PLACENTAL SITE TROPHOBLASTIC TUMOUR WITH BRIEF REVIEW OF LITERATURE

PLACENTAL SITE TROPHOBLASTIC TUMOUR

Anna Pulimood, M.B.B.S.
Hemalatha Krishnaswami, M.D., D. Ch.
N. Balasubramaniam, M.D., D. G. O., F.I.C.S., M.N.A.M.S.

Norman Institute of Pathology and Dept. of Gynaecology and Obstetrics
Christian Medical College & Hospital
Vellore - 632 004. S. India.

SUMMARY

A case of placental site trophoblastic tumour is reported with a brief review of literature.

INTRODUCTION

Placental site trophoblastic tumour (PSTT) is a rare form of gestational trophoblastic disease. Originally described by Kurman in 1976 it was reappraised in 1981 by Scully and Young and given the present designation: PSTT. Of the approximately one hundred cases reported to date, none are from the South Asian region. This is the first reported case from India.

CASE REPORT

A 26 year old lady diagnosed to have complete hydatidiform mole, was treated with methotrexate following which hCG values returned to normal. Two years later...

Fig. 1: PSTT. Note the poorly circumscribed nodular tumour in the anterior wall extending from subserosal to subserosal regions.

Indian Journal of Cancer, March 96
she had irregular bleeding per vagina and low grade elevation of hCG for which she was treated with methotrexate, but the low grade elevation of hCG persisted. A D&C was done and a possibility of placental site trophoblastic tumour was considered and the patient treated with hysterectomy. One year after surgery the patient remains well and her hCG levels are normal.

PATHOLOGICAL FINDINGS

Macroscopically, the uterus showed a poorly circumscribed, nodular, yellow tumour measuring 5cm across, in the anterior wall, extending from submucosal to subserosal regions (Fig. 1). The cut surface revealed occasional small foci of haemorrhage. Microscopically the tumour was composed of polyhedral to rounded cells with cosinophilic to clear cytoplasm. The nuclei exhibited moderate variation in size and shape. There were less than 2 mitoses per 10 HPF. The tumour cells were arranged singly or in cords and sheets, characteristically separating individual or groups of muscle fibres (Fig. 2). A few of the tumour cells were spindle shaped and closely opposed to myometrial cells. Some blood vessels appeared completely replaced by tumour cells and masses of fibrin. Syncytial cells, spotty necrosis, haemorrhage and calcification were present. The tumour extended from endometrium to serosa. Immunohistochemically, there was diffuse positivity for cytokeratin and hPL, and focal positivity for hCG.

DISCUSSION

PSTT is a rare form of gestational trophoblastic disease believed to arise from extravillous intermediate trophoblast. The tumour occurs in the reproductive period at a mean age of 28 years, though it has been reported in postmenopausal women. The patients present with amenorrhoea, menorrhagia, metrorrhagia, uterine enlargement, occasionally with virilization, and rarely with nephrotic syndrome.
PSTT may follow normal pregnancy. In five percent of the cases there is a preceding history of spontaneous abortion or hydatidiform mole as was seen in our case. Serum hCG levels are elevated, but lower than those found in choriocarcinoma. Serum hPL levels are also elevated

The macroscopic appearance of PSTT is varied. The tumour presents as a polypoidal mass projecting into the uterine cavity or as a well defined or poorly demarcated intramural mass in the uterine wall. The cut section is tan, white or yellow. The tumour is soft with occasional foci of haemorrhage. The cervix, broad ligament, fallopian tube and ovary are occasionally involved

