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(Case Report)

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Sertoli-Leydig tumor of the ovary with an extensive heterologous intestinal mucinous component. Case report and a brief review of the literature

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Abstract

Sertoli-Leydig cell tumors (SLCTs) represent an uncommon group of ovarian sex cord-stromal neoplasms constituting less than 0.5 percent of all ovarian neoplasms. Heterologous elements, especially of intestinal type, are rare, and usually form small component. We present a diagnostically difficult case of an SLCT that has an exceptionally large intestinal heterologous component that resembles a mucinous epithelial neoplasm.

A 67-year-old woman complained of progressive abdominal distension, pelvic pain, and virilization. A solid-cystic adnexal mass was detected by imaging. The laboratory workup revealed a high level of serum testosterone and normal tumor markers. The intraoperative frozen section was not conclusive, and the concern was a possible mucinous epithelial tumor. Conclusive histopathology identified a moderately differentiated SLCT with large (about 30 per cent) intestinal-type mucinous components. Positive reaction with Inhibin, calretinin, SF1, FOXL2, and CDX2 supported the diagnosis. The staging was initially FIGO IA, but after intraoperative communication clarified the possibility of a tumor spillage, the final staging was revised to IC1, which was later confirmed by positive peritoneal cytology. The patient received adjuvant platinum-based chemotherapy and was disease-free at 28 months.

This case demonstrates that the presence of a large amount of heterologous mucinous intestinal component may cause diagnostic errors, especially with limited sampling or during frozen section intraoperative consultation. It highlights the importance of being cautious when making intraoperative diagnosis where the findings are uncertain to prevent any unneeded surgical procedures. Above all, it points out the significant role of multidisciplinary communication in the process of correct staging and efficient management of rare ovarian neoplasms.

Keywords: Sertoli-Leydig; Sex cord-stromal neoplasms; Heterologous intestinal element; Immunohistochemistry

1. Introduction

Sertoli-Leydig cell tumors (SLCTs) are uncommon sex cord-stromal ovarian tumors. They represent less than 0.5 percent of all ovarian neoplasms. It normally occurs in young women between the ages of 20 and 30. [1] These tumors are usually benign, and the prognosis is good in case of early diagnosis. However, approximately 10-20 percent may be malignant, and there is a chance of recurrence of the disease even following initial treatment. SLCTs are characterized by their ability to differentiate along testicular lines and, therefore, often lead to androgen production and ensuing virilizing symptoms in those affected by it. [2]

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Although SLCTs are typically well differentiated to a certain degree, the presence of heterologous components, particularly those of intestinal mucinous nature, is extremely rare and may be a serious diagnostic challenge. [3] The uniqueness of such manifestations contributes to the notion that it is quite challenging to classify and treat such rare malignancies in the ovaries appropriately.

A physical examination and hormonal tests are common initial steps to diagnosis, and they usually show increased testosterone levels. A major initial step is pelvic ultrasound, which in many cases reveals a solid or solid cystic unilateral ovarian mass. The gold standard definitive diagnosis is by histopathological examination of the resected tumor. [4]

Surgical resection is the main treatment of SLCT. With early-stage disease, the procedure of choice to maintain fertility is the unilateral salpingo-oophorectomy. With more advanced or malicious tumors, a more radical surgery might be needed, including a complete hysterectomy and bilateral salpingo-oophorectomy. [5] The outcome of benign SLCTs is superb after surgery. In case of malignant variants, the prognosis is worse, and close monitoring and possible adjuvant treatment may be necessary. [6]

This case highlights the tremendous importance of thorough histopathologic examination, massive immunohistochemical analysis, and interdisciplinary communication in reaching an accurate diagnosis, adequate staging, and effective management strategy of unusual ovarian neoplasia with atypical features. In addition, it demonstrates the significance of increasing awareness of these unusual presentations to prevent misdiagnosis and obtain the most favorable patient outcomes.

2. Case presentation

A 59-year-old postmenopausal woman presented to the gynecology department at the recommendation of her primary care provider with a history of worsening abdominal bloating as well as chronic pelvic pain and new-onset virilization. During the last six months, she had experienced growing hair on her face, chest, and limbs. Her medical and family histories were unremarkable. Physical examination revealed prominent hirsutism and a large palpable abdominal mass. In laboratory testing, the level of serum testosterone was found to be high, and other tumor markers such as DHEAS, CA-125, CA-19, CA-15-3, and AFP were normal.

