

Atypical placental site nodule detected via hysteroscopy – first case report from Brazil

Atypický uzlík v placentární oblasti detekovaný hysteroskopií – první kazuistika z Brazílie

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Summary: Atypical placental site nodule (APSN) is a rare form of gestational trophoblastic disease (GTD) originating from the proliferation of intermediate trophoblasts, with uncertain clinical behavior. It is considered a potential precursor to rare forms of gestational trophoblastic neoplasia (GTN), such as placental site trophoblastic tumor (PSTT) and epithelioid trophoblastic tumor (ETT). This report describes the first Brazilian case of APSN diagnosed via hysteroscopy in a 43-year-old woman following miscarriage. Histopathological analysis revealed a circumscribed, hyalinized aggregate of intermediate trophoblasts with nuclear atypia, positive immunostaining for PLAP and p63, and a Ki-67 index > 5%. The patient declined hysterectomy, opting for conservative management with close monitoring. After 12 months of follow-up, no progression was observed. This case emphasizes the diagnostic challenges of APSN, given its subtle presentation and overlap with benign placental site nodules or even PSTT/ETT. Hysteroscopy proved valuable for both diagnosis and fertility-preserving management. Although hysterectomy remains the definitive treatment in many cases, individualized approaches balancing oncologic safety and reproductive goals are increasingly considered. Long-term clinical vigilance is essential, as APSN may precede aggressive GTN forms. Multicenter studies and registries are urgently needed to establish evidence-based guidelines for the diagnosis, treatment, and follow-up of this rare lesion, improving patient outcomes in these uncommon forms of GTD.

Key words: atypical placental site nodule – gestational trophoblastic disease – gestational trophoblastic neoplasia – placental site trophoblastic tumor – epithelioid trophoblastic tumor

Souhrn: Atypický uzlík v placentární oblasti (APSN – atypical placental site nodule) je vzácná forma gestační trofoblastické choroby (GTD – gestational trophoblastic disease), která vzniká proliferací intermediárních trofoblastů s nejistým klinickým chováním. Je považován za potenciální prekursor vzácných forem gestační trofoblastické neoplazie (GTN – gestational trophoblastic neoplasia), jako je trofoblastický tumor v placentární oblasti (PSTT – placental site trophoblastic tumor) a epitelioidní trofoblastický tumor (ETT – epithelioid trophoblastic tumor). Tato kazuistika popisuje první brazilský případ APSN diagnostikovaný hysteroskopií u 43leté ženy po potratu. Histopatologická analýza odhalila ohraničený, hyalinizovaný agregát intermediárních trofoblastů s jadernou atypií, pozitivním imunobarvením na PLAP a p63 a indexem Ki-67 > 5 %. Pacientka odmítla hysterektomii a zvolila konzervativní léčbu s pečlivým sledováním. Po 12 měsících sledování nebyla pozorována žádná progres. Tento případ zdůrazňuje diagnostické výzvy APSN vzhledem k jeho nenápadnému projevu a překrývání s benigními uzlíky v místě placenty nebo dokonce s PSTT/ETT. Hysteroskopie se ukázala jako cenná jak pro diagnózu, tak pro léčbu zachování fertility. Přestože hysterektomie v mnoha případech zůstává definitivní léčbou, stále častěji se zvažují individualizované přístupy vyvažující onkologickou bezpečnost a reprodukční cíle. Dlouhodobá klinická bdělost je nezbytná, protože APSN může předcházet agresivním formám GTN. Naléhavě jsou zapotřebí multicentrické studie a registry, které by stanovily směrnice založené na důkazech pro diagnostiku, léčbu a sledování této vzácné léze a zlepšily by výsledky pacientů s těmito neobvyklými formami GTD.

