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MR APPEARANCE OF PLACENTAL SITE GESTATIONAL TROPHOBLASTIC NEOPLASM

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The magnetic resonance imaging (MRI) findings of a patient with placental site trophoblastic tumor are presented. In this patient, a small focal mass distorting the junctional zone and invading the myometrium was identified. Due to the aggressive nature of this neoplasm and the difficulty in detecting it clinically, MRI may represent a useful tool in the evaluation of such tumors.

KEY WORDS:
Placental site trophoblastic tumors; Gestational trophoblastic disease; Magnetic resonance imaging

INTRODUCTION

Gestation trophoblastic disease (GTD) represents a spectrum of tumors ranging from the benign hydatidiform mole to the invasive mole to the highly malignant choriocarcinoma. These neoplasms typically contain syncytiotrophoblastic cells that produce the serum beta subunit of human chorionic gonadotropin (B-HCG), which serves as a marker of tumor presence or recurrence when serum levels are elevated (1, 2). Furthermore, once detected, these tumors have an excellent response to methotrexate chemotherapy (3).

Elston (4) proposed a classification of GTD tumors that included a fourth neoplasm named "trophoblastic pseudotumor." Initially thought to be a benign process representing an exaggerated form of placental site reaction (4), the tumor as reevaluated by Scully and Young (5) suggested a more malignant behavior and was concomitantly renamed the "placental site trophoblastic tumor (PSTT)."

Despite the exaggerated proliferation of trophoblastic cells present in the GTD tumor, the PSTT contains few syncytiotrophoblastic cells that secrete B-HCG (6). As a result, clinical detection by chemical monitoring is more difficult. Furthermore, this tumor is not responsive to chemotherapy, but rather requires surgery for cure (7, 8). Therefore, early detection is all the more critical. In the patient reported here, we were able to detect the presence of a PSTT using magnetic resonance imaging (MRI). This facilitated early surgical intervention and possibly prevented the development of metastatic disease.

CASE REPORT

A 33-year-old woman presented to her obstetrician with a chief complaint of "feeling pregnant" despite being on oral contraceptives following the delivery of a healthy male child 9 months earlier. She had taken a home pregnancy test that demonstrated positivity. The serum B-HCG level was 28 IU. The normal values are less than 5 IU. Following an episode of excessive uterine bleeding, a dilation and curettage was performed. Histopathological evaluation of the tissue revealed elements suggestive of placental insertion site trophoblastic tumor.

A chest X-ray and computed tomography (CT) scan showed no evidence of extrauterine disease. Serial serum B-HCG levels were measured and demonstrated slow but progressive elevation over time (21 IU on February 19, 54 on February 24, 60 on February 26, and 82 IU on March 5). On March 4, laparoscopy and hysteroscopy with dilation and curettage were performed. Although a mass could not be identified by directed visualization on hysteroscopy, dilation and curettage showed decidual fragments of an implantation site.

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Following the dilation and curettage, the B-HCG level was 45 IU. Despite the decrease in the serum B-HCG level, the possibility that residual tumor in the form of a PSTT remained. In an attempt to identify any residual GTD following the dilation and curettage, magnetic resonance imaging (MRI) was performed. On T2-weighted fast spin-echo images (TR 4000/TE 102EF; on a Sigma 1.5-T General Electric scanner) a 1.0- to 1.5-cm mass was identified bulging into the endometrial cavity in the anterior left portion of the uterine fundus (Figure 1). The small mass distorted the junctional zone at this site and was focally invading the myometrium. The MRI findings were compatible with a residual trophoblastic tumor with superficial invasion of the tumor into the myometrium. Despite the decreased serum B-HCG level, the findings on MRI were of enough concern that the patient was given the option of hysterectomy.

At pathology, the uterus measured 9.5 × 7.0 × 4.0 cm. Bivalving of the uterus displayed in the left cornual region an indurated area which was 1.2 × 1.2 cm. This was found in the anterior uterine wall. On serial cross section of this anterior uterine wall, a well-circumscribed tan-brown lesion was found. This lesion measured 1.2 cm in diameter and extended to 2.0 cm from the lateral margin of the uterine serosa. It did not grossly extend to the uterine serosa and its anterior surface. The entire area was serial sectioned. The sections demonstrated an intramural tumor. This tumor demonstrated a proliferation of trophoblastic tissue consisting primarily of cytotrophoblastic cells. Few syncytiotrophoblastic cells were present. There were 12 mitotic figures per high-power field, findings compatible with invasive PSTT. The follow-up B-HCG level was less than 2 IU and the patient remained asymptomatic.