Pelvic ultrasound showed a solid-cystic mass of dimensions 12 x 8 cm that is of probable left adnexal origin and has lower echogenicity compared with other tissues. No pelvic free fluid, no peritoneal nodularity was reported. There was no evidence of peritoneal disease or lymphadenopathy on the MRI. The solid component showed low signal intensity on T1W and low to intermediate signal intensity on T2W images, and the cystic part was hyperintense on T2W and hypointense on T1W images. The virilizing symptoms and the elevated level of testosterone were suggestive of a steroid-secreting ovarian tumor, particularly a Sertoli-Leydig cell tumor (SLCT), though the radiological imaging was unclear.

The differential diagnoses considered included an androgen-secreting ovarian neoplasm or an epithelial neoplasm. The case was presented to a multidisciplinary tumor board, and surgical management was recommended. A total hysterectomy (TH), bilateral salpingo-oophorectomy (BSO), and extensive staging were performed, which involved pelvic and para-aortic lymphadenectomy up to the renal vessels, as well as peritoneal washings and omental biopsy. The excised left fallopian tube and ovary were grossly examined to show a solid 12 cm tan-yellow mass. The frozen section analysis was not conclusive, showing that there was mucinous-looking columnar epithelium, raising suspicion of a mucinous epithelial tumor. The final diagnosis was deferred to the permanent sections.

Microscopic examination showed that large, variably sized cystic spaces lined by tall mucinous columnar cells, some of which were goblet-like in appearance, occupied large parts (estimated at 30 percent of tumor volume) of an atrophic ovarian stroma. These characteristics were in favor of heterologous intestinal differentiation in an SLCT rather than a mucinous epithelial neoplasm. Typical sex cord features, such as cords and trabeculae of Sertoli-like cells, were seen in other areas of the tumor. The tumor cells in these areas showed round nuclei, eosinophilic cytoplasm, and few mitotic figures. There were scattered Leydig cells, but no Reinke crystals were found. (Figure 1 A, B, C, D)

Immunohistochemical (IHC) analysis was essential to establish the diagnosis. The tumor cells were positive for Inhibin, calretinin, SF1, FOXL2, WT1, CD56, CD99, vimentin, and cytokeratin Cam5.2 (focally)—Leydig cells stained for Melan A and MART1. The heterologous mucinous component stained positively for CK20 and CDX2, confirming intestinal differentiation, and was negative for CK7, EMA, and PAX8. Molecular testing was negative for DICER1 mutations. The final diagnosis was a moderately differentiated Sertoli-Leydig cell tumor, 12 x 8 cm with prominent heterologous intestinal-type mucinous elements (30%), confined to the ovary, without surface involvement or lymph node

metastasis. The fallopian tube was uninvolved, but pelvic washings were positive for malignant cells. Initially staged as FIGO IA based on pathology, the stage was revised to IC1 following communication from the surgeon regarding intraoperative tumor spillage. The positive cytology confirmed this revised staging.

Given the tumor's size and the extensive heterologous component, the patient was started on adjuvant platinum-based chemotherapy (carboplatin and paclitaxel), which she tolerated well. Her postoperative course was uneventful. At the 28-month follow-up, she remained disease-free, with no evidence of recurrence.

