University Journal of Surgery and Surgical Specialities

ISSN 2455-2860 2019, Vol. 5(10)

RECURRENT MOLAR PREGNANCY- A RARE CASE REPORT

KALPANA S
Department of Obstetrics and Gynaecology,STANLEY MEDICAL COLLEGE AND HOSPITAL

Abstract : Hydatidiform moles are abnormal conception characterised by hydropic degeneration of villi and atypical proliferation of trophoblasts. Molar pregnancies are generally sporadic and the recurrence risk increases about 2 after a single molar pregnancy and it is about 10 to 23 after two or more hydatidiform moles. Presenting an interesting case of recurrent hydatidiform mole in 4 consecutive pregnancies.
Keyword : Hydatidiform mole, Recurrent mole

CASE REPORT:

36 year old nulliparous woman referred to our OG casualty as a case of recurrent molar pregnancy with a history of bleeding per vaginum for past one month. Patient had 4 consecutive molar pregnancies in her eight year married life. No history of similar illness in any other family members no history of consanguinity, and the interval between pregnancies were more than a year and her initial two pregnancies were diagnosed as molar pregnancy by ultrasound and were terminated by dilation and curettage at nearby hospital at three months amenorrhoea and histopathology report were unavailable and the patient was not followed up with beta HCG and she was not following any contraceptive measures. For the 3rd pregnancy patient admitted at our hospital at 3 months amenorrhoea and USG showed a molar pregnancy andpreevacuation beta HCG value is 42,029mIU/ml pregnancy was terminated by suction evacuation on 35-5-2011 and histopathology report came as partial mole And postevacuation weekly serum beta HCG values are 9410,8912,11516 IU/ml and metastatic workups are normal patient referred to medical oncology department and she was treated with single agent chemotherapy with weekly intramuscular methotrexate 75mg for 5weeks till beta HCG levels become undetectable. After that she did not come for follow up,and she could not be traced. Patient returned to our hospital on 21-02-2012 with a history of continuous bleeding per vaginum for 2 months.

Patient underwent dilatation and curettage for a fourth molar pregnancy at a nearby hospital on 06-12-2012 and post evacuation serum beta HCG was 32408 mIU/ml. patient was admitted and investigated. Haemoglobin 8.5grams and liver and renal function tests were within normal limits. Chest Xray was normal and USG showed multiple cystic spaces with hyperechoic solid areas suggestive of molar pregnancy. Patient was managed conservatively with antibiotics and anaemia corrected with two pints of blood transfusion and suction evacuation was done on22-02-2012 Products were sent for histopathological investigation. Patient followed up with serial beta HCG monitoring and post evacuation beta HCG values are11,981 on1-3-2013,12,500 on 09-03-13,13116 on 16-03-13 repeat USG showed s bulky uterus with thickened endometrium and cystic areas along lateral aspect with increased vascularity suggestive of invasive mole. Findings are confirmed by MRIpelvis, CT chest was normal, and the patient was referred to medical oncology department and and treated with single agent chemotherapy with intramuscular methotrexate 75mg weekly for seven weeks till beta HCG levels were undetectable. Karyotypingwas normal in both 0the partners and genetic counselling was given. Patient remains asymtomatic and she is under regular follow-up.

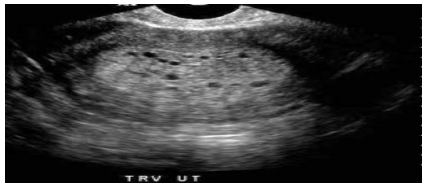

Fig-1

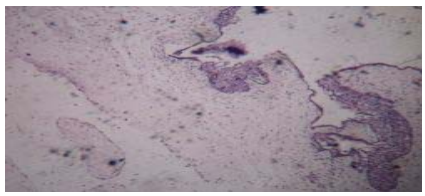

Fig-2-HPE-Partial Mole

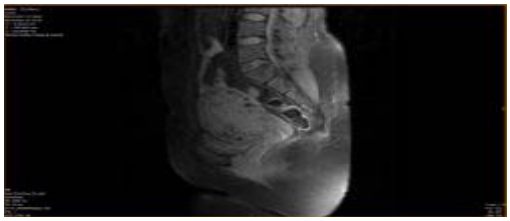

Fig-3- MRI-Invasive Mole

DISCUSSION:

