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CASE REPORT

Primary Epithelioid Trophoblastic Tumor of the Vagina: A Case Report and Literature Review

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Introduction: Primary vaginal epithelioid trophoblastic tumor (ETT) is an exceptionally rare malignant gestational trophoblastic neoplasm.

Case Presentation: A 51-year-old woman presented with irregular vaginal bleeding and elevated serum β-hCG levels. Examination revealed a vaginal wall mass, and local excision confirmed the diagnosis of ETT. The patient completed seven cycles of chemotherapy and was under regular surveillance thereafter. One year later, local recurrence was detected at the primary tumor site. Definitive surgical intervention was then performed, comprising subradical hysterectomy, bilateral salpingo-oophorectomy, and resection of the recurrent vaginal tumor, which was also histopathologically confirmed to be an ETT. Postoperative adjuvant chemotherapy was initiated. The patient has remained disease-free during the subsequent follow-up period.

Conclusion: This case demonstrates that ETT can occur primarily in the vagina, presenting as a vaginal mass with elevated serum β-hCG levels. Therefore, clinicians should include ETT in the differential diagnosis of such cases. Our experience suggests that chemotherapy alone may be insufficient to prevent disease recurrence and that a multimodal treatment strategy centered on complete surgical resection and vigilant surveillance may be effective in achieving long-term remission.

Keywords: ETT, β-hCG, diagnosis, treatment, follow-up

Introduction

Gestational trophoblastic neoplasia (GTN) encompasses various tumors that include choriocarcinoma (CC), placental site trophoblastic tumor (PSTT), and epithelioid trophoblastic tumor (ETT). Among these, ETT is an exceptionally rare subtype originating from chorionic-type intermediate trophoblasts.[1] ETT accounts for approximately 1.39–2.2% of all GTNs and is associated with a 10–24.2% risk of mortality.[2,3] This tumor primarily occurs in the uterine corpus or cervix, and rarely occurs primarily or secondarily in sites such as the lung, vagina, and ovary.[3] ETT is often misdiagnosed, diagnosed late, or poorly managed because of the rarity of the condition, paucity of specific clinical features, and lack of awareness among clinicians. The treatment of ETT in practice currently remains challenging.[4]

In this paper, we elucidate our experience with a case of primary vaginal ETT and review relevant literature, highlighting the importance of comprehensive treatment and regular follow-up and gaining valuable therapeutic insights.

Case Presentation

A 51-year-old woman, gravida 3 para 1, was admitted to our hospital on July 20, 2020, with irregular vaginal bleeding for three years and postcoital bleeding for one month. Before admission to our hospital, she was treated at a local center, where physical examination revealed a 2 cm × 2 cm mass on the right vaginal wall, with no significant palpable uterine or adnexal abnormalities; serum β-hCG was found to be elevated (82.55 IU/L); and ultrasonography showed attenuated

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echoes in the endometrium and endocervical canal, with a suspected myometrial leiomyoma. Surgical excision of the vaginal mass and diagnostic dilation and curettage were performed at that hospital, and the tumor was pathologically confirmed to be ETT. She also had a history of molar pregnancy in 2006, for which she had undergone uterine curettage.

After admission to our institution, the serum β-hCG level was found to be 37.63 IU/L; pelvic ultrasound confirmed a small uterine leiomyoma, and CT scans of the head and chest showed no evidence of metastatic disease. Since no visible lesion remained after the previous local excision, surgery was not indicated.

Considering that ETT remains within the spectrum of GTN despite its relative resistance to treatment, we initially employed the methotrexate (MTX) plus 5-fluorouracil (5-FU) regimen due to its lower systemic toxicity. Subsequently, we evaluated the dynamics of serum β-hCG levels in real time. However, the decline in serum β-hCG levels was unsatisfactory even after two chemotherapy cycles. Therefore, the regimen was switched to two cycles of etoposide plus nedaplatin, following which the serum β-hCG level normalized. Three additional cycles of consolidation chemotherapy were administered (completed on January 23, 2021). The patient was then placed on regular surveillance. On June 26, 2021, serum β-hCG was 5.83 IU/L, although gynecological and ultrasound examination revealed no abnormalities; therefore, no intervention was undertaken.