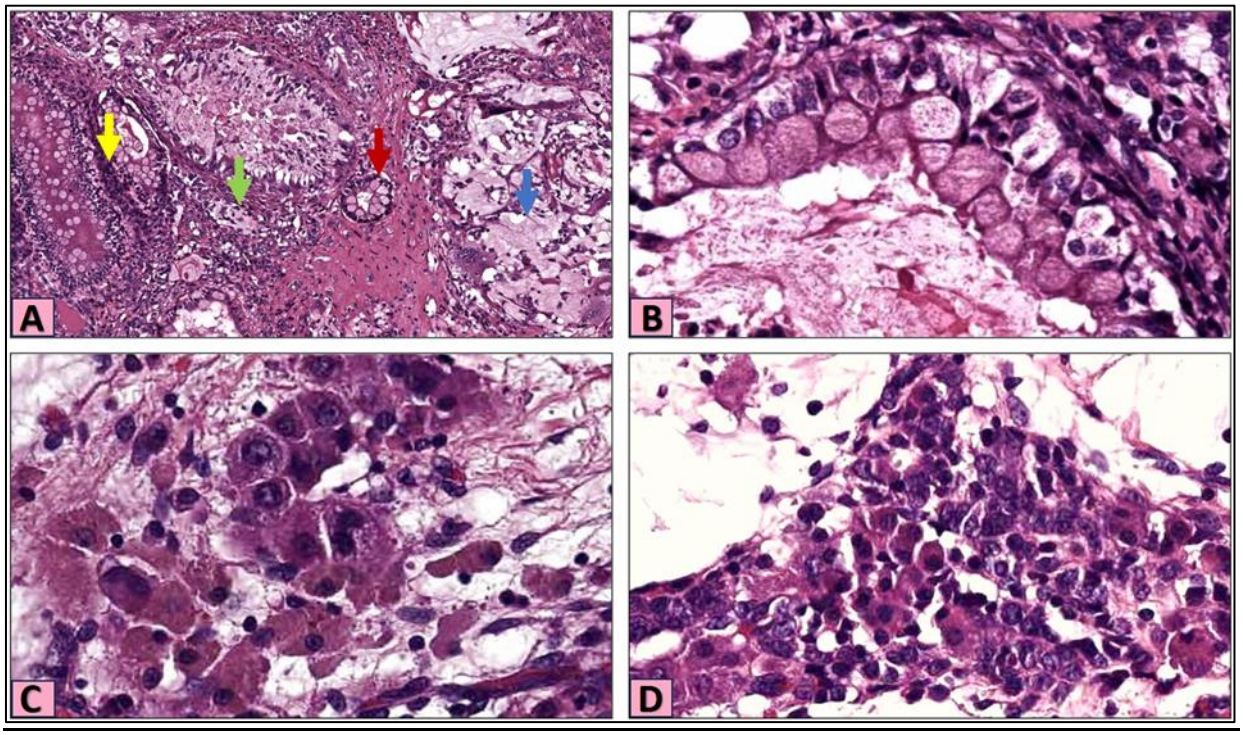

Figure 1 Histomorphology of spindle cell oncocytic pituicytoma (SCOP)

- **1A** Low power view showing lobulated architectural pattern, and the cellular components of the tumor, trabeculae of Sertoli cells (Yellow arrow), scattered Leydig cells (Green arrow), intestinal metaplasia (Red arrow), and atrophic ovary (Blue arrow) (H&E stain X20)
- **1B: High** power view showing intestinal metaplasia with prominent goblet cell features (H&E stain X60)
- **1C:** High power view showing Leydig cells with round nuclei and abundant eosinophilic cytoplasm (H&E stain X60)
- **1D**: High power view showing a mixed collection of Leydig and Sertoli cells (H&E stain X40)

3. Discussion

3.1. History

Sertoli Leydig cell tumors (SLCT) are rare ovarian neoplasms that mimic the cellular structure and endocrine function of testicular Sertoli and Leydig cells. [7] These neoplasms were initially recognized in the early 1900s and have remained diagnostically challenging tumors due to their variable morphology and hormonal activity. Most produce androgens, leading to clinical features such as hirsutism, voice deepening, or amenorrhea. [8] In our case, a 59-year-old postmenopausal woman presented with gradually worsening abdominal bloating and new onset virilization, including increased facial and body hair. The late age of onset and the presence of a large complex adnexal mass initially pointed toward a mucinous epithelial neoplasm. However, the combination of androgenic symptoms and elevated serum testosterone levels appropriately raised suspicion for a steroid-producing tumor.

3.2. Epidemiology

SLCTs are extremely rare, reported as fewer than 0.5 percent of all ovarian neoplasms. [9] They most commonly affect women under 40 years of age, with a peak incidence in the second and third decades of life. [10] Postmenopausal

Presentation, such as in our patient, is atypical and can obscure the clinical picture. While virilization is observed in up to half of patients, the initial workup often centers around imaging findings that may suggest more common epithelial pathologies. This was true in our case, where the solid cystic nature of the mass and its mucinous appearance on the frozen section led to a preliminary impression of a mucinous epithelial tumor. Only permanent sections with thorough immunohistochemical staining confirmed the diagnosis.