DISCUSSION

PSTT was initially described by Kurman et al. in 1976 (9). Their series consisted of 12 patients with a distinct exaggerated placental site reaction. The term "trophoblastic pseudotumor" was suggested to reflect the apparently benign nature of this tumor. However, as clinical experience with this tumor grew, a metastatic potential was demonstrated. In 1981, Scully and Young (5) published a reappraisal of these rare tumors, which in fact often demonstrated aggressive behavior, and they coined the current term "placental site trophoblastic tumor."

A review by Elston (4) includes PSTT in the spectrum of GTD. Similar to GTD, these tumors typically occur in women of reproductive age and usually follow any type of gestation. Also, both PSTT and GTD consist of a proliferation of trophoblastic tissue and can represent a spectrum from local disease to metastatic disease (10).

However, there are significant dissimilarities. Whereas GTD contains both cytotrophoblastic tissue and the B-HCG–producing syncytiotrophoblastic cells, PSTT characteristically contains few or no syncytiotrophoblastic cells. As a result, these tumors often do not have the elevated B-HCG levels that allow for chemical monitoring of GTD. Furthermore, even in the presence of metastatic disease, GTD has an excellent response to chemotherapy. There have been no long-term survivors with metastatic PSTT.

Patients with PSTT typically present complaining of menstrual disturbances. A history of some form of previous gestation is the norm, often within the past 12 months. In 5% of patients, a spontaneous abortion or hydatidiform mole precedes the development of PSTT. Some unusual presentations include amenorrhea, virilization, and nephrotic syndrome. B-HCG levels are normal or only mildly elevated in 69% of patients, with a moderate elevation demonstrated in only 31%. Histologically, PSTT is characterized by mononuclear and occasionally multinuclear trophoblastic cells that infiltrate the uterus and its blood vessels.

The radiological evaluation of GTD has been well described. The mainstay of diagnosis is ultrasound (11). The original descriptions of a characteristic snowstorm appearance and speckling on ultrasound were made by MacVicar and McDonald in 1963 utilizing transabdominal ultrasound (12). The sonographic evaluation has been enhanced by the development of transvaginal ultrasound, which enables increased resolution. Ultrasound is an effective imaging modality for GTD in that it is noninvasive and widely available.

CT examination of patients with GTD is reserved for evaluation of metastatic disease. CT has only limited value as a modality in the evaluation of intrauterine pathology. Further, it exposes the patient to ionizing radiation. Additionally, although angiography has been used to evaluate the increased vascularity of GTD, it too is invasive and is no longer in clinical use for the evaluation of these tumors (13, 14).

MRI has proved to be an excellent tool in the evaluation of intrauterine pathology. The uterine anatomy has been well described and MRI allows the evaluation of the layers of the uterus (i.e., endometrium, junctional zone, myometrium, and serosa). The adnexa, which is not always well seen on ultrasound, can also be well seen on MRIs (13–16).

The MRI appearance of GTD of the uterus was described by Hricak et al. (14) and Barton et al. (16). The neoplasms are typically seen to distort the uterine zonal structures and demonstrate hypervascular masses

