



A MILLIONAIRE OF DIFFERENT KIND- A CASE REPORT ON GESTATIONAL TROPHOBLASTIC NEOPLASIA

Obstetrics & Gynaecology

Dr Madhavi Baheti*	MBBS, Junior Resident, Department of Obstetrics and Gynaecology, Seth GS Medical college and KEM Hospital, Mumbai. *Corresponding Author
Dr Aditya R. Nimbkar	M.S. Obstetrics and Gynaecology, Senior Resident, Department of Obstetrics and Gynaecology, Seth GS Medical college and KEM Hospital, Mumbai.
Dr Jyotsna Dwivedi	M.S. Obstetrics and Gynaecology, Assistant Professor, Department of Obstetrics and Gynaecology, Seth GS Medical college and KEM Hospital, Mumbai.
Dr Kimaya Mali	M.S. Obstetrics and Gynaecology, Head of Unit, Department of Obstetrics and Gynaecology, Seth GS Medical college and KEM Hospital, Mumbai.

ABSTRACT

Aim and Background- Gestational Trophoblastic disease (GTD) is characterized by abnormal proliferation of trophoblasts, cell which produces B-hCG (human chorionic gonadotropin) and thus its used in diagnosis and plays a major role in management of the patient. **Case Description-** Gestational Trophoblastic disease ranges from premalignant- Hydatiform mole comprising of complete and partial mole and invasive mole to malignant conditions- choriocarcinoma, placental site trophoblastic tumor, epithelium trophoblastic tumor and atypical placental site tumor. **Conclusion-** We attempt to highlight the importance of B-hCG level and histopathological evaluation of early gestational failure products.

KEYWORDS

abortion, dilatation and curettage, early gestational failure, EMA-CO therapy, gestational trophoblastic neoplasia, serum B-hCG, suction evacuation, uterine arteriovenous malformation.

INTRODUCTION-

Incidence of GTN after ectopic pregnancy, miscarriage, normal pregnancy is ranging from 1:20000 to 1:50000.

Incidence after partial and complete mole is confounded by race and ethnicity ranging from 1-5% to 23-30% of cases respectively.

It comprises of choriocarcinoma, invasive mole and rarest form-placental site trophoblastic tumor and epithelium trophoblastic tumor (comprising 0.2% of GTN).

CASE-

A 27-year-old, P2L2A2 (previous 2 Full term normal delivery) came to emergency department with complaint of bleeding per vaginum and passage of clots along with abdominal pain, dull aching intermittent in nature. Patient was hemodynamically stable. She received 2 units of packed cell volume in view of hemoglobin of 6.6 at a private hospital. Patient gives history of spontaneous abortion 5 months back at 1.5 month gestation age which was followed by dilatation and evacuation and insertion of Copper T since patient wanted temporary method of contraception. Her complaints of bleeding persisted post procedure following which Copper T was removed, but still her complaint persisted. On USG uterus was bulky with heterogeneous mass of size 8.1* 4.6*7.2cm and showing raised internal vascularity with endometrial thickness of 60mm and right ovary showing 4.5*3.3 cm anechoic cyst with no internal vascularity, mostly benign in nature was seen. MRI showed T1 heterogeneous lesion 10*6 cm with few hyper intense areas, T2 heterogeneous lesion in endometrium with no myometrial infiltration - giving d/d of Arteriovenous malformation more than retained products of conception (RPOC). Serum B-hCG of 657141mIU/ml (25/11) and TSH of 0.01. Patient referred for interventional radiology to KEM Hospital. On examination Per abdominal uterus was 14-16 weeks size, soft, no guarding tenderness, rigidity elicited. On Per speculum cervix and vagina healthy, bleeding present with passage of clots. On Per vaginal examination uterus 16 weeks size, os open with clots inside uterine cavity. A repeat UPT (urine pregnancy test) was done which came out to be positive.

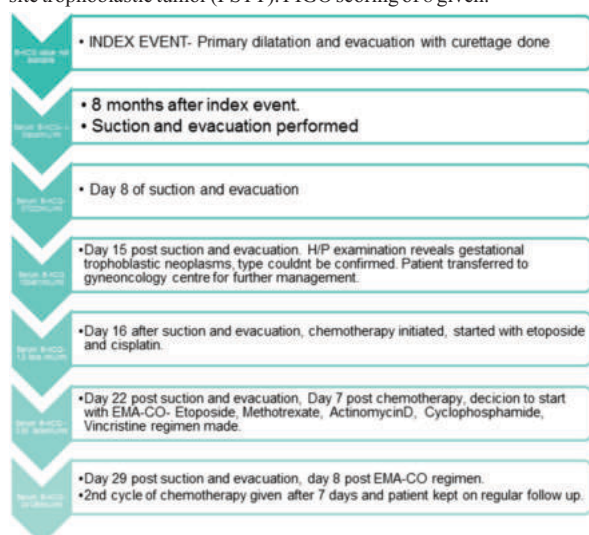
A repeat USG was done in KEMH and showed bulky uterus of size 8.2*9.1*10.2cm showing heterogeneous isoechoic content with numerous cystic spaces~ 140cc with no internal vascularity, fetal parts not visualized and right ovarian simple cyst of size 4*3cm seen - suggestive of complete molar pregnancy. Repeat CBC, Serum B-hCG, TSH done value of 10.8, >5lacs mIU/ml, 0.01 respectively (and all other investigations were normal) CXR was done to rule out metastasis, and was normal. Decision of suction and evacuation was made, and samples were sent for histopathology.

Weekly follow up of Serum B-hCG was done, > 5lacs mIU/ml (28/11) $\Rightarrow$ 57032 (5/12/23.) $\Rightarrow$ 100461 (11/12/23).