3.3. Risk Factors

There have been no well-established risk factors for Sertoli-Leydig tumors. Most of the cases are sporadic and have no association with hormonal treatments, reproductive history, or environmental exposures. In some tumors, especially those of moderate or poor differentiation, DICER1 mutations have been associated, whether germline or somatic. [11] In our case, testing for DICER1 was negative, and both the patient's personal and family medical histories were non-contributory. Her medical and family histories were unremarkable. The presence of a large mucinous heterologous component, while rare, did not appear to be linked to any known syndromic or hereditary predisposition in this instance.

3.4. WHO Classification

According to the 2020 World Health Organization classification of female genital tumors, SLCTs fall under the category of ovarian sex cord stromal tumors. [12] They are subclassified into well, moderately, or poorly differentiated forms, with prognosis and behavior correlating to the degree of differentiation. Tumors with heterologous elements, such as mucinous epithelium or skeletal muscle, are more frequently associated with intermediate or poor differentiation and a higher risk of recurrence. [3] In our patient, the tumor was classified as moderately differentiated and exhibited an unusually prominent intestinal-type mucinous component estimated to comprise approximately 30 percent of the total tumor volume. This large heterologous element is well above the typical range seen in SLCTs and significantly contributed to the diagnostic challenge, particularly during frozen section consultation. Recognition of characteristic Sertoli tubules, Leydig cells, and supportive Immunohistochemistry was essential in reaching the final diagnosis.

3.5. Clinical Presentation and Imaging Findings

Clinical manifestation of SLCTs is extremely diverse and tends to vary depending on the hormonal activity of the tumor. A considerable percentage of such tumors secrete androgens, and this causes virilization symptoms among the affected individuals. [13] The most common presenting signs and symptoms are progressive abdominal distension, abdominal pain, and irregularities of menstrual periods in the form of amenorrhea or oligomenorrhea. [13] [14] When there is production of androgen, the patients might show signs of defeminization followed by gradual masculinization. Such virilization may take the form of hirsutism, and a few SLCTs can also create estrogen, which causes such symptoms as precocious puberty in younger patients or abnormal uterine bleeding in adults. However, the more typical is the virilizing Presentation. [15]

Imaging studies are very important in the preliminary assessment of suspected ovarian masses. First-line imaging is usually a pelvic ultrasound. SLCTs are found in adnexal masses, which are mostly solid-cystic, and the proportion of solid and cystic parts differs. [4] The solid parts can be less echogenic, and the cystic parts can be anechoic or with internal echoes about the contents. The existence of large blood flow in the solid components, as seen in color Doppler ultrasound, may show vascularity of these tumors. [4]

Magnetic Resonance Imaging (MRI) is more specific in terms of the anatomy and useful in the characterization of the mass and its extent. Solid components of SLCTs tend to appear with low signal intensity on T1-weighted (T1W) images and low to intermediate signal intensity on T2-weighted (T2W) images on MRI. [16] In case they exist, the cystic parts look hyperintense on T2W and hypointense on T1W. The solid components and patterns of enhancement can also be characterized using contrast-enhanced MRI. [16] Although these imaging characteristics are suggestive, they tend to be non-specific. They may overlap with other ovarian pathologies, and their imaging characteristics do not allow a specific diagnosis.

Imaging differential diagnoses of ovarian mass with virilizing symptoms include other androgen-secreting ovarian tumors, including Leydig cell tumors, and adrenal rest tumors. [8] Furthermore, solid-cystic neoplasms of the ovary may also include mucinous epithelial tumors, granulosa cell tumors, and even metastatic tumors. They must be included in the differential when hormonal symptoms are less apparent or absent. [7] [16] The heavy heterologous component that is observed in the presented case can also complicate the imaging differential diagnosis since it can be confused with other types of tumors, especially mucinous epithelial neoplasms, and create difficulties when making a diagnosis based

on intraoperative evaluation. [16] Thus, a complex of clinical manifestations, laboratory results (high levels of testosterone), and histopathology is required to make a proper diagnosis.