Between June 2021 and January 2022, the serum β-hCG level fluctuated between 5.83 and 17.93 IU/L, although no significant abnormalities were detected on physical or ultrasound examination. On January 10, 2022, the β-hCG level further increased to 20.35 IU/L. Bimanual examination revealed scarring and nodular consistency at the upper segment of the right vaginal wall, without any visible distinct mass. Transvaginal ultrasound revealed a 10×6 mm ill-defined, slightly hyperechoic area within the vaginal wall, without significant blood flow signals. The recommended hospital admission was declined by the patient for personal reasons. By June 20, 2022, the serum β-hCG had increased to 57.14 IU/L and physical examination revealed a cystic mass (~3 cm) in the mid-upper right vaginal wall, extending superiorly to the cervix. A positron-emission tomography/computed tomography (PET-CT) scan showed a 2.9 cm × 2.0 cm cystic low-density shadow in the right wall of the upper vagina, with increased metabolic activity in the cyst wall and adjacent cervical region, suggestive of tumor recurrence. On June 28, 2022, the patient underwent subradical abdominal hysterectomy, bilateral salpingo-oophorectomy, and excision of the vaginal tumor. During the operation, the uterus and adnexa showed normal appearance with a cyst (~3 cm) in the right vaginal fornix that was superiorly adherent to the cervix, extended inferiorly to the mid-vagina, closely opposed posteriorly to the rectum, and anteriorly adherent to the bladder. The cyst had a smooth inner lining contained dark red fluid (Figure 1). Postoperative pathological examination showed negative margins in the parametria, uterine cornua, and all surgical resection edges. No tumor involvement was noted in the cervix, cervico-uterine junction, ovaries, or fallopian tubes. The uterine corpus showed a leiomyoma and endometrial atrophy. Immunohistochemistry findings were CK (+), CK18 (+), GATA3 (+), P16 (partial +), P40 (partial +), p63 (partial +), HCG (focal +), hPL (−), α-inhibin (−), and Ki-67 (positive rate ≈ 60%). The histological and immunohistochemical findings are presented in Figure 2. A postoperative diagnosis of vaginal ETT was established. On

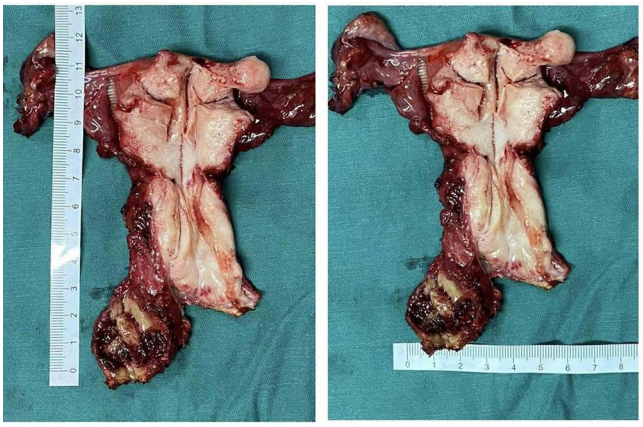

Figure 1 Surgical Specimen: A lesion was observed in the vaginal fornix.

