominal discomfort, ultimately diagnosed with Salmonella gastroenteritis. Her medical history included chronic obstructive pulmonary disease (COPD) with recurrent infective exacerbations. During the admission, she developed progressive abdominal distension. Physical examination demonstrated shifting dullness consistent with moderate ascites. There were no stigmata of chronic liver disease, cardiac failure, or peripheral oedema suggestive of hypoalbuminaemia-related fluid overload.

Laboratory Investigations

Serum biochemistry showed hypoalbuminaemia (albumin 28 g/L) with total protein 55 g/L, mild hypokalaemia (potassium 3.3 mmol/L), and low fasting glucose (3.2 mmol/L). Renal function was preserved (creatinine 57 µmol/L, eGFR 85 mL/min). Liver enzymes and coagulation profile were unremarkable. Tumour marker evaluation revealed a markedly elevated CA-125 level of 1876 U/mL (normal <35 U/mL), raising strong suspicion for ovarian or primary peritoneal carcinoma.

Imaging Findings

Contrast-enhanced CT of the abdomen and pelvis demonstrated multiple enhancing peritoneal nodules, nodular infiltration extending to the umbilicus (consistent with a Sister Mary Joseph nodule), and peritoneal disease outlining the pelvis with involvement surrounding the uterus and adnexa. There was small-volume ascites but no discrete visceral mass in the liver, pancreas, adrenals, or kidneys. Additional findings included hepatomegaly, aortoiliac atherosclerosis with mild infrarenal aortic ectasia, and pelvic floor descent with a posterior perineal hernia. CT chest showed no pleural effusion, pulmonary embolism, thoracic lymphadenopathy, or osseous metastases. Background centrilobular nodular opacities and mild emphysematous changes were consistent with her known COPD. A 6 mm subsolid nodule in the right lower lobe was noted for interval surveillance, but there was no evidence of thoracic metastatic disease (Figure 1).

Ascitic Fluid Analysis

Diagnostic paracentesis yielded fluid with albumin 20 g/L. The serum-ascites albumin gradient (SAAG) was calculated as 0.8 g/dL (serum albumin 28 g/L minus ascitic albumin 20 g/L, converted appropriately). A low SAAG (<1.1 g/dL) indicated non-portal hypertensive ascites, favouring peritoneal carcinomatosis, infection, or inflammatory causes over cirrhosis.

