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A Diagnostic Twist: Primary Bladder Adenocarcinoma Masquerading as an Ovarian Torsion: A Case Report on Unusual Cystic Presentation

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Abstract

Primary bladder adenocarcinoma (PBA) is a rare malignancy comprising of only 0.5–2% of all bladder cancers and typically presents with nonspecific urinary symptoms. Cystic presentation is extremely uncommon and can mimic other pelvic pathologies. We report a very rare case of PBA in a 40-year-old female initially diagnosed as ovarian torsion based on clinical features and ultrasound findings. Intraoperatively, a cystic mass was seen on bladder dome which on frozen suggested malignancy leading to diagnosis of urachal adenocarcinoma. But in resected specimen, histopathology along with immunohistochemistry confirmed the diagnosis of enteric-type PBA after ruling out its differentials like urachal, urothelial and metastatic adenocarcinoma. To the best of our knowledge, this is the first reported case of cystic PBA. This case highlights the diagnostic challenges and the need for a broad differential in pelvic mass evaluation.

Key words cystic pelvic mass, ovarian torsion mimic, female bladder cancer, enteric type adenocarcinoma

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Introduction

Primary bladder adenocarcinoma (PBA) is a rare malignancy accounting for only 0.5–2% of all bladder cancers [1-3]. It originates from urothelium showing pure glandular differentiation and have different histological variants [2, 3]. Predominantly seen in male, it often presents with nonspecific urinary symptoms such as haematuria, dysuria or pelvic discomfort [4]. Cyst formation by such tumour is exceptionally uncommon and can pose significant diagnostic challenges by mimicking other pelvic pathologies. We present a very rare case of PBA forming a cystic pelvic mass in a female patient that clinically and radiologically mimicked ovarian torsion. Ovarian torsion is a gynaecological emergency often associated with ovarian cysts or masses [5]. After review of all literatures, to the best of our knowledge, this is the first reported case of its kind. This report narrates the preoperative, intraoperative assessment, histopathological and Immunohistochemistry (IHC) confirmation, emphasizing the need for a broad differential when evaluating pelvic masses.

Case presentation

A 40-year-old female presented with gradually worsening lower abdominal pain and non-bilious vomiting for the past 1 day. There was no history of fever, any urinary symptoms, bowel disturbance or menstrual irregularities. Physical examination shows lower abdominal tenderness and mild leukocytosis in lab investigations. Ultrasound revealed a well-defined cystic lesion in the suprapubic midline pelvic cavity measuring 9.7x9.6x9.6 cm (volume-480cc) with echogenic debris and peripheral enhancing solid component, showing no vascularity on colour doppler (**Figure 1A**). Along with it non visualisation of left ovary was suggestive of torsion of left ovarian dermoid cyst.

Given the suspicion of torsion without any further imaging study, the patient underwent emergency explorative laparotomy. A Pfannenstiel incision was made, revealing a cystic mass seem to arise from the dome of bladder (**Figure 1B**). Both ovaries and fallopian tubes were normal. The cyst ruptured during manipulation exposing solid component inside. Intraoperative frozen from this showed mucin with neoplastic glands in muscle layer suggesting adenocarcinoma. Given its origin from the bladder dome, urachal adenocarcinoma was suspected. Intraoperative cystoscopy also showed solid ulcerating growth in bladder mucosa. Partial cystectomy with extended bilateral pelvic lymphadenectomy along with excision of urachus and umbilicus was done and sent for histopathology study.

