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# KRUKENBERG TUMOR: OVARIAN METASTASES FROM GASTROINTESTINAL TRACT – A RETROSPECTIVE CASE SERIES

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**Abstract**
Krukenberg tumors (KTs), metastatic ovarian carcinomas of gastrointestinal (GI) origin, frequently mimic primary ovarian neoplasms and pose significant diagnostic challenges. This retrospective case series evaluated the clinicopathological and immunohistochemical characteristics of confirmed KTs over a defined period. The objective was to characterize the origin, histological features, and metastatic patterns among affected patients. Clinical data, imaging, histopathology, immunohisto chemistry (IHC), and tumor marker profiles were systematically analyzed. The majority presented with abdominal masses and pain, with a mean age of 38.2 years. The most common primary sites were the appendix (35.7%) and gallbladder (25.0%). The most common histology was adenocarcinoma NOS (64.2%), followed by mucinous and signet- ring types. The peritoneum was involved in 75.0% and lympho- vascular invasion was seen in 42.8%. IHC showed robust CK20 (85.7%) and CDX2 (78.5%) immunoreactivity, with essentially universal PAX8 negativity, confirming GI origin. CA-125 was elevated in 66.7% of the patients but varied widely in extent. These findings underscore the importance of extensive histopathological and immunohistochemical analysis in distinguishing metastatic ovarian lesions from primary tumors. Accurate early detection and classification are of utmost importance for therapeutic planning and improved clinical results in patients with KTs.

**Keywords:** Krukenberg tumors, Ovarian metastases, Gastrointestinal carcinoma, Immunohisto chemistry, CA-125

## Introduction

Ovarian metastases from GI primaries constitute a distinct oncological entity with profound implications on prognosis and management. Among them, KTs—a condition initially described in 1896—designate metastatic ovarian lesions with mucin- producing signet- ring cells, most commonly

from the stomach, colon, and appendix.[1] KT refers to metastatic involvement of both ovaries, typically characterised by the presence of mucin- secreting cells, which is why it is also called carcinoma muco- cellular. However, in practice, the term "Krukenberg" is often used more broadly to describe any ovarian metastases, regardless of their primary site of origin.[2]

Despite having been classically and wrongly described as primary ovarian neoplasms, subsequent histopathologic, immunohistochemical, and molecular evidence has unequivocally established their secondary nature.[3] The incidence of KTs is low in worldwide cancer registry data, but the challenge they pose is disproportionate due to their indolent course, diagnostic delicacy, and failure to respond to conventional chemotherapeutic regimens.[4]

Pathogenesis remains unclear, although lymphatic and hematogenous dissemination have been proposed as mechanisms for the implantation of GI- derived mucinous adenocarcinoma cells within ovarian tissue.[2] These tumors typically occur in women of reproductive- age group but cases have also been reported in postmenopausal populations, clinically manifesting with insidious onset as pelvic masses, abdominal pain, or incidental discoveries during imaging or laparotomy.[5]

Clinical features of elevated serum CA-125, ascites, and bilateral ovarian involvement are commonly reported.[6,7] These frequently coincide with the diagnosis, further confounding clinical management.[8,9] Appendiceal primaries, though less common, have been a recent interest due to their occult nature and resemblance to primary ovarian tumors on imaging. Primaries from the gallbladder, duodenum and colorectum also widen the spectrum, necessitating a high index of suspicion in differential diagnosis.[10]