Klíčová slova: atypický uzlík v placentární oblasti – gestační trofoblastické onemocnění – gestační trofoblastická neoplazie – trofoblastický tumor v placentární oblasti – epitelioidní trofoblastický tumor

Introduction

Gestational trophoblastic disease (GTD) encompasses a heterogeneous group of lesions derived from villous and intermediate trophoblasts (IT), with varying potential for invasion, malignancy, metastasis, and prognosis [1]. The most common forms of GTD originate from the villous trophoblast and include the hydatidiform mole, which carries a 5–20% risk of progression to gestational trophoblastic neoplasia (GTN) [2,3], as well as the invasive mole and choriocarcinoma, both of which are highly responsive to chemotherapy [4]. In contrast, the IT gives rise to placental site nodules (PSNs) – both typical and atypical (APSNs) [5,6] – as well as placental site trophoblastic tumor (PSTT) and epithelioid trophoblastic tumor (ETT) [7]. The latter two

are chemoresistant and associated with poorer prognoses [8].

While typical PSNs appear to lack potential for progression to GTN, a subset of lesions has been described with intermediate features between a typical PSN and ETT [5]. These lesions are characterized by larger size (> 5 mm), a higher Ki-67 index (ranging from 8% to 15%), and increased cellularity compared to typical PSNs, and were subsequently designated as APSNs [9]. Despite these atypical features, such lesions were initially considered benign in terms of clinical outcome, similar to PSNs. However, in 2015, Kaur et al. [9] reported a series of 21 APSN cases in which 14% (3/21) progressed to GTN (PSTT and/or ETT) during a 16-month follow-up based on hormonal surveillance. Following this

publication, APSN was incorporated into the 2020 World Health Organization classification of tumors of the female genital tract as a precursor lesion to GTN [10].

The biological behavior of APSN remains uncertain, and clinical management is not yet well defined, ranging from expectant monitoring to prophylactic hysterectomy. The aim of this report is to present the first Brazilian case of APSN detected via hysteroscopy, discussing the diagnostic criteria for this entity, its potential progression to PSTT/ETT, and appropriate therapeutic approaches.

Case report

This case report was approved by the Research Ethics Committee of the Maternity School of the Federal University of Rio de Janeiro (CAAE: 90140425.3.0000.5275), and written informed consent was obtained from the patient authorizing its publication.

A 43-year-old Black woman, gravida 1 para 0, with no previous obstetric complications, experienced a spontaneous late miscarriage at 18 weeks of gestation. She had been admitted with abnormal vaginal bleeding and uterine contractions. Prenatal ultrasounds and routine exams had been normal, and she reported no symptoms during pregnancy. Manual removal of the retained placenta followed by uterine curettage was performed under anesthesia without complications.

Histopathological examination of the placenta showed no fetal vasculopathy and no features suggestive of proliferative trophoblastic activity. Prior to pregnancy, the patient had been using combined oral contraceptive pill continuously due to polycystic ovary syndrome and had no personal or family history of other relevant medical conditions.

As she expressed a desire to conceive again, a transvaginal ultrasound was performed one month later. It revealed a discrete focal thickening of the