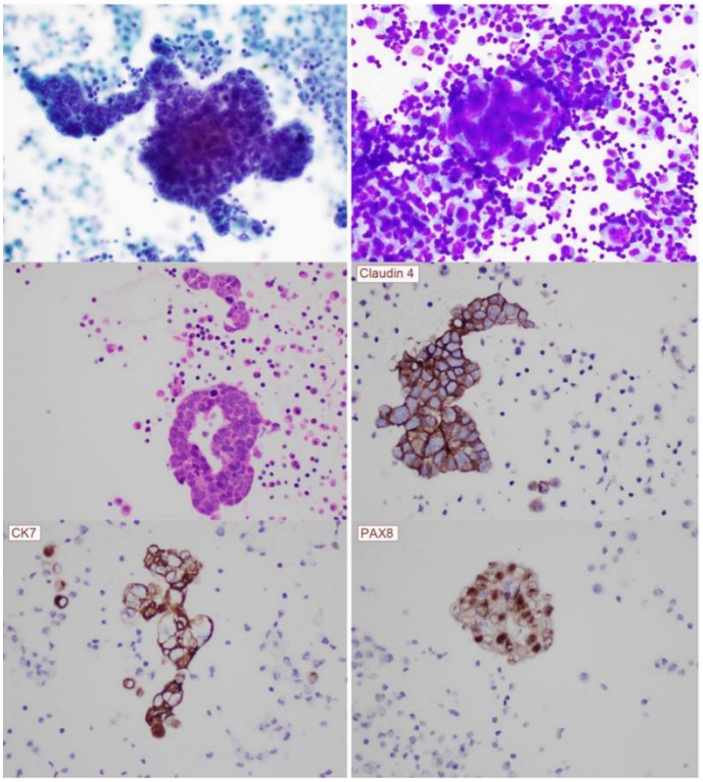


Figure 1: *Cohesive three-dimensional clusters and papillary/acinar groups of atypical epithelial cells in an inflammatory background. The cells showed nuclear enlargement, hyperchromasia, nuclear crowding and moderate cytological atypia, consistent with metasta me tic. Immunocytochemistry demonstrated strong membranous positivity for claudin-4 and CK7, supporting epithelial adenocarcinoma differentiation. The tumour cells also showed nuclear PAX8 positivity, favouring a Müllerian origin, including ovarian, fallopian tube or primary peritoneal carcinoma.*

Cytopathology

Cytological examination revealed clusters of malignant epithelial cells exhibiting nuclear atypia, prominent nucleoli, and cytoplasmic vacuolation consistent with adenocarcinoma. Immunohistochemistry on the cell block showed positivity for broad-spectrum cytokeratins (AE1/AE3), epithelial membrane antigen (EMA), BerEP4, and Claudin-4, with negativity for CK5 and calretinin. These markers confirmed metastatic adenocarcinoma of epithelial (non-mesothelial) origin and effectively excluded reactive mesothelial proliferation or malignant mesothelioma.

Diagnosis

The integrated clinical, radiological, biochemical, and cytopathological findings established advanced intra-abdominal malignancy, most consistent with ovarian or primary peritoneal serous carcinoma, manifesting as:

- Peritoneal carcinomatosis
- Malignant ascites with positive cytology
- Umbilical cutaneous metastasis (Sister Mary Joseph nodule)

The presence of malignant cells in the ascites definitively excluded Meigs' and pseudo-Meigs' syndromes.

Discussion

Pathophysiology of Ascites in Ovarian and Peritoneal Malignancy

Malignant ascites in advanced ovarian/peritoneal carcinoma arises through multiple mechanisms: tumour-induced neovascularisation and increased permeability via VEGF and other cytokines; lymphatic obstruction by peritoneal deposits; direct exudation from tumour surfaces; and systemic effects such as hypoalbuminaemia reducing oncotic pressure. The fluid is typically exudative with low SAAG, high protein, and often elevated lactate dehydrogenase. Cytology is positive in a significant proportion of cases, though sensitivity can vary (approximately 60–90% in ovarian cancer-related ascites).

Meigs' syndrome

True Meigs' syndrome requires a benign ovarian fibroma (or similar solid tumour), ascites, and pleural effusion (classically right-sided), with resolution after tumour resection and negative cytology. The exact mechanism remains unclear but is postulated to involve fluid transudation from the tumour surface or lymphatic leakage, possibly exacerbated by tumour size or mobility. Elevated CA-125 can occasionally occur due to peritoneal irritation but does not indicate malignancy.

Pseudo-Meigs' syndrome

This term encompasses similar fluid accumulations with ovarian or pelvic tumours other than fibromas, including malignant lesions, provided cytology remains negative. Reported associations include struma ovarii, leiomyomas, teratomas, and even Krukenberg tumours or other metastases when effusions lack malignant cells. Resolution after tumour removal remains a key criterion.

Malignant ascites in this case

In the present patient, positive cytology for metastatic adenocarcinoma, imaging evidence of diffuse peritoneal disease, Sister Mary Joseph nodule, and markedly elevated CA-125 confirmed true malignant ascites secondary to peritoneal carcinomatosis. Unlike Meigs' or pseudo-Meigs' syndromes, the fluid accumulation here reflects direct malignant involvement rather than a reversible paraneoplastic or mechanical phenomenon. The low SAAG further supported an exudative peritoneal process.