Current studies emphasize the heterogeneity of the primary sites in KTs, with gastric adenocarcinoma consistently reported as the most frequent source, followed by colorectal, appendiceal, and pancreato-biliary causes.[11] Histologically, the tumors reveal signet- ring morphology with mucinous differentiation.[12] Immunohistochemistry (IHC) is an essential modality to ascertain the origin of the primary tumor, with markers such as CK7, CK20, CDX2, and PAX8 offering differential diagnostic utility.[13] CDX2 and CK20/CK7 positivity are generally suggestive of GI origin, and PAX8 negativity helps to rule out primary ovarian carcinoma.[14] There is an overwhelming body of retrospective data to suggest that diagnosis is delayed and routine misclassification at presentation occurs, with metastatic disease being established only after careful histopathologic appraisal.[15] Further, survival outcomes are significantly poor, with a median survival of 12 to 24 months based on the primary source and extent of metastasis.[16] Diagnosis and accurate classification of KTs continue to be daunting issues in gynaecologic oncology.[17] Lack of uniform diagnostic criteria, morphologic overlap with primary ovarian malignancies, and restricted institutional experience have been accountable for underreporting and clinical mismanagement.[18] Despite the improvements in diagnostic imaging, surgical staging, and immunohistochemical protocols, there remains uncertainty regarding the optimum diagnostic approach and stratification of treatment.[19] This is an uncommon tumor type that also restricts large- scale prospective studies, such that retrospective analysis is by necessity the foundation for ascertaining patterns and outcomes. This study aims to retrospectively analyze the clinical and pathological profiles of patients diagnosed with KTs of GI origin, with a specific focus on identifying the primary site of origin, histopathologic subtypes, immunohistochemical expression patterns, and associated peritoneal or nodal dissemination. Through a systematic evaluation of institutional case records, this series seeks to contribute relevant evidence on diagnostic correlations and the metastatic behaviour of GI malignancies presenting as adnexal masses. Particular emphasis is placed on the role of IHC markers in differentiating metastatic ovarian tumors from primary epithelial ovarian neoplasms, along with assessment of tumor markers, peritoneal spread, and lymph node involvement.

## Materials and Methods

### 1. Study Design

This retrospective observational study was conducted at the Department of Pathology at Shri Ram Murti Smarak- Institute of Medical Sciences, SRMS- IMS. This study aimed to examine the

clinicopathological profiles of KTs, diagnosed between November 2022 and December 2024, considering their GI origins and corresponding ovarian metastases.

**2. Case Identification and Eligibility Criteria**

Archived histopathology reports were reviewed to identify female patients diagnosed with ovarian tumors of metastatic GI primary. Cases were included in the study if the ovarian lesion was histologically established to be metastatic and was related to immunohistochemical or clinical correlation with an established GI primary. The exclusion factors were unknown primary site tumors, histopathological characteristics indicative of primary ovarian malignancy, or lack of critical diagnostic or clinical information. A total of 28 cases that qualified according to these criteria were used in the final analysis.

**3. Histopathological Examination**

Formalin- fixed, paraffin- embedded ovarian tissue biopsy or resection surgery samples were procured from departmental archives. Routine Hematoxylin and Eosin (H&E) staining was performed on representative sections. All cases were examined by pathologists for evidence of characteristic features of metastatic disease, such as signet-ring cell morphology, mucinous differentiation, glandular architecture not suggestive of primary ovarian tumors, stromal invasion, lympho- vascular invasion and surface involvement. Bilateral ovarian involvement and the involvement of tumor deposits in peritoneal and omental tissues were also noted.

**4. Immunohistochemistry Protocol and Interpretation**

All of them were subjected to immunohistochemical analysis to determine tumor origin and exclude primary ovarian neoplasms. The IHC panel included CK7 and CK20 for cytokeratin expression profiling, CDX2 as an intestinal differentiation marker, and PAX8 as a Müllerian lineage marker. Other markers, such as WT1 and Estrogen Receptor (ER), were used to exclude serous ovarian carcinoma, whereas p53 and Ki-67 were used to assess tumor grade and proliferation index in some cases. Staining was assessed independently by two experienced histopathologists. Positivity for markers was assessed by cytoplasmic or nuclear staining in at least 10% of tumor cells. Diagnosis was established with a combination of histologic features, IHC profile, and correlation with known clinical history.