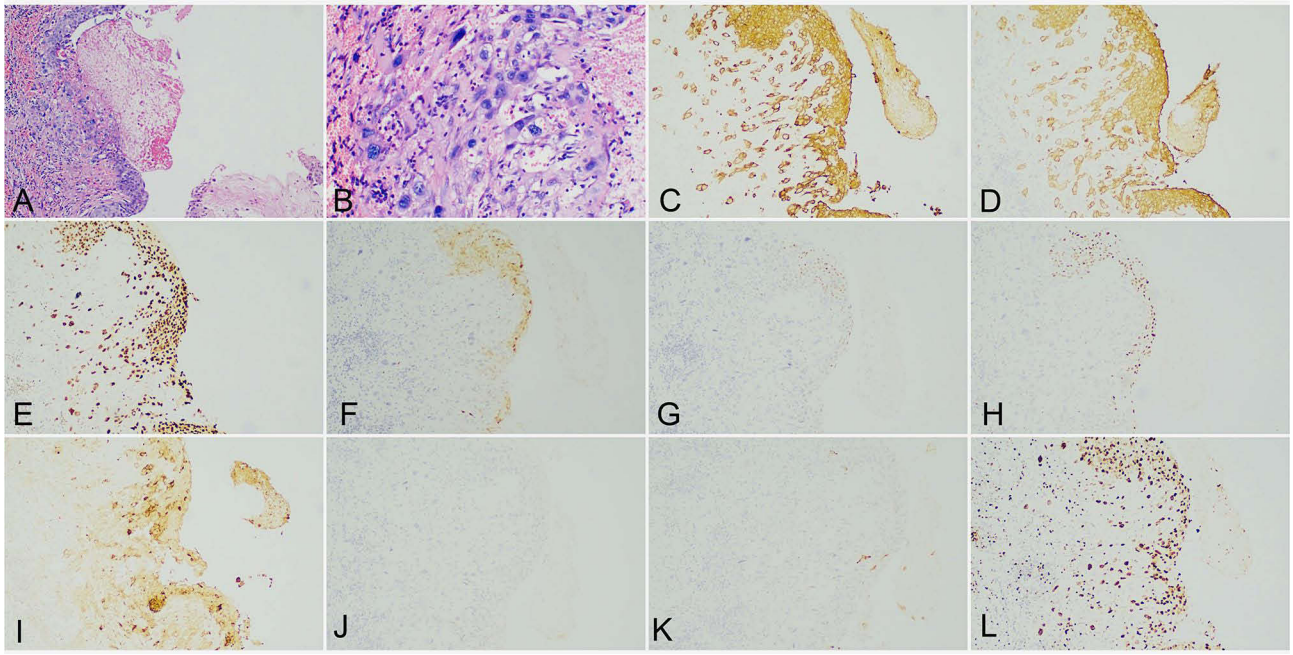

Figure 2 Hematoxylin–eosin (H&E) and immunohistochemical (IHC) staining of epithelioid trophoblastic tumor (ETT): (**A**) Tumor cells arranged in nests surrounded by extensive coagulative necrosis (H&E, ×100). (**B**) Tumor cells exhibiting mild to moderate nuclear atypia (H&E, ×200). Immunohistochemical staining showing positive expression for: (**C**) CK, (**D**) CK18, (**E**) GATA3, (**F**) p16, (**G**) p40, (**H**) p63, (**I**) hCG, (**J**) hPL, (**K**) α-inhibin, and (**L**) Ki-67 (all ×100).

the seventh postoperative day, the patient's serum β-hCG level had normalized. Subsequently, three cycles of consolidation chemotherapy comprising etoposide plus cisplatin were administered. The changes in the serum β-hCG levels are shown in Figure 3.

