CHORIOADENOMA DESTRUENS

(Case Report)

by

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Gestational trophoblastic disease, including hydatiform mole, invasive mole, and choriocarcinoma, constitutes a group of biologically interrelated conditions which are characterized by 3 unique features:

(1) they are derived from a mixed genetic background resulting from fertilization (hence the term gestational).

(2) their growth is represented by a biological tag in the form of chorionic gonadotrophin (HCG) which is similar to the HCG produced by normal human pregnancy and

(3) they are almost universally responsive to anti-tumour drugs which lead to a persistent remission in a high percentage of patients.

More than half a century age, Ewing while postulating the classification of trophoblastic tumours, named this entity as charioadenoma destruens. Chorioadenoma destruens, is also called invasive mole, malignant hydatiform mole and penitrative mole. Even when the penetration and proliferation is most marked, there is recognizable attempt at villus formation. It occupies an intermediate position between the benign hydatiform mole and the highly malignant choriocarcinoma.

This report is of a case which was misdiagnosed in absence of typical history and clinical symptomatology.

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CASE REPORT

A 32 years old female was admitted to hospital on 1st August 1975 with a history of amenorrhea of 5 months, breathlessness of 15 days, fever, cough of 3 days, abdominal pain and vomiting of 1 day duration. Abdominal pain was localised to the lower abdominal quadrants, and right hypochondrium, not related to food and not radiating anywhere. Vomiting was not projectile, no haemetemesis. Breathlessness was of exertional type to start with and later it was present even at rest. Fever was irregular in nature. Previous menstrual history was normal.

Obstetric History: Gravida IX and all the 8 were full term normal deliveries. Last delivery was 2 years ago.

Past history was not significant and no history of bleeding per vagina at all.

On examination patient was restless, pale, poorly nourished, and thin. Her pulse was 116/min. regular with fair volume and tension. Respiratory rate was 32/min. Temperature was around 99.6°F. Blood pressure was 130/70 mm Hg. and slight oedema of both feet was present.

On abdominal examination, uterine size was of 20 weeks' gestation, tenderness was present in right iliac fossa and right hypochondrium. Liver was palpably enlarged, 1 finger below right costal margin, tender; Spleen was not palpable, no other mass was felt.

A soft systolic murmer (haemic) heard in all areas, best heard in tricuspid area. On respira-

tory system examination, coarse crepitations were heard in left mid lower zones. Central nervous system did not reveal any abnormality.

Investigations microcytic hypochromic anaemia (Hb-42%). Total white cell count 9000/cum, E.S.R. 11 mm at the end of the 1st hour. Examination of the urine and stool showed no abnormality. Sputum for acid fast bacilli was negative. Screening of the chest did not reveal any radiological abnormality. The clinical diagnosis was respiratory tract infection with severe anaemia and early pregnancy.

She was put on Inj. Cryst, penicilin and on antianaemic treatment. After rest and therapy she was improving, but on 8th August, 1975, patient complained of excruciating pain in the right iliac fossa, and on the same evening she complained of reddish brown discharge per vaginam. On abdominal examination there was tenderness in right iliac fossa and muscle guarding was present. On pelvic examination cervix was normal, internal os was closed, there was tenderness in the right formix. General Surgeon suspected it to be a case of acute appendicitis and early pregnancy.

She was posted for an emergency laparotomy. On opening, the peritoneal cavity was full of blood and clots and to our surprise, vesicles were seen in the posterior aspect of the broad ligament on the right side and they were deeply infiltrating into myometrium. External surface was irregular, and nodular, showing haemorrhagic areas. Right ovary was enlarged. As general condition of the patient was poor subtotal hysterectomy with right salpingo-oophorectomy was done.

Postoperatively X-ray of the chest showed multiple secondaries in the lungs. Pregnancy test was positive. On dilution (1:100) the test was negative on the 10th day of surgery. Treatment with methotrexate (amethoptrin) 5 mg. thrice a day, was given for five days and repeated again, upto 150 mg. with due precautions. The patient was discharged on 30th day of admission. Check up after one month showed signs of regression of secondaries with negative pregnancy test. Patient was followed up with good result.

Histological section showed hyperplasia of the myometrial cells. Endometrial cavity and myometrium showed chorionic villi and trophoblasts, Chorionic villi showed degenerative and necrotic changes. Section from fallopian tube showed dark soft tissue masses with predominant necrotic changes and acute inflammatory infiltration, confirming the diagnosis of chorioademoma destruens. (Figs. 1 and 2).

Discussion

Novak has put forth the distinguished features of chorioadenoma destruens (1) Excessive trophoblastic overgrowth and (2) extensive penetration of the trophoblastic elements including whole villi into the depths of the myometrium, peritoneum, parametrium and vaginal vault. Such moles are locally invasive, though they generally lack the tendency to widespread metastasis as in choriocarcinoma. Unlike choriocarcinoma, it has a well preserved villous pattern.

Novak reports, a 10% incidence of intra-abdominal haemorrhage as in the present case. In almost every case the diagnosis is made at laparotomy and histological examination.

As subtotal hyterectomy was performed in this case the invasive mole was diagnosed on histological examination of myometrium.

The significance of the metastatic trophoblast is stressed by Attwood and Park in 1961, Villis in 1948, in the lungs and in the vagina by Haines (1955). It is established that in normal pregnancy, emboli of trophoblast may be transported by the blood stream to the tissues in the pelvis and in the lungs atleast in 43.6% cases, and they suggested the cause of the emboli are uterine contraction; placental commotion, and degenerative loosening of trophoblast from Villi. Lung deposits are known to regress with and without X-ray treatment after removal of the primary growth. The disappearance of the shadows in the lungs following treatment of a hydatidiform mole, whether benign or invasive, makes it likely that the deposits were benign emboli and not metastases. When there are Villi present in the

uterine lesion the lung deposits usually regress.

The prognosis of non-metastatic trophoblastic disease is better than that of metastatic disease whether treated by surgery or by chemotherapy. Chemotherapy is now an accepted method and in this case 150 mg methotrexate was given in two courses of 75 mg. each.

Summary

A case of Chorioadenoma destruens diagnosed at laparotomy is reported.

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Reference

1. Novak, E. and Woodruff, J. D.: Gynec. & Obst. Pathology 5th ed. Philadelphia W. B. Saunders Co., 1962.

See Figs. on Art Paper IX