**5. Data Analysis and Statistical Approach**

Clinical and pathological data were logically organized in a preformatted chart for descriptive analysis. Categorical variables, including primary tumor site, histologic type, IHC marker expression, and extent of metastatic involvement, were summarized as frequency and percent. \

Continuous variables, including patient age and tumor size, were reported as means with their respective standard deviations. Due to the retrospective nature and limited sample size, there was no hypothesis testing or inferential statistics performed. Qualitative trending comparison of tumor origins was conducted to search for trends of interest in terms of presentation and progression.

**6. Ethical Consideration**

All practices were as per prevailing ethical standards for retrospective studies. Anonymization of all information was conducted before the corresponding analysis to retain the confidentiality of patients. There was no direct intervention or contact with patients. The experiment respected the moral principles in the Declaration of Helsinki as well as all norms of the institution related to the conduct of the study on human tissue.

## Results

### 1. Demographics and Clinical Presentation

Table 1 presents the age- related distribution and clinical symptoms of the patients with KTs. The 28 analyzed patients had an average age of about 38.2 years, between the ages of 25 and 64 years. The most frequent point of origin was the appendix (n=10) followed by gall bladder (n=7).

**Table 1:** Demographic and Primary Tumor Distribution

| **Parameters** | | **Value** | **Interpretation** |
|---|---|---|---|
| Age | Mean ± SD | 38.2 ± 11.5 | Reproductive age dominance |
| | Range (in years) | 25–64 | Adults |
| Primary sites | Appendix | 10 (35.7%) | Most common |
| | Gall bladder | 7 (25.0%) | The second most common |

### 2. Histological Morphology

Table 2 shows the histopathological categories and the status of LVI. Histologic analysis revealed adenocarcinoma with maximum frequency of 64.2% and signet-ring cell carcinoma with 14.2%. Out of the total patients with adenocarcinoma, LVI was present in 42.8%.

**Table 2:** Histologic Type and LVI Presence

| **Histologic Type** | **n (%)** | **LVI Present (n, %)** |
|---|---|---|
| Adenocarcinoma | 18 (64.2%) | 12 (42.8%) |
| Signet- ring cell | 4 (14.2%) | 3 (10.7%) |
| Mucinous carcinoma | 6 (21.4%) | 3 (10.7%) |