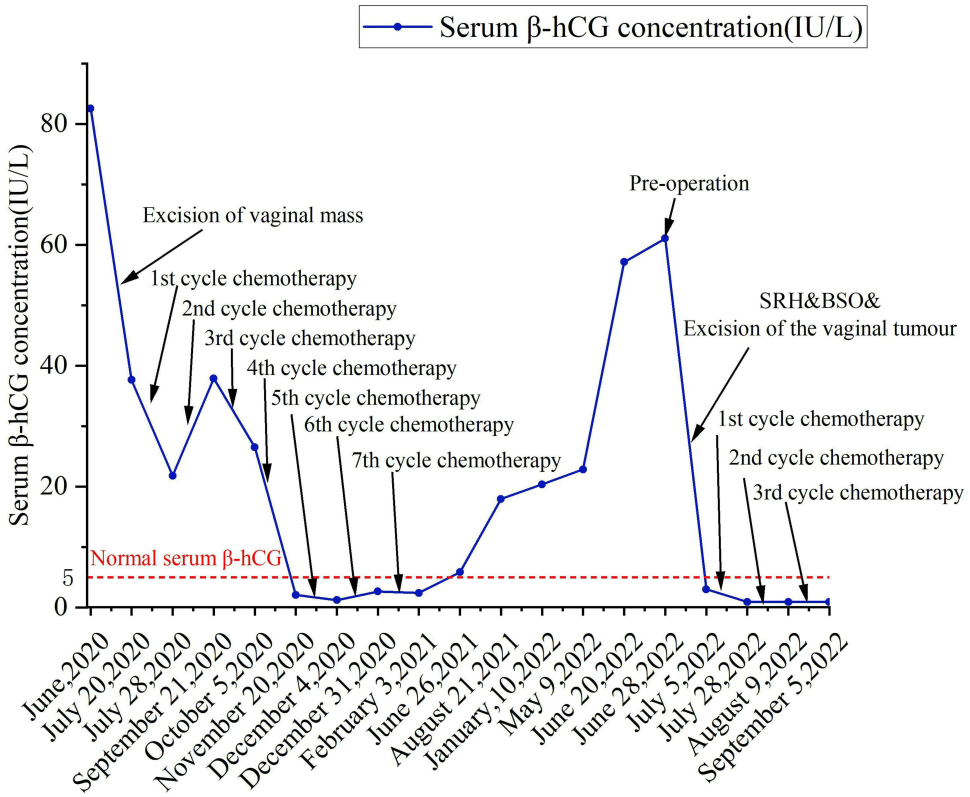

Figure 3 Evolution of serum β-hCG levels over time.

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The patient was regularly followed up with serial measurements of serum β-hCG levels and serial imaging studies. During the surveillance, the patient has remained asymptomatic, with normal serum β-hCG levels and no radiological evidence of recurrence.

Discussion

ETT is an extremely rare subtype of GTN.[3] While its histopathological features are well defined, the pathogenic mechanisms remain unclear.[5] The abnormal presence of Y chromosome material and paternal alleles within ETT lesions is suggestive of its gestational origin.[6] ETT is often associated with term pregnancy, hydatidiform mole, or spontaneous abortion,[3] predominantly affecting reproductive-aged women (age: 15 to 66 years)[5] and, occasionally, postmenopausal women.[7] Our patient was a 51-year-old woman who had undergone suction curettage for molar pregnancy—a clinical scenario commonly associated with ETT.

Clinical manifestations of ETT are diverse and nonspecific and commonly include irregular vaginal bleeding with amenorrhea, hemoptysis, or abdominal pain.[3] Some patients remain asymptomatic.[8] The initial symptoms may sometimes be related to extrauterine or metastatic disease, with the lung being the most frequent metastatic site (~19% of cases),[7] followed by small intestine, vagina, fallopian tube, ovary, broad ligament, and gallbladder.[3] Approximately 80% of patients exhibit mild-to-moderate elevation of serum β-hCG (typically: <2500 IU/L),[3–5] although rarely, the levels may remain undetectable.[7] While serum β-hCG level has shown no clear correlation with disease progression or prognosis,[9] it is a reliable marker for diagnosing and monitoring extrauterine ETT.[10] Our patient's symptoms of irregular vaginal bleeding and mildly elevated serum β-hCG (82.55 IU/L) are common, but the primary vaginal location of the ETT, with no evidence of uterine involvement at diagnosis or during follow-up, is highly unusual. To date, only three such cases have been documented (Table 1). In one such case, the patient presented with vaginal soft tissue obstruction, which was diagnosed as vaginal ETT following local excision.[11] In the remaining two cases, the condition was initially misdiagnosed as vaginal squamous cell carcinoma.[12,13]

Thus, the preoperative diagnosis of ETT remains challenging. Conventional imaging modalities offer limited diagnostic value. Ultrasonography serves as an initial screening tool, demonstrating well-circumscribed solid masses with peripheral hypoechoic halos and peripheral vascularity. CT and magnetic resonance imaging are mainly useful for staging and detecting complications.[3,14] PET/CT allows early metabolic identification of active lesions and facilitates outcome assessment and recurrence monitoring. PET/CT also helps improve survival outcomes by guiding curative surgery through detection of solitary metabolically active lesions.[15,16] In our case, PET/CT clearly revealed a cystic lesion in the upper right vagina with increased FDG uptake in the wall and adjacent cervical stroma, providing the first indication of recurrence and enabling timely intervention. Despite being costly, PET/CT is a valuable supplementary tool for ETT surveillance, although further validation in larger cohorts is warranted.