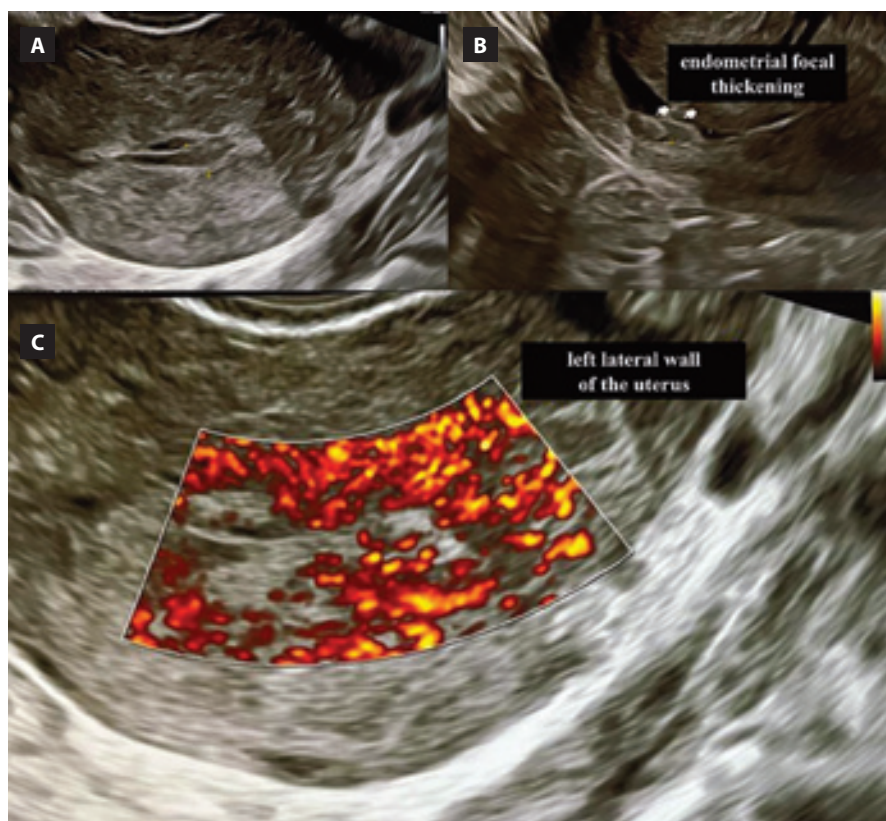


Fig. 1. Pelvic-transvaginal ultrasound, showing focal, heterogeneous endometrial thickening in the left cornual uterine region (A and B), followed by exuberant myometrial vascularization in this topography (C).

Obr. 1. Pánevní transvaginální ultrazvuk, ukazující fokální, heterogenní ztlustění endometria v levé rohové oblasti dělohy (A a B), následované nadměrnou vaskularizací myometria v této topografii (C).

endometrium with increased vascularity on color Doppler mapping (Fig. 1). Pelvic magnetic resonance imaging (MRI) was requested but showed no abnormalities (Fig. 2). Subsequently, hysteroscopy confirmed a focal endometrial thickening in the left cornual region of the uterus, measuring approximately 0.7 cm (Fig. 3).

Histological analysis of an endometrial biopsy obtained during hysteroscopy revealed a circumscribed, hyalinized aggregate of intermediate trophoblasts with central hyalinization and cohesive nests and cords exhibiting cytologic and mild nuclear atypia. Immunohistochemical staining was positive for placental alkaline phosphatase (PLAP) and p63, with a Ki-67 proliferation index > 5%, as shown in Fig. 4. The diagnosis of APSN was confirmed by two independent pathologists.

The patient was referred to the Rio de Janeiro GTD reference center, where her serum human chorionic gonadotropin (hCG) levels were within normal limits. A chest X-ray showed no evidence of metastatic disease, and pelvic MRI revealed a centered, homogeneous endometrium measuring 1 mm in thickness, with no expansile lesions identified in the uterine cavity.

The patient was counseled regarding the uncertain natural history of APSN, including its potential progression to PSTT or ETT. Hysterectomy was proposed as definitive treatment; however, she declined surgical intervention due to her nulliparity and desire to preserve fertility.

Monthly serum hCG levels remained within the normal range over 12 months of follow-up. Control hysteroscopies performed at 6 and 12 months post-diagnosis revealed a homogeneously distributed endometrium with a smooth surface, reddish coloration, normal vascularization, and the presence of glands with a dotted pattern, consistent with a proliferative endometrium. Histological confirmation through biopsy corroborated these findings.