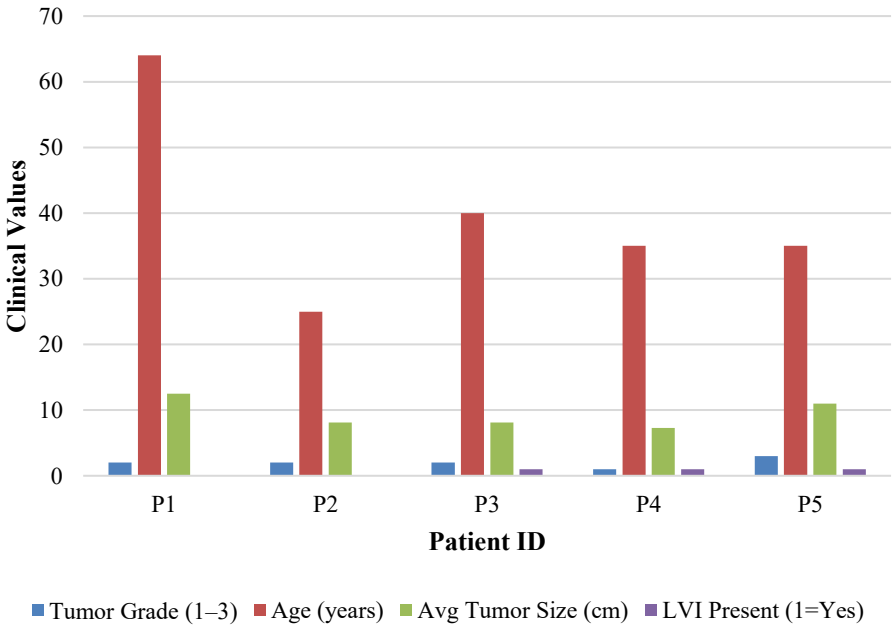


**Figure 1:** Comparative Profile of Tumor Grade, Age, Tumor Size, and LVI in KT Cases

Figure 1 shows a patient- based comparison of the grade of tumor, patient ages, their average tumor size, and the presence/ absence of LVI. The maximum age at which a patient has reached is 64 years (P1), and the largest tumor was 12.5 cm. LVI was reported in 3 cases, and all of them with moderate or high- grade tumors (grade 2 and 3).

### 3. Patterns of Metastatic Spread

Table 3 shows the extent of metastatic spread to the peritoneum, omentum, and lymph nodes. Peritoneal involvement was present in 75% (21/28) of the patients. Omental involvement was histologically confirmed in 67.8% (19/28). 13 patients underwent dissection of lymph nodes; 4 had positive pelvic nodes. Peritoneal involvement was the most frequent, suggesting the diffuse nature of the disease in most patients.

**Table 3:** Metastatic Spread Profile

| Site of Spread | Positive Cases (n) | Percentage (%) |
|---|---|---|
| Peritoneum | 21/28 | 75.0% |
| Omentum | 19/28 | 67.8% |
| LN Involvement | 4/13 dissected | 30.7% |

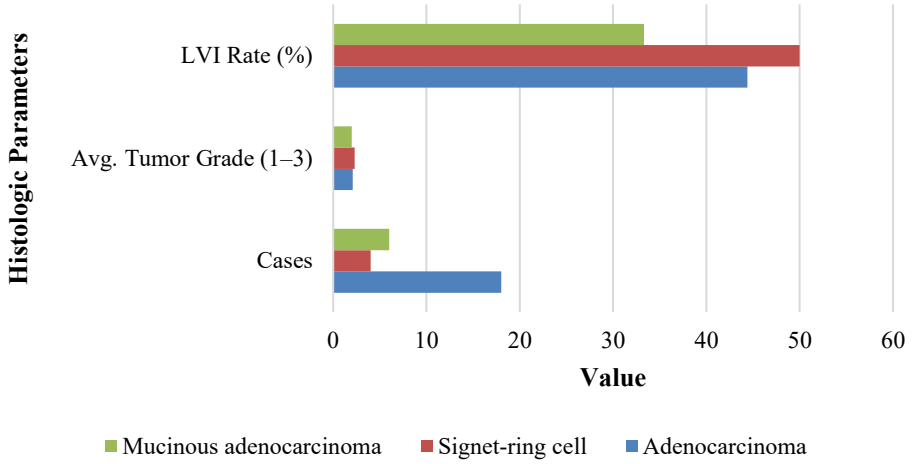


**Figure 2:** Comparative Histologic Profile of Krukenberg Tumors by Type

Figure 2 shows an array of histologic characteristics of three kinds of tumors. The most number of cases were adenocarcinoma (18), second, mucinous adenocarcinoma (6) and third, signet ring cell carcinoma (4). The mean grade of the tumor ranged closely between 2.0 and 2.3, whereas the LVI positivity was highest in signet- ring cell carcinoma (50%), and the lowest in adenocarcinoma (44.4%).

### 4. Immunohistochemical Profile

Table 4 shows immunohistochemical patterns of expression in KTs. Strong CK20 and CDX2 positivity guaranteed GI origin, and PAX8 negativity excluded primary ovarian cancer.

CK20 and CDX2 were positive in 85.7% and 78.5% of cases, respectively, indicating intestinal origin. CK7 was focally or patchy positive in 42.8%, while PAX8, a classic ovarian tumor marker, was negative in 96.4%, again supporting the metastatic nature. All of these findings demonstrate a consistent IHC pattern useful in the differentiation of KTs from primary ovarian cancers, particularly in a scenario where morphology is not definitive.