ETT is frequently misdiagnosed as PSTT or CC due to its nonspecific and overlapping features with these conditions. Furthermore, given its predilection for the lower uterine segment and cervix, it can morphologically resemble cervical squamous cell carcinoma (CSCC), resulting in misdiagnosis.[2,17] Therefore, postoperative histopathology with immunohistochemistry remains the gold standard for diagnosing ETT.[10] Macroscopically, ETT is characterized by expansile nodules or cystic hemorrhagic masses with central necrosis and hemorrhage, frequently accompanied by ulceration and fistula formation. Microscopically, the tumor shows nests and cords of monomorphic epithelioid cells with mild-to-moderate nuclear atypia and frequent mitotic figures, with extensive ("geographic") necrosis and eosinophilic hyalinized material.[18] These features are similar to those of CSCC, particularly if biopsy specimens are too small to allow detection of typical hyalinization and geographic necrosis.[17] ETT may be differentiated from PSTT on basis of the latter's diffuse and highly infiltrative growth pattern.[7] Immunohistochemically, ETT demonstrates positive staining for inhibin-α, cytokeratins (CK18, CKAE1/AE3), epithelial membrane antigen (EMA), HLA-G, p63, E-cadherin, prolyl-4-hydroxylase, and EGFR. Additionally, ETTs show focal positivity for hCG, hPL, PLAP, and Mel-CAM, which are typically absent in CSCC. The Ki-67 proliferation index is typically 10–25% in ETT, but usually >50% in CSCC. However, extrauterine ETT with Ki-67 expression exceeding 50% has been associated with significantly increased recurrence risk, although low Ki-67 expression neither indicates favorable outcomes nor precludes disease-related mortality.[10,19] Positive staining for

Table 1 Clinical Features of Patients with Isolated Vaginal ETT in the Literature

Case	Age(y)	AP/ Interval	Symptoms	hCG (IU/L)	Size (cm)	Initial Treatment	Chemotherapy Regimens	Recurrence	Subsequent Treatment	Outcome	Follow-Up
Zhao et al[11]	43	Abortion/ 2y	soft tissue blocking vagina	/	Unknown	Cystectomy	VCR+FUDR+Act-D+VP-16	+	Vaginal lesion resectionandCurettage	NED	8 mo
Wang et al[12]	41	HM/ Unknowm	AUB	1038. 8	9×6×2	TAH & Vaginal tumor enucleation	Unknown	+	ND	Death	14 mo
Li et al[13]	50	FTD/ Unknown	a 3-year history of vaginal pain	<2	Unknown	TAH & BSO & Partial vaginectomy	EMA/CO	-	/	NED	12 mo
Present study	51	HM/14y	AUB	82.55	2 ×2	Excision of vaginal mass and Curettage	MTX+5-FU& (etoposide + nedaplatin)	+	SRH & BSO & Excision of the vaginal tumor and EP	NED	36 mo

Abbreviations: -, none; &, and; +, yes; Act-D, actinomycin-D; AP, antecedent pregnancy; AUB, abnormal uterine bleeding; BSO, bilateral salpingo-oophorectomy; EMA/CO, etoposide; methotrexate; actinomycin-D/ cyclophosphamide; Vincristine; EP, etoposide, cisplatin; FUDR, floxuridine; FTD, full-term delivery; HM, hydatidiform mole; mo, month; MTX, methotrexate; ND, not described; NED, no evidence of disease; SRH, sub-radical hysterectomy; TAH, total abdominal hysterectomy; VCR, vincristine; VP-16, etoposide; y, year;5-FU, 5-fluorouracil.

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p63 and its truncated isoform p40 favors ETT over PSTT.[3] The immunohistochemical profile in our case was consistent with characteristic ETT features.