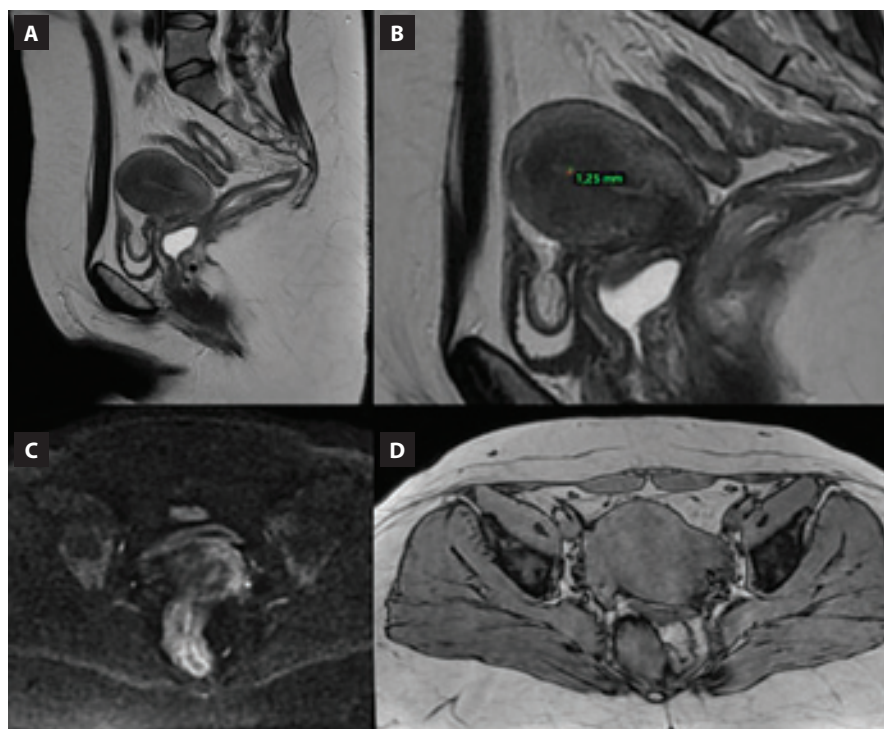


Fig. 2. Pelvic magnetic resonance imaging showed a uterus measuring $79 \times 44 \times 40$ mm, with a homogeneous myometrium, no myometrial nodules, and no signs of adenomyosis (A). The endometrium was centrally located, homogeneous, and measured 1.25 mm in thickness (B). The uterine cavity was preserved, with no expansive lesions or disruption of the junctional zone (C and D).

Obr. 2. Magnetická rezonance pánve ukázala dělohu o rozměrech $79 \times 44 \times 40$ mm s homogenním myometriem, bez myometrických uzlíků a bez známek adenomyózy (A). Endometrium bylo centrálně umístěno, homogenní a měřilo 1,25 mm na tloušťku (B). Dutina děložní byla zachována, bez rozsáhlých lézí nebo narušení junkční zóny (C a D).

As the patient continues to express a desire to conceive, she is currently under follow-up at the preconception care service of our institution.

Discussion

This is the first reported case of an APSN identified by hysteroscopy in Brazil, emerging from a GTD reference center that has managed over 10,000 patients across the full GTD spectrum [11]. In the entire case series from the largest Brazilian referral center, this is the 10th identified APSN, corresponding to a prevalence of less than 1 in 1,000 GTD cases. The rarity of this diagnosis is underscored not only by the scarcity of reports in international literature but also by its

low incidence even in high-volume, specialized centers. This emphasizes the uniqueness of the present case and the relevance of its publication for both the scientific and clinical communities.

Diagnosing APSN remains a considerable challenge due to its subtle clinical presentation and histopathological overlap with other entities within the trophoblastic spectrum. In routine pathology practice, APSNs may be misinterpreted as benign PSNs or underestimated as regressive gestational remnants. However, emerging evidence suggests that APSNs may serve as precursor lesions to malignant entities such as PSTT and ETT, both derived from intermediate trophoblasts [8]. This highlights