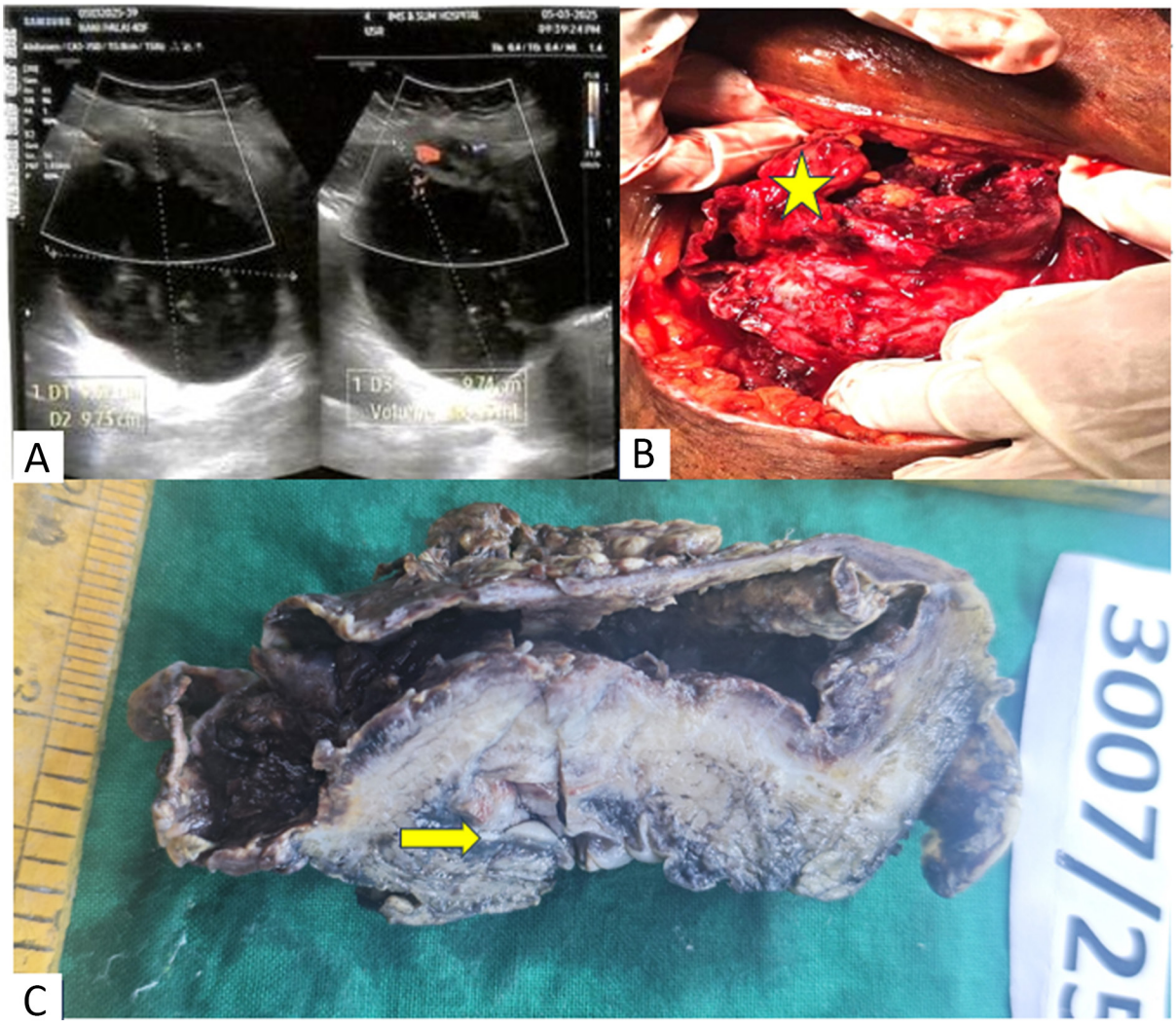

Figure 1. (A) Ultrasound showing suprapubic cystic lesion with lack of doppler blood flow, (B) Intraoperatively a ruptured cyst above bladder dome (yellow star), (C) Gross showing a cyst and mucosal growth (yellow arrow).