In contrast to choriocarcinoma, which is often curable with chemotherapy alone, ETT is relatively chemoresistant, making surgical resection (including hysterectomy and excision of extrauterine lesions) the cornerstone of management.[20] Early-stage disease confined to the uterus can be effectively treated by total hysterectomy alone, without the need for adjuvant chemotherapy. Thus, for patients with non-metastatic, low-risk, Stage I disease with uterine-only involvement, hysterectomy alone is the first-line treatment.[2,4,21] For patients with isolated extrauterine lesions, surgical resection affords favorable outcomes. Multiple conservative surgeries may be required for tumor control if fertility preservation is required, with hysterectomy being reserved for recurrence or specific circumstances.[10,20,22] Given the low incidence of ovarian metastasis, oophorectomy has not shown prognostic benefit and is not routinely recommended.[2] Advanced or metastatic disease requires a multimodal strategy combining surgery and chemotherapy.[2,3,20] Our patient initially underwent local excision of the vaginal mass. Upon recurrence, given the suboptimal efficacy of the previous treatment regimen, the anatomical adherence of the recurrent lesion to the cervix, and the need for a hysterectomy to ensure negative surgical margins, as well as the patient's perimenopausal status and lack of fertility concerns, we performed definitive surgery after a thorough discussion with the patient and her family. In accordance with the principle of individualized treatment, this procedure included a sub-radical hysterectomy, bilateral salpingo-oophorectomy, and excision of the vaginal tumor. Adjuvant chemotherapy is crucial to ETT management. Although no standardized chemotherapeutic regimen has been established, multi-agent protocols are commonly employed. Current recommendations include platinum-based regimens such as EMA/EP, cisplatin/paclitaxel, and etoposide/platinum combinations for improved survival, particularly in cases with persistent postoperative β-hCG elevation, metastatic disease unsuitable for surgical resection, or high-risk features.[2,8,23] Timely modification of strategy is imperative in chemoresistant disease. Repeat surgery or targeted biological therapy may offer viable options for improving outcomes.[4,7] Immunomarkers such as EGFR, PD-L1, PD-L2, B7-H3, VISTA, and CD105 in ETT tissues have shown potential therapeutic relevance in studies on metastatic refractory GTN in which disease stability was achieved with TRC105 plus bevacizumab.[4,10,24,25]

As a rare disease, ETT does not yet have well-established prognostic criteria. An interval of >48 months between the antecedent pregnancy and ETT diagnosis, maternal age of >40 years, mitotic counts of >5/10 HPFs, advanced FIGO stage, presence of necrosis and deep invasion, and extrauterine disease spread have been identified as high-risk factors.[5,7,17] Systemic therapy is recommended if any of these high-risk factors are present. Since our patient met several of these criteria, adjuvant chemotherapy was initiated postoperatively, followed by regular surveillance with serial β-hCG measurements.

We suggest that ETT should be included in the differential diagnosis of patients presenting with unexplained vaginal bleeding with or without elevated β-hCG levels, particularly when accompanied by a history of gestational trophoblastic disease. Furthermore, patients with extrauterine ETT who present with incompletely resected primary tumor or post-operative re-elevation of serum β-hCG levels should undergo PET/CT when conventional imaging is inconclusive. This would allow for earlier detection of residual or recurrent disease, thereby potentially preventing systemic dissemination and disease progression and enable timely intervention with secondary surgery or salvage chemotherapy.

Conclusions

To conclude, the successful management of this rare case of primary vaginal ETT highlights the importance of diagnostic awareness, personalized multimodal therapy, and vigilant follow-up incorporating advanced imaging technologies. Our experience provides valuable insights for establishing standardized diagnostic and therapeutic approaches for ETT.

Abbreviations

β-hCG, β-subunit of human chorionic gonadotrophin; CC, choriocarcinoma; CSCC, cervical squamous cell carcinoma; EMA, epithelial membrane antigen; ETT, Epithelioid trophoblastic tumor; GTN, gestational trophoblastic neoplasia; MTX, methotrexate; PET-CT, positron-emission tomography/computed tomography; PSTT, placental site trophoblastic tumor.

Data Sharing Statement

The datasets generated and analyzed during the present study are available from the corresponding author on reasonable request.

Ethics Approval and Informed Consent

Ethical approval for this case report was obtained from the Ethics Committee of the Affiliated Hospital of North Sichuan Medical College and has received the patient's written informed consent.

Consent for Publication

Written informed consent has been provided by the patient to have the case details and any accompanying images published.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that they have no conflicts of interest in this work.

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