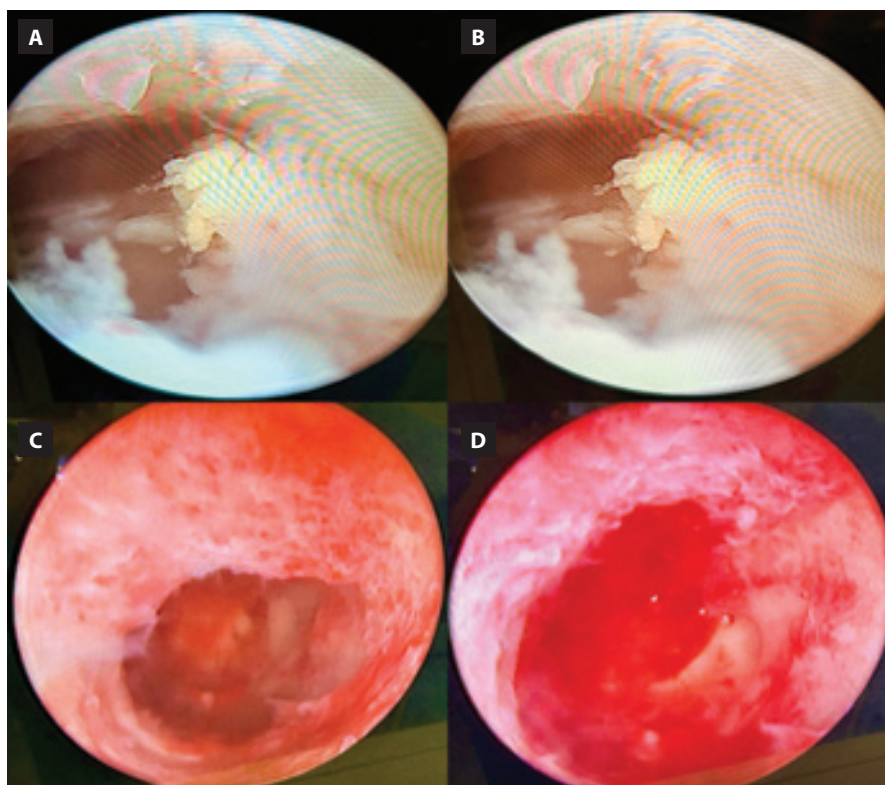


Fig. 3. Hysteroscopy showed a uterine cavity with a smooth endometrial surface, reddish coloration, normal vascularization, and a proliferative appearance (A). A focal thickening was identified near the left cornual uterine region (B), which was removed using forceps, resulting in a homogeneous endometrial cavity (C and D).

Editor's note: The optical effect observed in the image is a consequence of the method of its acquisition and does not affect the interpretation of the displayed information.

Obr. 3. Hysteroskopie ukázala děložní dutinu s hladkým povrchem endometria, načervenalým zbarvením, normální vaskularizací a proliferativním vzhledem (A). V blízkosti levé rohové oblasti dělohy (B) bylo identifikováno ložiskové ztlustění, které bylo odstraněno kleštěmi, čímž vznikla homogenní endometriální dutina (C a D).

Pozn. red.: Optický efekt pozorovaný na snímku je důsledkem metody jeho pořízení a nemá vliv na interpretaci zobrazených informací.

the critical need for heightened awareness among pathologists, particularly those working in reproductive pathology and gynecologic oncology [12–14].

In this context, the use of strict diagnostic criteria and immunohistochemical panels is essential. While PSNs typically exhibit minimal proliferative activity (Ki-67 index < 5%), APSNs show increased mitotic activity, nuclear atypia, and a higher Ki-67 index, often ranging from 10% to 20%. Immunostaining for hPL, CD146 (Mel-CAM), and p63 plays a crucial

role in distinguishing APSN from PSTT, ETT, and benign remnants [9,12–14]. Nevertheless, despite the availability of these tools, diagnostic variability and interpretive uncertainty remain common, complicating clinical management [15,16].

One of the most significant clinical implications of APSN is its potential for malignant progression [9,14,17]. Although data are limited, available reports suggest that APSNs may precede the development of PSTT and/or ETT, both of which are rare, aggressive GTN forms

with variable prognosis and limited response to chemotherapy. Notably, prognosis worsens when the interval between the antecedent pregnancy and GTN diagnosis exceeds four years [7,8]. Therefore, clinical vigilance and long-term monitoring are pivotal following an APSN diagnosis.