Grossly, a ruptured cyst was seen over the bladder along with a solid ulcerating growth inside the mucosa of dome and posterior wall. Cut surface of both lesions contained mucin and debris (**Figure 1C**). Histopathology revealed a tumour arising from mucosa of bladder showing pure glandular differentiation and lined by mucin producing pseudostratified columnar epithelium with lumen containing mucin and dirty necrosis (**Figure 2A, 2B**). Tumour penetrates outer half of deep muscularis propria. The mucin dissected the muscle layer (**Figure 2C**) and formed cyst which sat over the dome of bladder. Background shows feature of chronic cystitis with cystitis cystica (**Figure 2D**). Thorough sampling revealed no Invasive or non-invasive urothelial carcinoma components or urachal involvement. IHC were positive for CK20, CDX2, SATB2 suggesting adenocarcinoma with intestinal differentiation (**Figure 3**). Tumour cells were negative for CK7, GATA3, Beta catenin (nuclear reactivity), Pax8 and AMACR (**Figure 4**), thus confirming it to be primary adenocarcinoma bladder, enteric type after excluding its other differentials. Post-op colonoscopy showed no neoplastic growth.

The patient recovered well post-surgery and remains asymptomatic on regular follow-up.

Discussion

Primary bladder adenocarcinoma which originates from bladder urothelium with pure glandular differentiation is very rare, comprising only 0.5–2% of all bladder cancer. Various histologically variants of PBA have been described including enteric, mucinous, signet ring, clear cell, mixed and NOS type, with the enteric type being the most common [1-3]. Secondary involvement of bladder by adenocarcinoma of other organs are far more common [2, 3]. Also, adenocarcinoma may arise from urachus remnant and mullerian remnants. So, all these differentials are excluded before diagnosing PBA [6, 7].

PBA mainly seen in sixth decade with a male predominance [1], but in our case the patient is a 40-yr female. Precursors of this tumour include chronic irritation, cystitis glandularis with intestinal metaplasia, exstrophy and schistosomiasis [1, 3]. Histopathology of our case shows cystitis cystica and chronic inflammation which are likely the trigger. Common symptoms of PBA are haematuria, dysuria, pelvic pain which overlap with other conditions causing diagnostic delay [3, 4]. Ovarian torsion, typically caused by a cyst, presents with sudden abdominal pain & vomiting [5]. Patient in our case had no haematuria but exhibited classic torsion signs. Cystic mass with non-visualisation of left ovary in ultrasound and lack of doppler blood flow further supported the diagnosis of ovarian torsion. Although Computed tomography or magnetic resonance imaging could have provided better characterization and identified bladder involvement, the urgency of suspected ovarian torsion necessitated immediate surgery without further imaging.

However, during surgery, the absence of ovarian involvement and the discovery of a cystic mass arising from the bladder dome prompted a change in diagnosis. Further intraoperative frozen

