IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 15, Issue 10 Ver. VI (October. 2016), PP 14-17
www.iosrjournals.org

Gestational Trophoblastic Disease: Report of A Rare Case of Placental Site Trophoblastic Tumor

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Abstract: *Gestational trophoblastic disease (GTD) is a spectrum of cellular proliferations arising from the placental villous trophoblasts encompassing four main clinicopathologic forms : hydatidiform mole (complete and partial), invasive mole, choriocarcinoma and placental site trophoblastic tumor (PSTT). Placental site trophoblasic tumours (PSTT) are rare and usually diagnosed after dilatation and curettage for missed abortion, but have also been described following term pregnancies and a hydatidiform mole. PSTT has a wide spectrum of clinical behaviour, ranging from a self-limited state to persistence to a highly aggressive metastatic neoplasm. A 23year old female G3p1l1a1 with 12weeks of gestation presented in the department with c/o per vaginal bleeding since 15days, her usg revealed triploid pregnancy with 4th fetus which revealed placental site trophoblastic tumour. Here, we study a rare case of placental site trophoblastic tumor.*
Keywords: *PSTT, GTD, hydatiform mole, trophoblast, invasive mole.*

I. Introduction

Gestational trophoblastic disease (GTD) is a spectrum of cellular proliferations arising from the placental villous trophoblasts encompassing four main clinicopathologic forms : hydatidiform mole (complete and partial), invasive mole, choriocarcinoma and placental site trophoblastic tumor (PSTT). The term "gestational trophoblastic neoplasia" (GTN) has been applied collectively to the latter three conditions, which can progress, invade, metastasize, and lead to death if left untreated.[1] Although GTD may occur as a pregnancy complication in women of any age, it is more common at teenage or advanced maternal age (40– 50 years)[2,].

GTD was historically associated with significant morbidity and mortality. Hydatidiform moles were often accompanied by serious bleeding and other medical complications prior to the development of early detection and effective uterine evacuation. The mortality rate for invasive mole approached 15%, most often because of hemorrhage, sepsis, embolic phenomena, or complications from surgery. Choriocarcinoma had a mortality rate of almost 100% when metastases were present and approximately 60% even when hysterectomy was done for apparent non metastatic disease.[3]

Placental site trophoblasic tumours (PSTT) are rare and usually diagnosed after dilatation and curettage for missed abortion, but have also been described following term pregnancies and a hydatidiform mole. PSTT has a wide spectrum of clinical behaviour, ranging from a self-limited state to persistence to a highly aggressive metastatic neoplasm. Here, we study a rare case of placental site trophoblastic tumor.

II. Case Report

A 23year old female G3p1l1a1 with 12weeks of gestation presented in the department with c/o per vaginal bleeding since 15days. 1st was a live issue of 4years and the 2nd was spontaneous abortion 1 year back. She underwent all the routine investigations along with thyroid profile and ultrasonography. Her usg revealed triploid pregnancy with 4th fetus suggestive of complete vesicular mole (as seen in fig 1). Her βhcg value was 7,45,000 and all the blood investigation reports were within normal limits including her thyroid profile. As the patient had given some relevant history, her CMV and HSV tests were done which showed positive. Her chest xray was normal. She was induced with misoprostol and she delivered all the 3 fetus with mole and underwent suction and evacuation on the 2nd day and the sample was sent for histopathology which revealed placental site trophoblastic tumour (as seen in fig 2 and 3). Regular followup is done with β HCG report. Her 1st week β HCG after suction and evacuation showed drastic fall of <5000, and the next week report showed 1545. And she's also presently on gancyclovir 500mg bd and has not undergone hysterectomy as she is desiring to conceive.

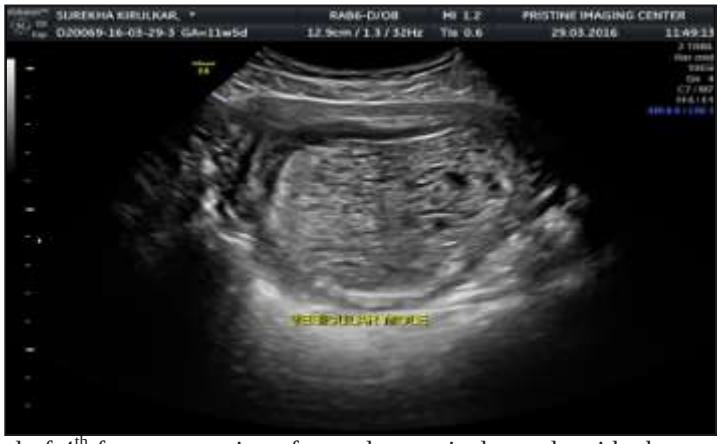

Fig 1: Pelvic ultrasound of 4th fetus suggestive of complete vesicular mole with characteristic vesicular pattern of multiple echoes, holes within placental mass, and no fetus.

Fig 2: Multiple grape – like vesicles that fill the uterus and entire placenta are seen. No fetal parts or membrane present.