Hormonal surveillance, particularly serial serum hCG measurements, remains an essential component of follow-up. While APSNs may not always result in elevated hCG levels, rising titers can indicate progression to GTN [9,15,17]. Unfortunately, many APSN cases present with normal hCG levels. In addition, imaging modalities such as transvaginal ultrasound and chest X-ray may be useful for evaluating local recurrence or progression, especially in patients desiring uterine preservation [18].

Management of APSN remains a subject of debate. Some authors advocate definitive surgical treatment, typically via hysterectomy, especially in cases with atypical features, incomplete resection, or in women who have completed childbearing. This approach ensures diagnostic certainty and eliminates the potential risk of subsequent GTN [9,17]. However, in younger women desiring future fertility, a conservative approach may be considered. This includes complete local excision (preferably via hysteroscopy or, alternatively, curettage), combined with close clinical, hormonal, and radiological follow-up [18,19].

Imaging methods such as transvaginal ultrasound and pelvic MRI may yield normal results in APSN cases, as demonstrated by Lockett et al. [20]. In that cohort, conventional imaging frequently failed to detect abnormalities associated with APSNs. Among all imaging modalities evaluated, hysteroscopy had the highest diagnostic yield, though 44% of cases still showed a normal-appearing endometrial cavity. The remaining cases showed 11% with endometrial polyps, 22% with focal endometrial lesions, 11% with retained nonviable placental tissue,

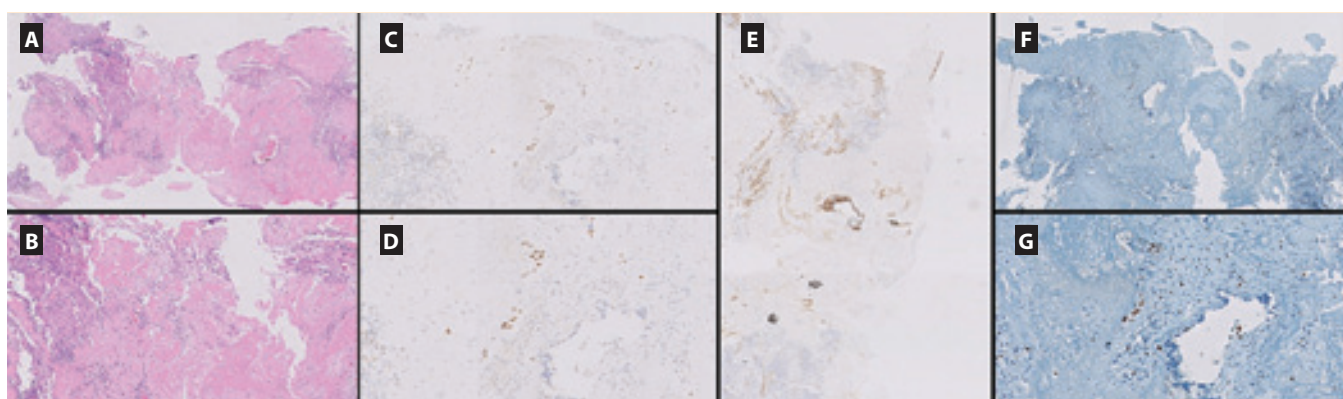


Fig. 4. Histopathological evaluation of the endometrial biopsy specimen.

A, B) Circumscribed, hyalinized aggregate of intermediate trophoblasts with central hyalinization, arranged in cohesive nests and cords exhibiting cytologic and mild nuclear atypia, at 10× and 20× magnification, respectively (hematoxylin-eosin stain). C, D) Immunostaining for p63 showing positive nuclear staining in epithelioid cells, at 10× and 20× magnification, respectively (clone kp10/SP 118, 1 : 400 dilution, Dako® Corporation, Carpinteria, CA, USA. Isotope: IgG; Antigenic reactivation: citrate pH 6.0; Overnight incubation 6 °C). E) Positive immunostaining for placental alkaline phosphatase in epithelioid cells, at 10× magnification. F, G) Ki-67 immunostaining showing a proliferation index > 5%, (clone BC4A4, 1 : 900 dilution, Dako® Corporation, Carpinteria, CA, USA. Isotope: IgG; Antigenic reactivation: citrate pH 6.0; Overnight incubation 6 °C) at 10× and 20× magnification, respectively.