Post suction and evacuation a repeat USG - showing bulky uterus and isoechoic content with multiple cystic spaces measuring 6.5*6.9* 7.6cm (170-180cc) and internal vascularity with in along with right ovarian simple cyst measuring 3.7*3.2cm- suggestive of? choriocarcinoma.

On examination, per abdominal uterus 14 weeks size. On per speculum cervix vagina healthy no nodules seen. On per vaginal uterus 14-16 weeks size, mobile, fornices free.

Provisional histopathology report showing sheets of trophoblastic cells with marked pleomorphic nucleoli suggestive of choriocarcinoma, epithelium trophoblastic tumor (ETT) and placental site trophoblastic tumor (PSTT). FIGO scoring of 8 given.



Patient was referred to higher center (GTD center), all investigations were done, diagnosis of choriocarcinoma was made, CT scan of abdomen, pelvis, CT brain done to rule out metastasis and was started on chemotherapy starting with prephase Etoposide and Cisplatin, patient tolerated it well but drop of Hb from 9.6 to 7.7 was observed on day 3 and thereby and urgent CT angiogram was done- showing large heterogeneously enhancing ill-defined uterine mass with dilated

tortuous aneurysmal intramural and uterine vessels showing possibility of AVM. On day 7 Serum B-hCG value raised up to 3.93 lacs from 1.5 lacs (pre chemotherapy) and thereby decision to start with EMA- CO regimen- etoposide, methotrexate, actinomycin D, cyclophosphamide and vincristine was made, post day 8 of its first cycle serum B-hCG drastically reduced to 24126.

Decision to continue EMA-CO for 6 cycles and close follow up with any new symptoms and serum B-hCG is taken along with monitoring of USG ,X ray chest.

DISCUSSION:-

Gestational Trophoblastic disease ranges from premalignant- Hydatiform mole comprising of complete and partial mole and invasive mole to malignant conditions- choriocarcinoma, placental site Trophoblastic tumor, epithelium Trophoblastic tumor and atypical placental site tumor .

Hydatiform mole is Trophoblastic and villi proliferation with stromal edema. Persistent presence of GTD, marked by persistence presence of B-hCG level points towards Gestational trophoblastic neoplasia (GTN), therefore follow up with Serum B-hCG level is done for 6 months post complete mole and in partial mole 2 values being normal taken 4 weeks apart. GTN will show high value of B-hCG, exception is PTT and ETT where it may be within normal limits, this might be attributed to there cell of origin, choriocarcinoma arises from syncytio and cytotrophoblast while later arises from intermediate Trophoblastic cells.

Most common symptom being irregular vaginal bleeding. Final diagnosis is made with sonographic and histopathological findings. While medical management is preferred treatment, PTT and ETT are more resistant to chemotherapy and thereby surgical treatment is preferred in them. FIGO scoring system is use to grade severity of disease and also to decide on medical regimen, a score of less than 6 is required to give single drug regimen while 7 or more, Multi drug regimen - EMACO is used as in our case, where FIGO score is 8. The patient needs to be explained regarding the risk of premature ovarian failure with Multi drug regimen and thereby patient was given choice to freeze ovum prior to start of chemotherapy. Treatment is continued till serum B-hCG level is normal and 6 weeks thereafter.

One of the differentials for GTD is arterio venous malformation- a vascular hamartoma characterized by presence of shunts in between myometrial arteries and veins. One of the cause of AV malformation is post abortal curettage which was in our case, but it is ruled out on basis of B-hCG level and doppler findings, though gold standard diagnosis for AV malformation is digital subtraction sonography and gold standard treatment being hysterectomy.

Patient is counselled to not conceive for next one year and a regular follow up for 10years is recommended. For contraception hormonal pills, DMPA, barrier methods are considered better options.

CONCLUSION-

Histopathological assessment of products obtained from surgical or medical management of miscarriage should be done in the absence of any fetal parts, as many of them are diagnosed as early gestational failure on sonography and one of the major differential diagnoses being GTD. And thereby every miscarriage should be followed by urine B-hCG level check after 3 weeks and curettage needs to be avoided as it increases the risk of dissemination if GTD is diagnosed.

Timely recognition (from the index pregnancy to detection of disease) is most important prognostic factor.

With early recognition and prompt management GTN has a cure rate of up to 100%.

Acknowledgements:

A role of mention to all the assisting departments of Seth GS Medical College and King Edward Memorial Hospital, Mumbai for there unwavering support.

Conflicts Of Interest: None

Financial Disclosures: None

REFERENCES:

1. Brătîlă E, Ionescu CA, Vlădescu CT, Cîrstoiu MM, Berceanu C. Gestational choriocarcinoma after term pregnancy: a case report. Rom J Morphol Embryol.

2015;56(1):267-71. PMID: 25826515.

2. Bruce S, Sorosky J. Gestational Trophoblastic Disease. [Updated 2023 Nov 12]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK470267/>
3. Tamang T, Tshomo U. Gestational Choriocarcinoma with Varied Clinical Presentation and Treatment Outcome: A Case Series. J South Asian Feder Obs Gynae 2018; 10 (4):276-280.
4. Chandran JR, Devi SD, Gorhatti SN. Epidemiology of Gestational Trophoblastic Disease at a Tertiary Hospital in India over Last 8 Years. J South Asian Feder Obst Gynae 2019;11(1):27-29.
5. Calzolari S, Cozzolino M, Castellacci E, Dubini V, Farruggia A, Sisti G. Hysteroscopic Management of Uterine Arteriovenous Malformation. JSLS. 2017 Apr-Jun;21(2):e2016.00109. doi: 10.4293/JSLS.2016.00109. PMID: 28439193; PMCID: PMC5385144.