3.6. Pathology findings: Gross, microscopic, immunohistochemical, and molecular features

Grossly, ovarian SLCTs are generally unilateral, solid, or solid-cystic masses with the size of the tumor being highly variable (usually between several cm to more than 20 cm). [16] [17] The lipid nature of the Leydig cells is typically tan-yellow to golden brown on the cut surface. It may have hemorrhagic, necrotic, or cystic degeneration areas. [4] [17] The consistency may be firm or soft, according to the cellularity and the presence of cystic areas. [17]

Microscopically, the tumor appears to form well-defined tubules, resembling testicular tubules, interspersed with Leydig cells containing eosinophilic cytoplasm and sometimes Reinke crystals. [7] SLCTs are represented by a dual population of Sertoli cells and Leydig cells in different patterns. Mature Sertoli cells with well-formed tubules that resemble the testicular tubules are characteristic of well-differentiated tumors, which are interspersed with Leydig cells with eosinophilic cytoplasm and occasionally with Reinke crystals. [7] The patterns in intermediate and poorly differentiated tumors are less organized, and Sertoli cells are organized into cords, nests, or diffuse sheets, and Leydig cells are less prominent or absent. They may include heterologous elements (tall mucinous columnar cells with goblet-like in appearance, as in the given case, or cartilage, and skeletal muscle), which may complicate the diagnosis, especially in intermediate and poorly differentiated tumors. [3] [16]

SLCTs are immunohistochemically positive with sex cord-stromal markers. Inhibin-alpha, calretinin, SF-1, and FOXL2 are usually Sertoli cell positive. [16] [18] Leydig cells also react positively with inhibin-alpha, calretinin, and steroidogenic enzymes such as Melan-A and MART-1. [16] Epithelial immunohistochemical stains (e.g., cytokeratins, e.g., CK7, Cam5.2) are typically negative or focally positive in the sex cord elements, and this distinction can be used to distinguish SLCTs and epithelial ovarian tumors. The presence of the heterologous mucinous component, when present, generally expresses the intestinal markers such as CK20 and CDX2, which will verify its differentiation. [16]

At the molecular level, a large percentage of SLCTs, especially the poorly differentiated ones or harboring heterologous components, are linked to mutations in the DICER1 gene. [14] [19] DICER1 is a tumor suppressor gene encoding the microRNA processing enzyme that is associated with mutations causing a syndrome that predisposes to multiple tumors, including SLCTs. The other molecular changes are not as consistently present [14], and studies are ongoing to identify further genetic drivers and pathways in SLCT development and progression, which will potentially result in more specific treatment.

3.7. Treatment and outcome

The primary treatment for SLCTs is surgery, based on the patient's age, disease stage, and tumor grade. Preserving fertility is a major problem that arises when diseases strike patients who are of reproductive age. Fertility is preserved through cystectomy or adnexectomy while the remainder of the female genital tract remains intact. [20] Although the necessity of a lymphadenectomy is still up for debate, the best course of action for patients who are past childbearing age is a hysterectomy with bilateral salpingo-oophorectomy and staging surgery (cytology, omentectomy, pelvic-paraaortic lymphadenectomy, or peritoneal biopsies). [21] Brown *et al* (w) demonstrated that in SLCTs, lymph node metastasis is markedly reduced, and sampling of retroperitoneal nodes may be omitted. [22] However, fertility-sparing surgery, with or without staging surgery, is necessary for young women, especially those with stage I disease. [21] After surgery, chemotherapy may be used, especially for tumors with poor or moderate differentiation. Adjuvant chemotherapy has no discernible benefits, according to Gui *et al*, and should only be taken into consideration in cases of disease recurrence or tumors that are poorly or moderately differentiated. [10]

Furthermore, a prior study (MITO) demonstrated that patients with advanced stage (II–IV) disease benefited from postoperative chemotherapy. [23] The extent and degree of tumor differentiation determine the prognosis. Malignancy is found in 59% of poorly differentiated tumors. SLCTs (10-year survival rate: 41%) and 11% of moderately differentiated SLCTs (10-year survival rate: 87%). In addition, stage I disease patients have a nearly 92% 5-year overall survival rate. [21]