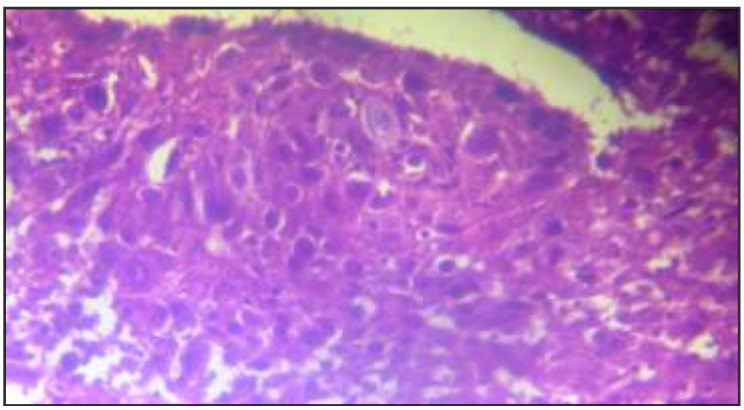

Fig 3: Placental site trophoblastic tumor with sheets of mononuclear intermediate trophoblast cells without chorionic villi infiltrating between myometrial fibers. (H&E 40X)

III. Discussion

Trophoblast is a gestational tissue which covers the blastocyst and provides route for nourishment between the maternal endometrium and the developing embryo in early pregnancy. Ultimately, it covers the surface of chorionic villi and forms the fetal portion of the placenta. Trophoblast is comprised of cytotrophoblast, syncytiotrophoblast, and intermediate trophoblast. In the various forms of GTD, different components of trophoblast show abnormal proliferation to a variable extent[5](as seen in table). PSTT represents a neoplastic proliferation of intermediate trophoblasts [2]. Unlike other forms of GTD, it is characterized by low β-hCG levels due to lack of syncytiotrophoblastic proliferation[6,7,8]. However, it shows increased expression of tissue as well as serum human placental lactogen (hPL)[8].

Partial moles are triploid, with 2 sets of paternal and 1 maternal haploid set. An embryo is usually present which dies by week 8-9. They most often occur following dispermic fertilisation. Complete moles are usually diploid, derived from paternal duplication or dispermic fertilisation of an "empty" ovum (lacking in maternal genes). The Chromosome count is either 46XX, from one sperm (75%) that duplicates its DNA, or 46XX or 46XY from the presence of two different sperms (25%). Placental site tumour is diploid from either the normal conceptus or a complete mole[9].

Two main risk factors increase the likelihood for the development of GTD - firstly the woman being under 20 years of age or over 35 years of age, and secondly history of previous GTD. Cases of GTD can be diagnosed through routine tests given during pregnancy such as blood tests and ultrasound, or through tests done after miscarriage or abortion. Vaginal bleeding, enlarged uterus, pelvic pain ordiscomfort, and hyperemesis are the most common symptoms of GTD. Since pregnancy is by far the most common cause of elevated serum HCG, clinicians generally first suspect a pregnancy with a complication. Therefore, if GTD is clinically suspected, serum beta HCG is also measured. The initial clinical diagnosis of GTD should be confirmed histologically, which can be done after the evacuation of pregnancy in women with hydatidiform mole however, malignant GTD is highly vascular. Histopathology of hydatidiform mole stained with hematoxylin and eosin shows villi of different sizes. Villous in the center exhibits marked edema with a fluid-filled central cavity which is known as cistern. Marked proliferation of the trophoblasts is observed. The syncytiotrophoblasts stain purple, while the cytotrophoblasts have a clear cytoplasm and bizarre nuclei. No fetal blood vessels are in the mesenchyme of the villi.

Treatment is always necessary. The treatment for GTD consists of the evacuation of pregnancy. Evacuation will lead to the relief of symptoms, and also prevent later complications. Suction curettage is the preferred method of evacuation. Hysterectomy is an alternative if no further pregnancies are wished for by the female patient. Methotrexate and dactinomycin are among the chemotherapy drugs used in GTD. Only a few women with GTD suffer from metastatic gestational trophoblastic disease which has poor prognosis. Their treatment usually includes chemotherapy. Radiotherapy can also be given to places where the cancer has spread, e.g. the brain. The risk can be estimated by scoring systems such as the modified WHO Prognostic scoring System, FIGO and the AJCC.

TABLE
Clinicopathologic features of gestational trophoblastic disease

Gestational trophoblastic disease	Pathologic features	Clinical features
Hydatidiform mole, complete	46,XX (mainly); 46,XY Absent fetus/embryo Diffuse swelling of villi Diffuse trophoblastic hyperplasia	15-20% trophoblastic sequelae hCG often ≥100,000 mIU/mL Medical complications
Hydatidiform mole, partial	Triploid (69, XXY; 69, XYY; 69 XXX) Abnormal fetus/embryo Focal swelling of villi Focal trophoblastic hyperplasia	<5% trophoblastic sequelae hCG usually <100,000 mIU/mL Rare medical complications
Invasive mole	Myometrial invasion Swollen villi Hyperplastic trophoblast	15% metastatic–lung/vagina Most often diagnosed clinically, rather than pathologically
Choriocarcinoma	Abnormal trophoblastic hyperplasia and anaplasia Absent villi Hemorrhage, necrosis	Vascular spread to distant sites–lung/brain/liver Malignant disease
PSTT	Tumor cells infiltrate myometrium with vascular/lymphatic invasion Intermediate cells/absent villi Less hemorrhage and necrosis Tumor cells stain positive for hPL	Extremely rare hCG levels less reliable indicator Relatively chemoresistant Mainly surgical treatment

hCG, human chorionic gonadotropin; *hPL*, human placental lactogen; *PSTT*, placental site trophoblastic tumor.
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