Microscopically, the tumour is composed of aggregates of intermediate trophoblastic cells that may be polyhedral, rounded or occasionally spindle shaped. The cells infiltrate, singly, or in cords and sheets, between muscle bundles and individual fibres. The cytoplasm is abundant and amorphophilic, but may be eosinophilic or clear. Multinucleate cells and nuclear pleomorphism may be present. Some nuclei are small, round and pale whereas others are large, convoluted and hyperchromatic or smudgy. Nucleoli may be prominent. The mean mitotic count is usually around two per 10 HPF. Occasional abnormal mitoses may be seen. There is characteristic extensive invasion of vessel walls, but these maintain their structure. The walls are partly or completely replaced by fibrin and the tumour cells are enmeshed in the fibrin and line the vessels. Plugs of tumour cells often fill vascular lumina. Fibrin deposition may be extensive. Chorionic villi are not usually seen. Immunohistochemically the tumour cells show diffuse positivity for hPL, cytokeratin and focal positivity for hCG. The features that suggest malignancy are a mitotic count of more than 5 per 10 HPF, a greater extent of necrosis and the presence of many cells with clear rather than amorphophilic cytoplasm. Metastases are commonly found in the lungs, vagina and paraaortic lymphnodes. Kidney, spleen, liver, stomach and brain may also be involved

PSST has to be differentiated from choriocarcinoma and other infiltrating malignant tumours. A history of a hydatidiform mole is much less common in PSTT than in cases of choriocarcinoma. Grossly, smaller foci of haemorrhage may be present in PSTT unlike the diffuse haemorrhagic appearance of choriocarcinoma. Microscopically, PSTT lacks the characteristic biphasic pattern with admixture of cyto and syncytiotrophoblastic cells. Also, the PSTT usually invades the myometrium by separating muscle bundles and fibres whereas choriocarcinoma forms a haemorrhagic mass that invades and destroys the myometrium. The pattern of vascular invasion and the presence of large masses of fibrin is also distinctive in PSTT. Immunohistochemically, in contrast to choriocarcinoma, there is a diffuse distribution of hPL, and a focal distribution of hCG. The beta-hCG levels are high and range from 1000 mIU/ml to over 100,000 mIU/ml in choriocarcinoma whereas in PSTT the levels are much lower

The infiltrating malignant tumours especially epithelial leiomyosarcoma can resemble PSTT. The distinctive pattern of vessel invasion and deposition of fibrinoid material found in PSTT are not found in other malignancies. The combination of histological features with diffuse staining for cytokeratin, hPL and focal staining for hCG also helps in the differential diagnosis

Exaggerated placental site reaction can present a diagnostic problem especially on

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Indian Journal of Cancer, March 96

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s and the rather than astases are vagina and ovary, PSTT pattern with trophoblastic invades the scele bundles and destroys vascular masses of in PSTT. contrast to a diffuse distribution are high and over 100,000 whereas in erant tumours sarcoma can ve pattern of a of fibrinoid not found in combination of se staining for ning for HCG diagnosis. reaction can especially on
curette specimens. Exaggerated placental site is only a focal lesion composed of intermediate cells infiltrating decidua and superficial myometrium without disturbing the normal architecture to a significant degree. There is a lack of mitotic activity. Fragments of chorionic villi and spiral arteries may be present. When in doubt, following a curettage, a close clinical follow up is suggested with monitoring of hCG levels. In PSTT the elevated serum hCG levels will persist following curettage whereas in normal gestation, it will return to normal. Persistent elevation of hCG titre is an indication for hysterectomy in view of the apparent lack of response to chemotherapy of these neoplasms.

Most cases follow an indolent and self limited course with tumour remaining confined to the uterus. A few however can invade the myometrium causing perforation and also invade broad ligament and ovary. Approximately 10-15% of cases may behave in a malignant fashion with disseminated metastases. The factors indicating poor prognosis, in addition to the presence of distant metastases, are a preceeding term pregnancy, high mitotic ratio and an older age of occurrence. The treatment of choice when the disease is localized is hysterectomy followed by hCG level estimations. In patients who wish to retain their fertility and in those who can be adequately followed up with hCG levels, a curettage can be a satisfactory procedure. The response to chemotherapy is generally poor. The treatment of malignant tumours is currently unsatisfactory, for these neoplasms do not respond well to the therapeutic regime that has met with such success in the treatment of choriocarcinoma.

REFERENCES

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