**Table 4:** IHC Marker Positivity Rates

| Marker | Positive Cases (n) | Percentage (%) |
|---|---|---|
| CK20 | 24/28 | 85.7% |
| CDX2 | 22/28 | 78.5% |
| CK7 | 12/28 | 42.8% |
| PAX8 | 1/28 | 3.5% |

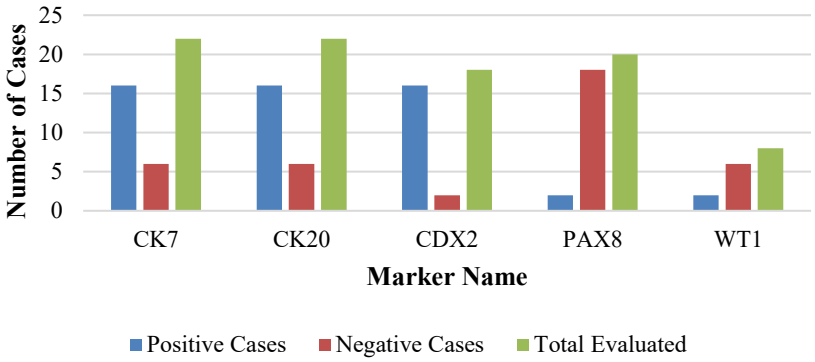


**Figure 3:** Frequency of IHC Marker Expression in KT Cases

Figure 3 shows the distribution of IHC marker- positive and negative in certain cases. The presence of CK7, CK20, and CDX2 gave 16 positives representing non- Mullerian origin, and PAX8 gave 18/20 negatives, indicating non- Mullerian origin. The marker that was least assessed was WT1, in just 8 cases and gave 6 negative results.

**5. Tumor Marker Analysis (CA-125)**

Table 5 shows a statistical overview of serum CA-125 levels in patients. The majority presented with elevated levels, but variability means that CA-125 is supportive, but not definitive, in diagnosis. Serum concentrations of CA-125 were determined in 24 patients. The mean was 1023.6 U/ml, and the median was 102.5 U/ml with a right- skewed distribution caused by a few very high levels. The lowest recorded value was 7 U/ml, and the uppermost was 6890 U/ml. 16 patients (66.7%) had levels greater than the normal upper limit of 35 U/ml, frequently associated with widespread peritoneal dissemination.

**Table 5:** CA-125 Level Summary

| Parameter | Value | Interpretation |
|---|---|---|
| Mean CA-125 (U/ml) | 1023.6 | Elevated in most cases |
| Median CA-125 (U/ml) | 102.5 | Central tendency lower |
| Range (Min–Max) | 7–6890 | Wide biological variation |
| Above Normal (>35 U/ml) | 16 cases (66.7%) | Frequent but not universal |