Obr. 4. Histopatologické vyhodnocení vzorku biopsie endometria.

A, B) Ohraničený, hyalinizovaný agregát intermediárních trofoblastů s centrální hyalinizací, uspořádaný v kohezivních hnízdech a provazcích vykazujících cytologickou a mírnou jadernou atypii, při 10× a 20× zvětšení (barvení hematoxylinem-eosinem). C, D) Imunobarvení na p63 vykazující pozitivní jaderné barvení v epitelioidních buňkách, při 10× a 20× zvětšení (klon kp10/SP 118, ředění 1 : 400, Dako® Corporation, Carpinteria, CA, USA. Izotop: IgG; Antigenní reaktivace: citrát pH 6,0; Inkubace přes noc při 6 °C). E) Pozitivní imunobarvení na placentární alkalickou fosfatázu v epitelioidních buňkách, při 10× zvětšení. F, G) Imunobarvení Ki-67 vykazující index proliferace > 5 % (klon BC4A4, ředění 1 : 900, Dako® Corporation, Carpinteria, CA, USA. Izotop: IgG; Antigenní reaktivace: citrát, pH 6,0; Inkubace přes noc při 6 °C) při 10× a 20× zvětšení.

and 11% with endometrial thickening. These findings support the role of hysteroscopy in both diagnostic and therapeutic management.

Hysteroscopy enables direct visualization of the endometrial cavity and precise localization of focal lesions, which is especially useful when APSNs are small, asymptomatic, or incidentally discovered [20,21]. Hysteroscopic resection allows for complete lesion excision while preserving the uterus, making it a favorable option for women of reproductive age. Moreover, hysteroscopy facilitates targeted sampling for histopathological and immunohistochemical analysis – crucial for distinguishing APSNs from PSNs and malignant counterparts such as PSTT and ETT. In selected cases, hysteroscopy may serve as both a diagnostic and fertility-preserving therapeutic tool, provided that careful long-term follow-up is maintained.

Two of the most comprehensive APSN studies come from Charing Cross Hospital

(London) [9] and Brigham and Women's Hospital (Boston) [17]. Both centers consider hysterectomy the definitive treatment. However, at the Boston center, there is a broader inclination toward conservative management in patients with reproductive desires. These differing approaches reflect the current uncertainty surrounding APSN and underscore the importance of individualized care, multidisciplinary decision-making, and referral to specialized GTD centers, where diagnostic expertise and fertility-preserving strategies can be integrated. Prospective data are urgently needed to establish evidence-based management guidelines.

In the present case, the patient's reproductive age and desire for fertility preservation were central to the clinical decision-making process. Conservative management was deemed acceptable, contingent on close monitoring. A limitation of this report is the relatively short follow-up period after remission,

which may affect the timely detection of late recurrence. Nevertheless, the case contributes to the growing body of literature supporting the feasibility of fertility-preserving strategies in selected patients, although definitive recommendations remain premature.

Ultimately, this case highlights the many uncertainties that persist regarding APSN – from diagnostic criteria and classification to optimal treatment and surveillance strategies. Further research is needed to better define its natural history and malignant potential. Multicenter registries and collaborative research initiatives are essential to advance knowledge in this area.

Conclusion

In conclusion, APSN is a rare and diagnostically challenging lesion with significant clinical implications. Although generally considered a benign or borderline entity, its potential association

with malignant GTN warrants thorough evaluation and sustained follow-up. Raising awareness among clinicians and pathologists is crucial for timely diagnosis and appropriate management. In reproductive-aged women, treatment must balance oncologic safety with fertility preservation. As more cases are reported, a clearer clinical pathway is expected to emerge.

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Authors' contributions

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