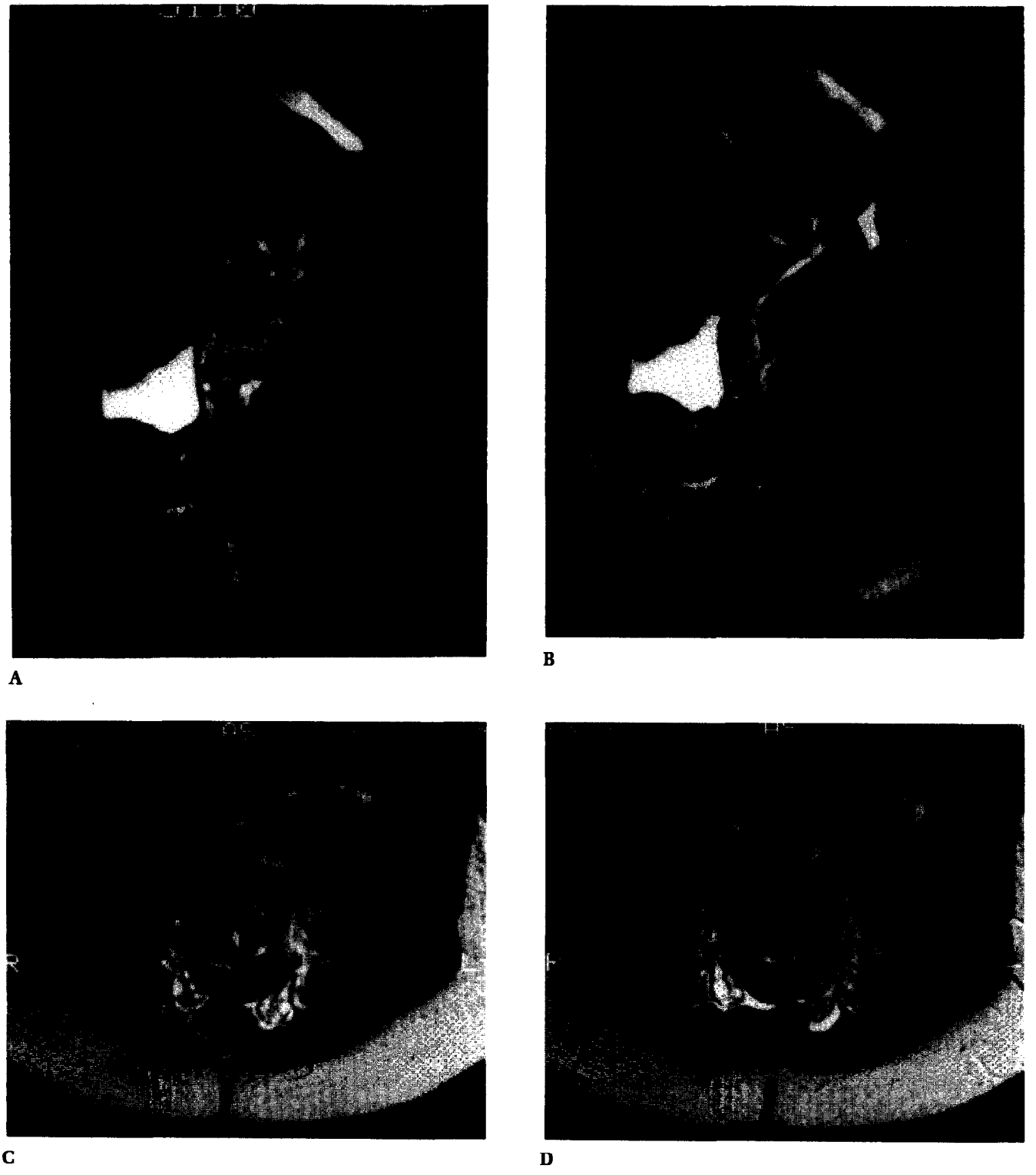

Figure 1. MRI of placental site trophoblastic tumor. **(A** and **B)** Sagittal T2-weighted fast spin-echo images through the uterus (TR 4000/TE 102EF/ETL 8; 256 × 256, 2 NEX, fat saturation) demonstrate a small 1.0- to 1.5-cm mass bulging into the upper endometrial cavity *(arrows)* **(C** and **D)** Axial T2-weighted fast spin-echo images also show the mass and demonstrate distortion of the junctional zone with invasion of the underlying myometrium *(arrows)*.

of heterogeneous signal intensity. Further, changes in the MRI appearance of these tumors such as regression of vascular abnormalities, development of intralesional hemorrhage, and return of the normal zonal anatomy correlate with decreasing B-HCG levels (14). Our review of the literature demonstrated no previous discussions of the MRI appearances of PSTT.

MRI played a significant role in the management of this patient with PSTT. Although the initial dilation and curettage produced tissue fragments suggestive of PSTT, a follow-up hysteroscopy failed to visualize a tumor mass and B-HCG levels were actually returning to normal. The MRI appearance, however, was so suggestive of an invasive tumor mass that given the aggressive nature of PSTT, it was deemed prudent to perform a hysterectomy. Pathological investigation of the surgical specimen supported the initial diagnosis of PSTT. MRI facilitated the prompt removal of the tumor and prevented a delay in treatment, which could have allowed for metastatic disease to develop.

In summary, PSTT is a variant of GTD that typically fails to secrete the characteristically high B-HCG levels and does not respond well to therapy. Hysteroscopy and/or dilation and curettage can result in false-negatives if the tumor is subendometrial. We present a case of PSTT in which direct visualization by hysteroscopy failed to identify the tumor, whereas MRI succeeded in demonstrating a focal mass. MRI demonstration of tumor in this patient resulted in appropriate early intervention. Given the otherwise silent growth and malignant potential of PSTT, such early intervention is crucial and MRI can play a significant role in management.

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