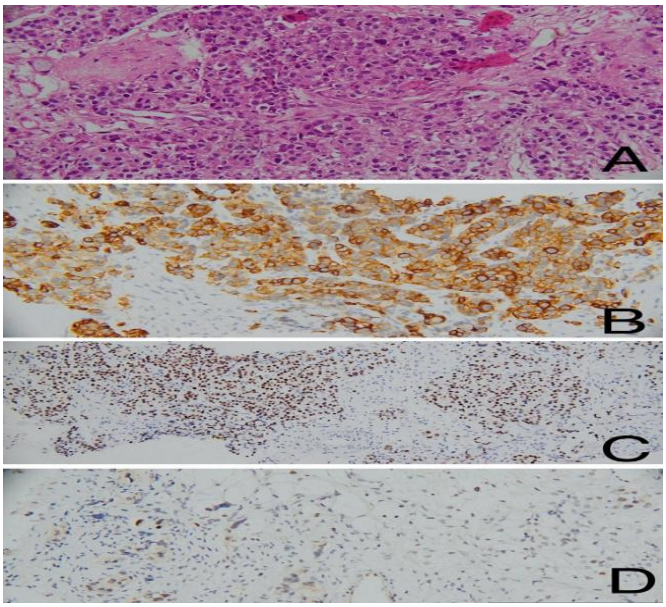


**Figure 4: A.** Photomicrograph showing adenocarcinoma of ovary (H&E staining, 40x) Immunohistochemical analysis, **B.** Membranous expression of CK7 (40x); **C.** Nuclear expression of CDX2 (20x); **D.** Negative staining with PAX8 (40x)

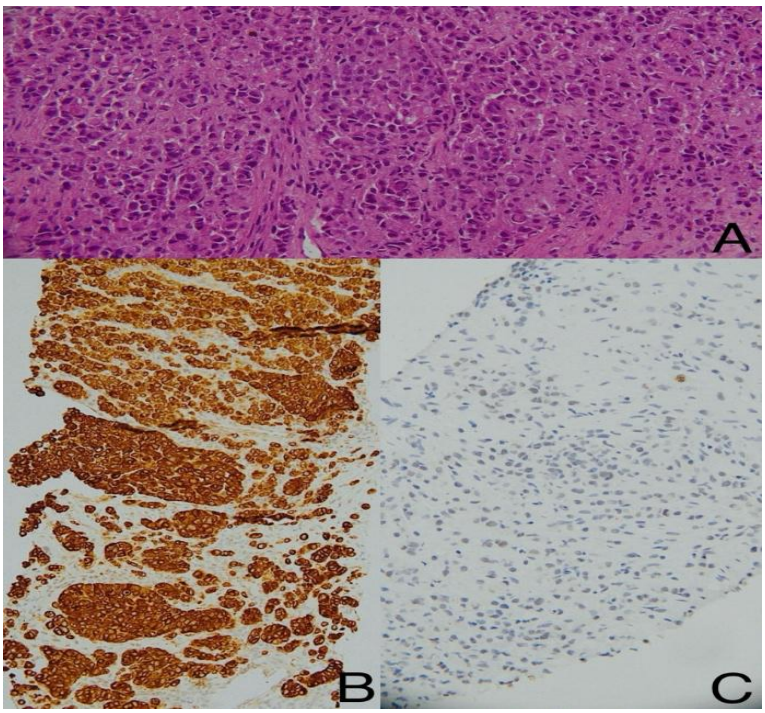


**Figure 5: A.** Photomicrograph showing poorly differentiated signet ring cell carcinoma of ovary (H&E staining, 40x) Immunohistochemical analysis, **B.** Positive strong membranous expression with CK7; **C.** Negative staining with PAX8.

**Discussion**

This case series retrospectively describes the clinicopathological profile of KTs to be multifaceted, having a predilection at reproductive age, with an average of 38.2 ± 11.5 years and ranging from 25 to 64 years. The initial symptom was most commonly abdominal pain in 85.7% of the cases, followed by ascites and abdominal mass. Appendix (35.7%) and gallbladder (25.0%) were observed to be the most common primary sites, deviating from the conventional literature, where gastric primaries are the most common (Table 1).

Histologically, the most frequent subtype was adenocarcinoma (64.2%), followed by mucinous carcinoma (21.4%) and signet- ring cell carcinoma (14.2%), and LVI was seen in 42.8% of the cases (Table 2). Signet- ring cell carcinoma also had the highest frequency of LVI at 50%, indicating aggressive behaviour. This was also seen from the correlation of tumor grade– LVI shown in the figure comparing the tumor grade, age, tumor size, and status for LVI, wherein LVI was more frequently seen in moderate and high- grade tumors. Widespread metastatic dissemination was observed: 75.0% of the patients had peritoneal deposits, 67.8% had omental nodules, and 30.7% of the patients who underwent lymphadenectomy had positive pelvic nodes (Table 3). These findings are in keeping with the tendency for trans- coelomic dissemination and nodal spread and reflect the importance of thorough staging at presentation.