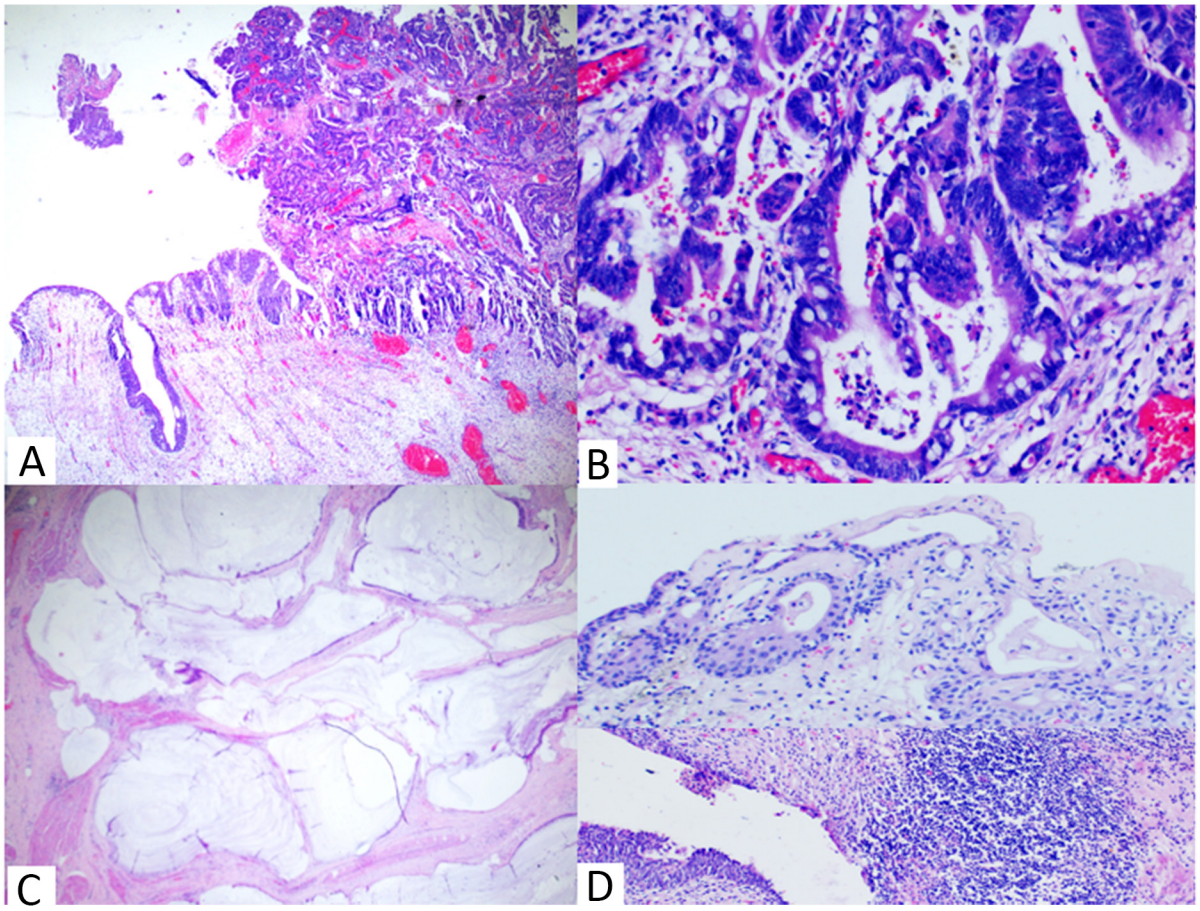

Figure 2. Histopathology (H&E staining) showing. (A) Tumour arising from mucosa (40x), (B) Tumour with Intestinal differentiation & necrotic debris (400x), (C) Mucin in muscle layer (100x), (D) Cystitis cystica and Chronic cystitis (100x).

from cystic mass was suggestive of adenocarcinoma. In our case as the cyst was over the dome, which is typical of urachal adenocarcinoma, an intra-op diagnosis of urachal adenocarcinoma was made [6]. A Partial cystectomy with lymph nodes dissection were performed and sent for histopathology.

The definite diagnosis of PBA was established through histopathology, IHC and clinical history. Histopathology was suggestive of adenocarcinoma, lined by mucin secreting cells. The tumour invaded muscle layer with mucin dissecting it and forming a cyst with a thick fibromuscular wall that seem to siting over the dome, which mimic a cystic lesion and ovarian torsion in imaging. We didn't classify it as mucinous adenocarcinoma due to mucin content being less than < 50% [3]. None of the literature showed this type of features. Kamel K et al. reported a paediatric case mimicking ovarian torsion, later diagnosed histopathologically as complicated urachal cyst [8]. Histomorphology along with positivity for CK20, CDX2 and SATB2 IHC lead to broad diagnosis of adenocarcinoma with intestinal differentiation [9]. In our case CK7 was negative. Eissa et al. found CDX2 positivity in 100%, CK20 in 92%, CK7 in only 15 % of 100 enteric type PBA cases [10]. Similarly, Wang et al. reported CDX2 and SATB2 expression in 100% of bladder malignancy with intestinal differentiation [9].