Gestational trophoblastic disease refers to heterogenous group of interrelated lesions that arise from abnormal proliferation of placental trophoblasts. Incidence is about 1in 500 to 1in 1500 pregnancies. The world health organization has classified gestational trophoblastic diseases into two benign diseases termed complete and partial hydatidiform mole three malignant lesions termed invasive mole, choriocarcinoma, and placental site trophoblastic tumours(1,2). Hydatidiform moles are can be classified on the basis of histological examination and genetic origin as complete and partial moles. Partial hydatidiform moles are genetically triploid with two paternal and one maternal haploid sets of chromosomes (69,XXX, 69XXY, 69XYY). Complete hydatidiform moles are generally diploid(46XX, 46XY) and androgenetic in origin, all 46 chromosome being derived from the father. Complete molesmay be monospermic arising from fertilization of an anucleate egg by single spermatozoa which then duplicates its own chromosomes after meiosis or dispermic where anucleate egg is fertilised by two spermatozoa rarely, they can be biparental in origin having chromosome complement from both partners.(3) The etiology of hydatidiform mole are still unclear but several epidemiological risk factors for the development of molar pregnancy are recognised.

The most important one are extremes of maternal age and geographical factors. For women over 40 years of age ten fold increase of molar pregnancy compared to 1.3 fold increase with younger age (3,4). Other risk factors are vit A deficiency, low dietry of carotene,smoking ,irregular menstrual cycles and oral contraceptive use(6,7). Recently familial predisposition has also been evaluated. Recurrence is a rare, risk increases about 2% after a single molar pregnancy and 10to 20% after 2 or more molar pregnancies(1,2). Identification of genetic origin of recurrent molar pregnancies is important since it is related to future recurrences, increased risk of malignancy, future fertility is compromised and limited treatment options. Patients with positive family history of recurrent moles can be divided into two groups. Those with positive family history of recurrent complete moles and consanguinity usually genetically biparental in origin, they are extremely rare with only 21 families are reported in medical literature.

Genetic studies shows mutation in the NALP7 gene located in the chromosome 19q13.3 possibly responsible for this condition. Mutation in this gene responsible for abnormal ovum leading to complete hydatidiform mole and is inherited as autosomal recessive disorder(8,9).Patients with recurrent moles without positive family history and consanguinity usually androgenetic in origin. Suction evacuation is the preferred method of treatment, regardless of uterine size for patients who desire to preserve fertility(15). Follow up of patients with hydatidiform moles are by serial quantitative estimation of serum beta HCG is very essential since it allows early detection of trophoblastic diseases which has high potential of malignant change. Follow up is done with weekly serum beta HCG till it becomes normal thereafter monthly testing for 6 months is recommended. Criteria for diagnosing gestational trophoblastic neoplasia are plateauing of 4 beta HCG measurements over a period of 3weeks, rise of three weekly consecutive beta HCG measurements, beta HCG remains elevated for 6 months or more, histologic evidence of choriocarcinoma.Our patient had a rise of 3 weekly consecutive measuements of serum beta HCG values over a period of 2 weeks. Invasive mole is a common manifestation of gestational trophoblastic neoplasia characterised by the prescence of of chorionic villi accompanied with excessive trobhoblastic overgrowth and invasion. It penetrates deep into the myometrium involving peritoneum, adjacent parametrium and vaginal vault . It is locally invasive and lack the tendency to develop widespread metastasis typical of choriocarcinoma. It is called non metastatic form of gestational trophoblastic neoplasia. It develops 15 to 20% following complete moles compared with only 2 to 4% of partial moles (Sebire 2005a). Possible complications are intra peritoneal bleeding following perforation of the myometrium, vaginal bleeding following erosion of uterine vessels or nidus for infection. The prognosis is excellent for non metastatic form of GTN(Lurain, 1982).

STAGING:

Anatomic staging system for GTN was adopted by the International Federation of Gynaecology and Obstetrics (FIGO) (12) Stage I,Patient have persistently elevated HCG levels and tumour confined to the uterine corpus. Stage II, Patients have metastases to the genital tract. Stage III, Patients have pulmonary metastases with or without uterine, vaginal, or pelvic involvement. Stage IV, Patient have advanced disease and involvement of the brain, liver, kidneys, or GIT A prognostic scoring system proposed by the World Health Organisation reliably predicts the potential for resistance to chemotherapy. When the score less than 7 disease is low risk, score more than or equal to 7 disease is high risk.Patients with stage I disease usually have low risk, stage IV have ahigh risk score. The Gynecologic oncology group recommends weekly intramuscular methotrexate in a dose of 30to50 mg/m2 for non metastatic gestational trophoblastic neoplasia (Homesley,1988). Genetic aspect of our evaulation was not possible, except karyotyping the reason being that it is expensive investigation and our patient could not afford it. