

Case Report

Computed Tomography and Angiographic Findings in Metastatic Choriocarcinoma

Arthur S. Watanabe and Wendy R. K. Smoker

Abstract: We report a patient with metastatic gestational choriocarcinoma who presented with headaches, speech difficulties, motor aphasia, facial weakness, hemiparesis, and a large intraparenchymal hematoma. She subsequently developed a total of five intraparenchymal hemorrhages. Her clinical presentation posed a diagnostic dilemma and emphasizes the importance of a complete differential diagnosis of multiple intracranial hemorrhages. The features of CNS choriocarcinoma are reviewed. **Index Terms:** Brain, neoplasms—Brain, hemorrhage—Computed tomography.

Gestational choriocarcinoma in the United States is an uncommon malignancy of conception occurring in ~1/50,000 term pregnancies and 1/30 hydatidiform moles (1). Among all cases of gestation related choriocarcinoma, ~45% occur after a molar pregnancy, 24% after a normal term pregnancy, 25% after a spontaneous abortion, and 5% after an ectopic pregnancy (2). Gestational choriocarcinoma that follows hydatidiform mole usually is readily diagnosed on the basis of rising or plateauing human chorionic gonadotropin (β -HCG) titers following evacuation of the uterus if the disease is suspected. Gestational choriocarcinoma, however, may present in subtle ways and be more difficult to diagnose, especially if unsuspected. We report a patient with metastatic gestational choriocarcinoma. The case presented a diagnostic dilemma and emphasizes the importance of considering all differential possibilities in patients who present with intracranial hemorrhage.

CASE REPORT

A 24-year-old, gravida 3 para 2, woman developed Rh isoimmunization at 19 weeks' gestation, eventually ne-

cessitating three intrauterine transfusions and a cesarean delivery at 35 weeks' gestation. A 1,250 g placenta delivered spontaneously and exhibited changes of erythroblastosis fetalis.

Fourteen weeks post partum, the mother began to develop headaches and awoke one morning with difficulty in speaking. She presented to a local hospital with transcortical motor aphasia, right central facial weakness, and evolving hemiparesis. Unenhanced CT of the head demonstrated a large left frontal intraparenchymal hematoma (Fig. 1) and she was admitted with a presumptive diagnosis of an arteriovenous malformation.

Four vessel cerebral angiography was performed 10 days following the onset of symptoms. The left carotid injection revealed avascular mass effect due to the hematoma. In addition, multiple areas of segmental vascular dilatation and narrowing, intraarterial thrombosis, and contrast extravasation were identified and believed to be compatible with a necrotizing vasculitis (Fig. 2). The right carotid injection demonstrated irregularity of a parietooccipital branch of the middle cerebral artery with a possible aneurysm but no thrombosis or contrast extravasation (Fig. 3). The posterior circulation was normal. Suspected underlying etiologies at that time included subacute bacterial endocarditis, polyarteritis nodosa, systemic lupus erythematosus, and a granulomatous vasculitis. Serum analysis demonstrated an antinuclear antibody titer of 1:1,280, rheumatoid factor of >60 IU, and Farr assay of 7%. Repeat CT 12 days after the initial scans and 2 days after angiography demonstrated a new right occipital hemorrhage in the distribution of the abnormal right middle cerebral branch identified angiographically, as well as enlargement of the left frontoparietal hemorrhage (Fig. 4).

From the Department of Radiology, University of Utah, 50 North Medical Drive, Salt Lake City, UT 84132, U.S.A. Address correspondence and reprint requests to Dr. W. R. K. Smoker.

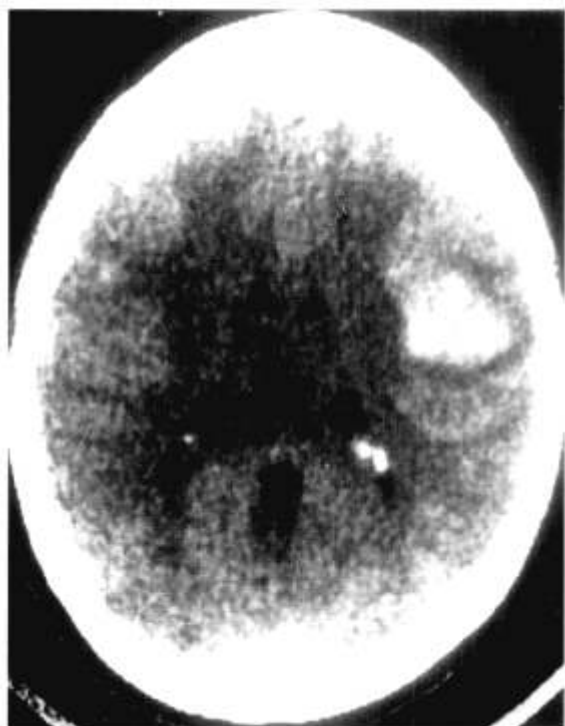


FIG. 1. Noncontrast CT scan of the head on the day of the initial hemorrhage demonstrates a large left frontal intraparenchymal hematoma. There is some mass effect and surrounding edema.

Serial CT scans ultimately demonstrated a total of five separate supratentorial intraparenchymal hemorrhages.

Her platelet count dropped to a low of 5,000/mm³ from an admission value of 441,000/mm³. Idiopathic thrombocytopenic purpura, unresponsive to steroids, was suspected and presumed to be an autoimmune phenomenon related to her phenytoin therapy. She underwent emergency splenectomy.

Gross examination of the spleen revealed a single 1.0 × 1.0 × 0.5 cm gray-white parenchymal nodule. Microscopic evaluation of this nodule was reported to demonstrate choriocarcinoma. Evaluation for additional metastases, including lungs, liver, kidneys, bladder, pelvis, gastrointestinal tract, and cervix, was negative.

Therapy began immediately with whole brain irradiation and simultaneous methotrexate, actinomycin D, and cyclophosphamide chemotherapy. Her pretreatment β-HCG titer was 52,728 mIU/ml. Forty-one days later, after the third course of chemotherapy, her β-HCG titer was negative. She received three additional full courses of chemotherapy after the negative β-HCG titer. More than 1 year later, her β-HCG titers have remained negative and she has made a complete neurologic recovery.

DISCUSSION

Trophoblastic disease is thought to be due to uncontrolled invasion of maternal uterine tissue and

maternal blood sinuses by trophoblastic tissues. The release of trophoblastic cells into the maternal venous circulation is thought by some to be a normal event. Accordingly, metastatic trophoblastic disease and choriocarcinoma are disseminated hematogenously with the most common sites of tumor origin and involvement being the uterus and lung.

Intracranial metastases adversely affect the prognosis of the disease and are the leading cause of death in patients who die of choriocarcinoma. The incidence of CNS metastases has been reported to range between 3 and 28% (1). Clinically the presentation is nonspecific and patients may demonstrate headache, nausea, vomiting, hemiparesis, seizures, or neurological deficits. A small percentage of patients demonstrate no symptoms. Half of those who die from intracranial metastases present with CNS disease and the other half are reported to develop CNS metastases after treatment. About 33–76% of patients with brain metastases have multiple lesions.

It has been reported that the location of intracranial metastases corresponds to the proportion of cerebral blood flow supplying each part of the brain with the majority of metastatic lesions occurring in the parietal lobe followed by the occipital, frontal, and temporal lobes. The brain stem and cerebellum are least frequently involved. A tendency for right hemispheric involvement has been reported.

These metastases are noted for their propensity to produce hemorrhage and they present in 50% of the cases as intracerebral hemorrhages due to the tendency of the trophoblast cells to penetrate vascular walls (3). Intracranial metastases may be manifested as aneurysms, usually involving the middle cerebral artery or its branches. These aneurysms are usually solitary although occasionally they may be multiple.

About 20% of patients with lung metastases also have CNS lesions. Many studies have reported that 95–99% of patients with brain metastases also have pulmonary metastases since the source of the brain lesions is thought to be the lungs. The average number of metastatic sites in patients with intracranial involvement is four, with liver, kidney, gastrointestinal tract, spleen, and peritoneum frequently involved sites. Intracranial choriocarcinoma has been reported in one patient who had a possible genital source of metastatic disease, without evidence of trophoblastic disease removed prior to the clinical evidence of the metastatic process (3). Another patient has been reported whose original tumor was thought to have disappeared and who subsequently died of metastatic disease (3).

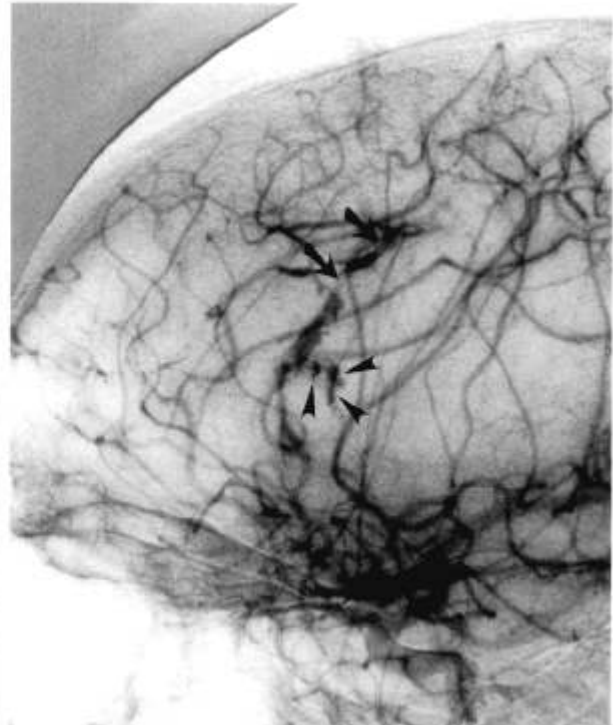
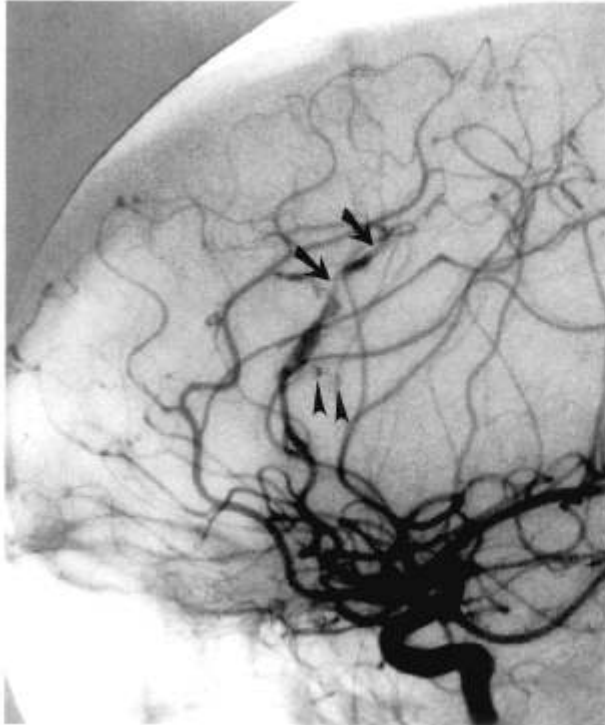
Primary CNS choriocarcinoma, although rare, has also been reported (4). It occurs predominantly in the pineal region or anterior third ventricular re-

gion in a younger population (mean age 12 years). Males are more commonly affected, usually presenting with symptoms of precocious puberty (4,5).

Although metastatic CNS choriocarcinoma is

well recognized in the obstetrical literature, it is poorly reported in the radiological literature and not well emphasized in standard major cranial CT textbooks (6,7). This case illustrates the importance of

2a, b



2c

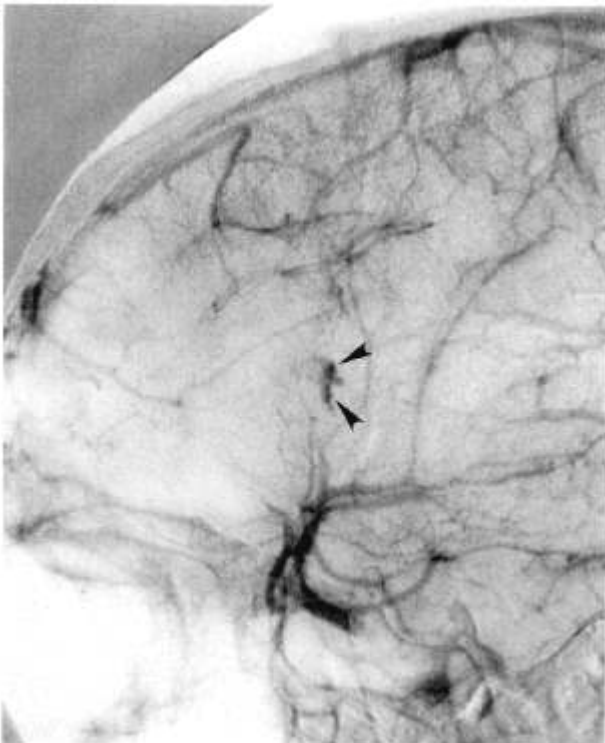


FIG. 2. a-c: Lateral views of the left carotid injection at 2, 3, and 6 s, respectively, reveal avascular mass effect due to the hematoma with depression of the Sylvian triangle, multiple areas of segmental vascular dilatation and narrowing, intraarterial thrombosis (arrows), and contrast extravasation (arrowheads).

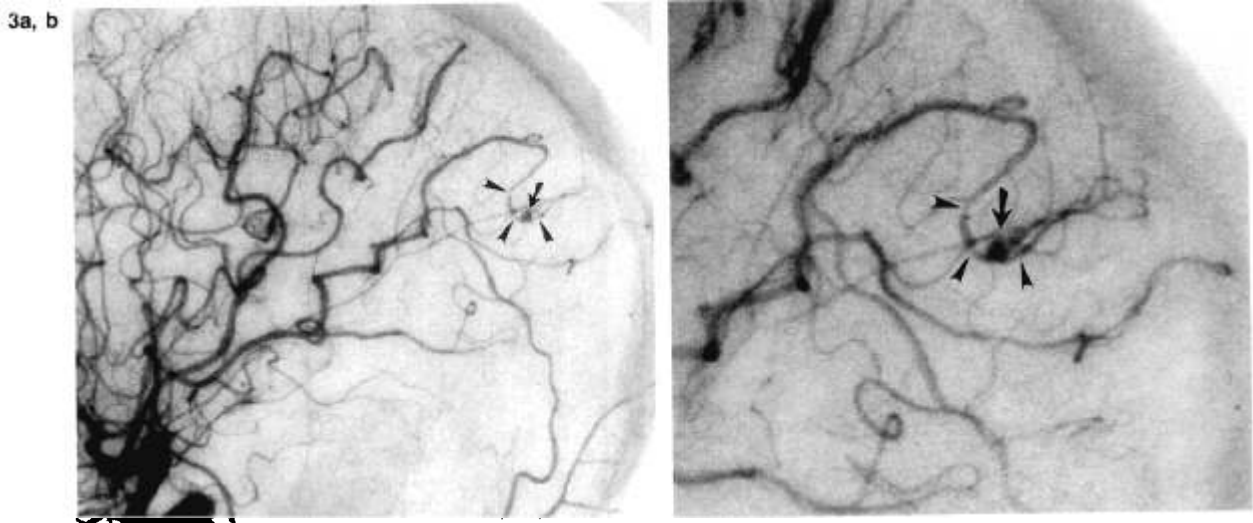


FIG. 3. a, b: Lateral views of the right carotid injection demonstrate irregularity of a parietooccipital branch of the middle cerebral artery (arrowheads) with a possible aneurysm (arrows) without thrombosis or contrast extravasation [(b) is a magnification of the vessel identified in (a)].

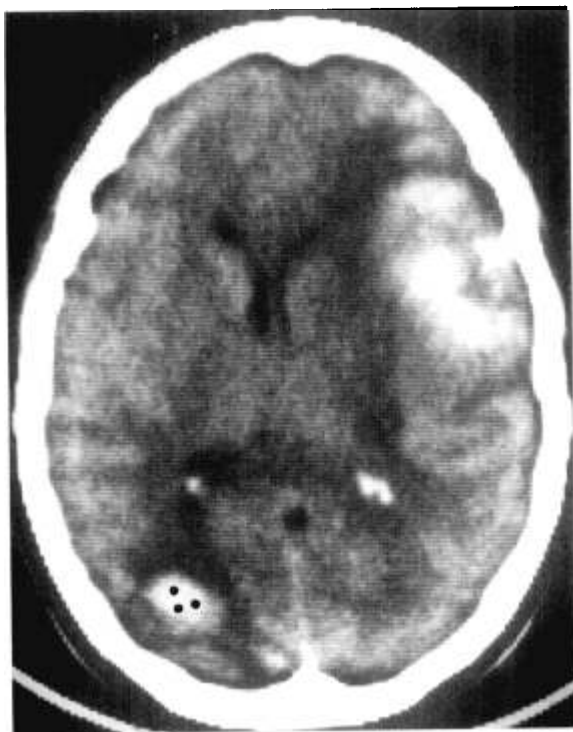


FIG. 4. Repeat CT 12 days after the initial scan demonstrates a new right parietooccipital hemorrhage (dots) and enlargement of the left frontoparietal hemorrhage with increased edema and mass effect.

considering all differential possibilities when a patient presents with multiple intracranial hemorrhagic lesions. In a young woman who has had a recent pregnancy, even without clinical suspicion of trophoblastic disease, and who presents with subarachnoid or intracerebral hemorrhage, choriocarcinoma should be considered.

REFERENCES

1. Athanassiou A, Begent RJH, Newlands ES, et al. Central nervous system metastases of choriocarcinoma. 23 years' experience at Charing Cross Hospital. *Cancer* 1983;52:1728-35.
2. Seigle JM, Caputy AJ, Manz HJ, Wheeler C, Fox JL. Multiple oncotic intracranial aneurysms and cardiac metastasis from choriocarcinoma: case report and review of the literature. *Neurosurgery* 1987;20:39-42.
3. van den Doel EMH, van Merrienboer FJMM, Tuleken CAF. Cerebral hemorrhage from unsuspected choriocarcinoma. *Clin Neurol Neurosurg* 1985;87:287-90.
4. Bjornsson J, Scheithauer BW, Leech RW. Primary intracranial choriocarcinoma: a case report. *Clin Neuropathol* 1986;5:242-5.
5. Krishna Rao CVG, Govinadan S. Case report. Intracranial choriocarcinoma. *J Comput Assist Tomogr* 1979;3:400-4.
6. Lee SH, Rao KCVG. *Cranial computed tomography*. New York: McGraw-Hill, 1983.
7. Houghton AL, Williams VM. *Cranial computed tomography. A comprehensive text*. St. Louis: CV Mosby, 1985.