3.8. Pathogenesis and Genetic Mutations of SLCT

The pathogenesis of SLCTs is complicated and still not well understood. However, recent studies have illuminated the most important genetic pathways, especially the mutations in the DICER1 gene. [14] The origin of SLCTs is postulated to be the sex cord-stromal cells of the ovary, which can differentiate along the testicular lines. [19] The precise processes

involved in the induction of this aberrant differentiation and the subsequent development of tumors remain a subject of research. However, genetic predisposition is a major factor in a minority of cases. [14]

The mutations of the DICER1 gene are among the most important genetic correlations with SLCTs. [24] [25] DICER1 is a tumor suppressor gene coding an endoribonuclease that processes microRNAs (miRNAs), important regulators of gene expression. A large percentage of poorly differentiated, retiform, and heterologous SLCTs have germline and somatic mutations in DICER1. [14] [25] [26] Such mutations are usually truncating or missense mutations altering the RNase IIIb region of DICER1 protein, thereby compromising miRNA processing and misregulation of numerous cellular processes related to cell proliferation, differentiation, and apoptosis. [14] [26]

DICER1 mutation in SLCTs is also associated with a more general tumor predisposition syndrome termed DICER1 syndrome, which also encompasses a range of other rare tumors, including pleuropulmonary blastoma, cystic nephroma, and thyroid nodules. [27] This relationship underscores the systemic effect of DICER1 malfunction on cancer development. Although DICER1 mutations comprise a significant genetic feature of most SLCTs, it is worth noting that not every SLCT has this mutation, as is the case in our report, especially the well-differentiated ones. [24] This is an indication that other genetic or epigenetic changes might also be involved in causing SLCTs even without DICER1 mutations. Further molecular pathways and genetic factors that play a role in the variation of clinical and pathological features of these rare ovarian neoplasms are still being investigated.

3.9. What did we learn from this case?

This is a case of SLCT with an abundant heterologous intestinal mucinous component, highlighting several lessons likely relevant clinically. In the first place, it highlights the difficulty in reaching a diagnosis of rare ovarian neoplasms when they manifest with atypical features of prominent heterologous elements. The initial misinterpretation during frozen section emphasizes the importance of waiting for permanent histology results with uncertain intraoperative findings to avoid unnecessary surgical procedures. Second, the case illustrates the key role of extensive IHC analyses for precise subclassification of challenging ovarian tumors. However, the unique combination of sex cord and intestinal differentiation markers yielded a correct diagnosis.

More interestingly, the case also highlights the necessity of communication and detailed multidisciplinary participation among the surgeons, pathologists, and oncologists in the management of such difficult cases. This effective communication was crucial in reporting the intraoperative findings, re-staging where applicable, and consequently also leading to the prescribing of the appropriate adjuvant therapy, which produced a good outcome in the patient. It is an integrated strategy that is essential to provide the optimal care to rare and hard-to-diagnose cancer patients.

4. Conclusion

Sertoli-Leydig cell tumors (SLCTs) are rare ovarian neoplasms that may exhibit a range of clinical and pathological characteristics, and at times involve abundant heterologous components. This case report demonstrates the diagnostic difficulties in these rare presentations, specifically their possible misinterpretation during intraoperative assessment. The optimal management of this case exemplifies the need for an aggressive and comprehensive diagnostic strategy that involves clinical correlation in conjunction with advanced imaging, close working relationships between radiologic pathologists, detailed histopathology review, including immunohistochemical assessment, and molecular testing when appropriate. This also stresses the importance of multidisciplinary team effort for dealing with challenging oncological diseases. High standards in terms of risk-appropriate diagnosis, proper staging, and optimal therapy are essential to improve patient outcomes. Further research of the molecular pathogenesis in SLCTs, especially with unusual characteristics, will help to further delineate diagnostic strategies and treatment options for these rare neoplasms.

Compliance with ethical standards

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Disclosure of conflict of interest

All authors make the following declarations:

- **Payment/services information:** All authors have declared that they received no financial support from any organization for the submitted work.
- **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Statement of informed consent

The patient's consent was obtained for publication of this case report

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

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