IHC pattern confirmed GI origin in most cases: positivity for CK20 in 85.7%, CDX2 in 78.5%, and CK7 in only 42.8%. PAX8, a Müllerian lineage marker indicating primary ovarian tumors, was negative in 96.4%, supporting the metastatic nature of these lesions (Table 4). Such IHC patterns are indispensable tools in distinguishing KTs from primary ovarian neoplasms, especially in the presence of morphological overlap. Serum CA-125 levels were extremely variable, ranging from a mean of 1023.6 U/ml and a median of 102.5 U/ml. While 66.7% of patients had elevated levels (>35 U/ml), a few high- grade tumors presented with normal CA-125, which limits its diagnostic sensitivity (Table 5). The extreme range (7 to 6890 U/ml) reiterates that CA-125 should be used cautiously and only as a component of radiologic and histopathologic data.

The present study highlights the necessity for a high index of suspicion for metastatic GI tumors in patients presenting with bilateral adnexal masses, especially with mucinous histology and signet- ring cell features. The dominance of appendiceal and gallbladder primaries reported here is atypical and

implies that atypical GI sources must be considered in differential diagnosis, especially in regions of mixed patterns of cancer incidence. The stable IHC pattern—CK20+, CDX2+, PAX8− is a robust diagnostic algorithm. However, sole reliance on CA-125 may be misleading with the inhomogeneous expression in grades of tumors and metastatic index. Therefore, multimodal assessment with integration of clinical, radiological, histological, and immunohistochemical data is still necessary to avoid misclassification and provide appropriate management.

Compared to prior studies, this series had a greater proportion of primaries of appendiceal and gallbladder origin, in contrast to gastric origin which is generally perceived to be the most frequent.[20] The pattern of histology is as would be anticipated with the majority of mucinous and signet- ring cell patterns, though the proportion of signet- ring cases in this collection (14.2%) is marginally lower than reported by other series.[21] The incidence of peritoneal and omental dissemination reported (75.0% and 67.8%, respectively) accords with earlier descriptions characterizing KTs as advanced metastases with extensive intra- abdominal spread.[22] The IHC findings reflect known patterns, namely the consistent presence of CK20 and CDX2 and the replicable lack of PAX8. However, the intermediate frequency of CK7 positivity (42.8%) emphasizes the potential for diagnostic ambiguity with an IHC panel of limited breadth.[23]

## Conclusion

KTs represent an unusual but clinically significant presentation of metastatic GI carcinomas to the ovaries, often in the form of primary ovarian neoplasms. This case series demonstrates the characteristic clinicopathologic pattern of these tumors among a regional population with remarkable trends toward primary tumor site of origin, histologic architecture and pattern of dissemination. A predominance of appendiceal and gallbladder primaries over the classically expected gastric origin underscores the necessity of appropriate GI workup in all putative metastatic ovarian tumors. Histologically, adenocarcinoma remained the most frequent histotype, being accompanied by mucinous and signet- ring types also significantly. Lymphovascular invasion, peritoneal and omental implants, and lymph node metastases further reflected the virulent course of these neoplasms and the advanced stage at presentation. Immunohistochemical stains such as CK20 and CDX2 were very sensitive for GI origin, and the lack of expression of PAX8 was good to exclude primary ovarian tumors, thus making IHC a precious technique in the diagnostic approach. Inadequacy of specificity by CA-125, even with the raised levels in the majority of cases, renders it even more necessary to be employed in addition to imaging and histopathology. Surgical staging often returned a profound burden of disease, emphasizing the need for timely identification and early treatment. Increased recognition of atypical metastatic patterns and histologies is vital in improving prognostication and tailoring therapy in patients with KTs.

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