This adenocarcinoma was confirmed as primary bladder origin after excluding its differentials such as urachal adenocarcinoma, urothelial carcinoma with glandular differentiation, metastatic adenocarcinoma and mullerian origin tumour. Urachal adenocarcinoma arises from urachus, typically at bladder dome and may appear cystic. Histologically it shows glandular differentiation with an intramural tumour epicentre with a sharp demarcation from surface mucosa [3, 6]. In contrast PBA originates from mucosa, mostly from posterior wall and trigone [3, 4]. In our case, a cystic mass at dome with positive frozen section suggested urachal adenocarcinoma. However resected specimen revealed a mucosal origin tumour involving dome and posterior bladder wall with evidence of cystitis cystica, ruling out urachal adenocarcinoma. IHC can't distinguish between two due to similar marker expression [11]. The most common differential is urothelial carcinoma with glandular differentiation, which must require foci of invasive or non-invasive urothelial carcinoma component and GATA3 positivity [1-3]. In our case, this was excluded due to absence of urothelial carcinoma component despite thorough sampling and negative GATA3 staining. Another key differential was secondary metastasis or direct spread from gastrointestinal

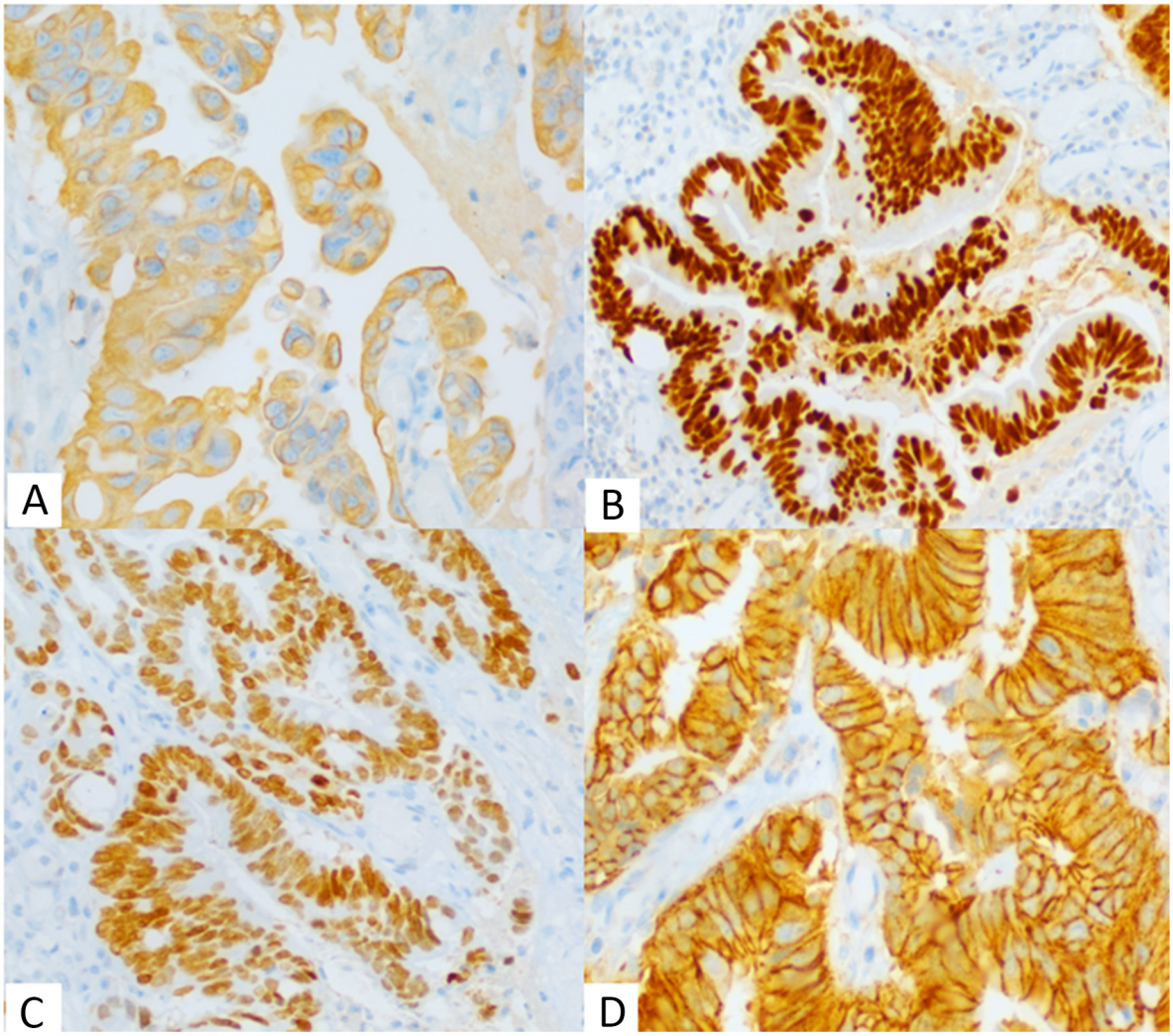

Figure 3. Immunohistochemistry showing tumour cells at 400x. (A) Cytoplasmic positivity for CK20, (B) Nuclear positivity for CDX2, (C) Nuclear positivity for SATB2 & (D) Nuclear negativity for beta catenin.