Diagnostic pitfalls and the role of cytology

This case highlights a common pitfall: ascites with pelvic masses and elevated CA-125 can mimic Meigs'-like syndromes, potentially delaying recognition of advanced malignancy. The Sister Mary Joseph nodule—a metastatic deposit at the umbilicus—is a classic sign of intra-abdominal adenocarcinoma (particularly ovarian, gastric, or colorectal) and is not associated with benign Meigs' or pseudo-Meigs' syndromes. Ascitic fluid cytology with immunohistochemistry (e.g., BerEP4 and Claudin-4 positivity for adenocarcinoma; calretinin negativity excluding mesothelial origin) is pivotal for definitive diagnosis. Failure to perform or correctly interpret cytology risks misclassification and inappropriate management. In ambiguous cases, additional markers (e.g., PAX8, WT1 for serous ovarian carcinoma) or biopsy of peritoneal nodules may be required.

Prognostic and Management Implications

Malignant ascites with peritoneal carcinomatosis typically indicates stage III/IV disease and portends a poorer prognosis compared with resectable benign tumours in Meigs' syndrome. Management involves systemic chemotherapy (often platinum-based), targeted therapies (e.g., PARP inhibitors in BRCA-mutated cases), and palliative measures such as repeated paracentesis or indwelling catheters for symptomatic relief. Surgical debulking may be considered in select patients with good performance status.

Conclusion

This case demonstrates that while clinical and radiological features may suggest a Meigs'-like syndrome, cytological confirmation of malignant cells is essential to establish the diagnosis of malignant ascites and peritoneal carcinomatosis. Routine performance of diagnostic paracentesis with cytopathology in such presentations prevents diagnostic error and ensures timely oncological intervention.

References

1. Mohammed SA, Kumar A, Cue L. Meigs Syndrome. StatPearls. Treasure Island (FL): StatPearls Publishing; 2024.
2. Meigs JV. Fibroma of the ovary with ascites and hydrothorax; Meigs' syndrome. Am J Obstet Gynecol. 1954.

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3. Lurie S. Meigs' syndrome: the history of the eponym. Gynecol Obstet Invest. 2000.
4. Saha S, Robertson M. Meigs' and Pseudo-Meigs' syndrome. Australas J Ultrasound Med. 2012.
5. Peparini N, Chirletti P. Ovarian malignancies with cytologically negative pleural and peritoneal effusions: demons' or meigs' pseudo-syndromes?. Int J Surg Pathol. 2009.
6. Radiopaedia. Meigs syndrome. Accessed 2026.
7. Schickler RL, Abdallah R, McClung EC, Shahzad MMK. Primary peritoneal carcinoma presenting as a Sister Mary Joseph nodule. Gynecol Oncol Rep. 2016.
8. Iavazzo C, Madhuri K, Essapen S, Akrivos N, Tailor A, Butler-manuel S, et al. Sister Mary Joseph's Nodule as a First Manifestation of Primary Peritoneal Cancer. Case Rep Obstet Gynecol. 2012.
9. Zivadinovic R, Petric A, Krtinic D, Milosevic JS, Dinic SPT. Ascites in Ovarian Carcinoma – Reliability and Limitations of Cytological Analysis. J Med Biochem. 2016.
10. Jo VY, Cibas ES, Pinkus GS. Claudin-4 immunohistochemistry is highly effective in distinguishing epithelioid malignant mesothelioma from metastatic carcinoma. Mod Pathol. 2014.
11. Mangla G, Baski P, Agarwal P, Verma K. Diagnostic Utility of Claudin4 and Comparison with BerEp4 in Malignant Effusions. Diagn Cytopathol. 2023.
12. Huang CJ, Jiang JK, Chang SC, Lin JK, Yang SH. Serum CA125 concentration as a predictor of peritoneal dissemination in colorectal cancer. World J Gastroenterol. 2016.
13. Timmerman D, Moerman P, Vergote I. Meigs' Syndrome with Elevated Serum CA 125 Levels: Two Case Reports and Review of the Literature. Gynecol Oncol. 1995.