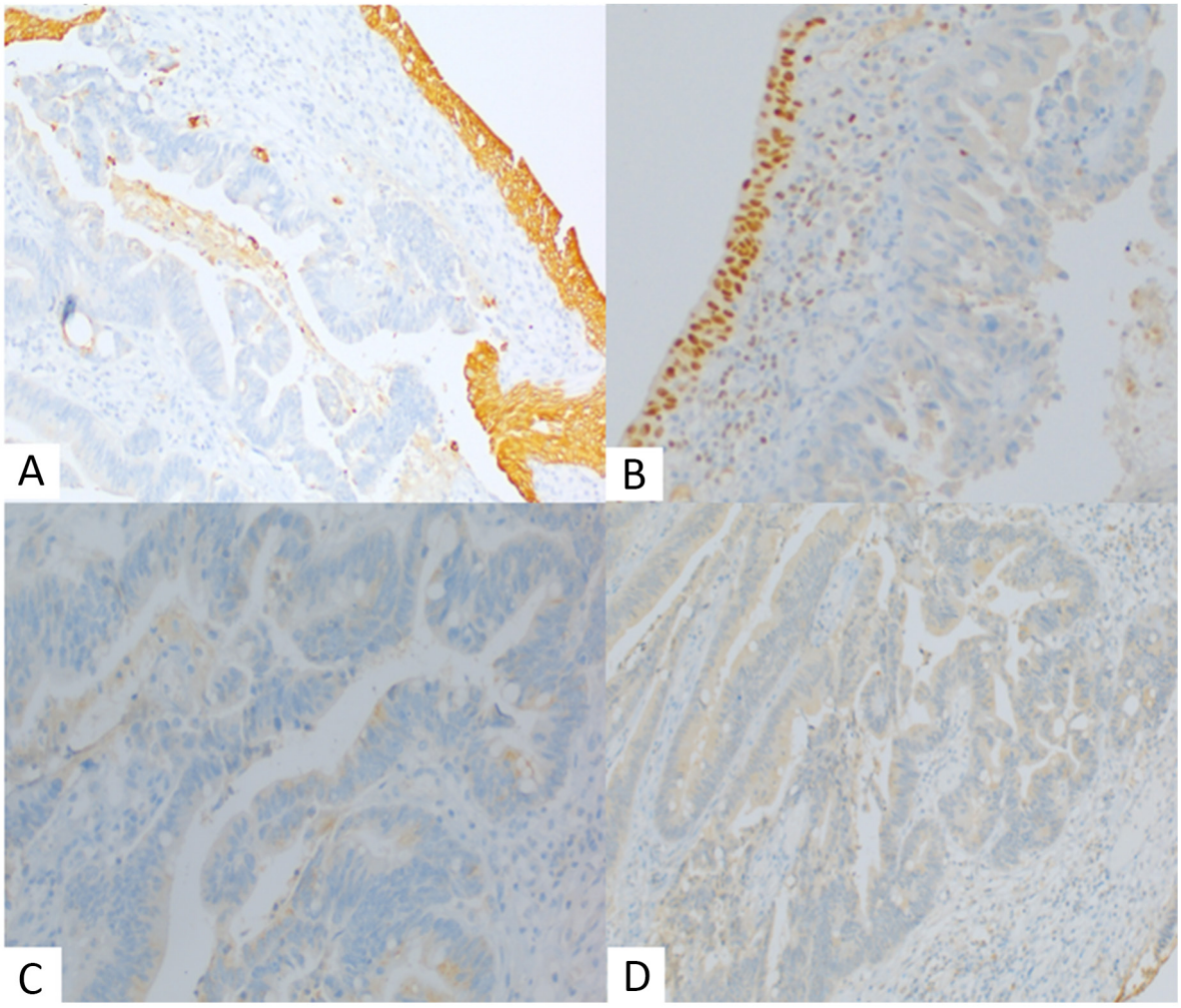

Figure 4. Immunohistochemistry showing tumour cells. (A) Cytoplasmic negativity for CK7 (100x, normal urothelium positive), (B) Nuclear negativity for GATA3 (100x, normal urothelium positive), (C) Negative for AMACR (400x), (D) Negative for PAX8 (100x).

or female genital tract. However, the tumour's mucosal origin with cystitis cystica, negative nuclear reactivity for beta-catenin, negative AMACR and normal colonoscopy rule out possibility of gastrointestinal adenocarcinoma metastasis [11]. Metastasis from female genital tract was also ruled out by Pax 8 negativity and unremarkable imaging. Mullerian origin adenocarcinoma was ruled out based on Pax8, AMACR negativity and the clinical context [11].

Limited data exist on the molecular profiling of PBA [12]. Most of PBA often shares genomic mutation such as KRAS, TP53, APC, PIK3CA with colorectal adenocarcinoma, indicating a common molecular pathway that may guide targeted therapies [2, 12].

Management depends on tumour stage and location. Surgery, including partial or total cystectomy is the mainstay for localized disease, while advanced cases may require chemotherapy or radiotherapy. In this case, the patient underwent partial cystectomy with clear margins and lymph nodes dissection. Prognosis remains poor, with a 5-year survival rate of 40-50%, influenced by stage and histological type [1]. Long-term follow-up is essential due to the potential for recurrence and metastasis.

Conclusion

This case highlights the diagnostic challenges of primary bladder adenocarcinoma presenting as a cystic mass, mimicking ovarian torsion. It underscores the need for clinicians to maintain a broad differential diagnosis when evaluating pelvic masses, particularly those with atypical clinical features. Given the rarity of this presentation, further studies are needed to clarify imaging characteristics and management guidelines for cystic bladder adenocarcinomas. Additionally, Molecular genetics remains an area for research that could lead to better management strategies.

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Ethical policy

Prior informed consent as well as consent for publication was taken from the patient.

Availability of data and materials

No new data were generated during this study.

Author contributions

Study concept and design: Dr. Sonali Kar; Data acquisition and analysis: Dr. Neeta Verma, Dr. Sonali Kar; Drafting of manuscript: Dr. Sonali Kar, Dr. Monali Kar; Critical revision of manuscript: Dr. Suren Kumar Das, Dr. Monali Kar.

Competing interests

The authors declare that they have no competing interests.

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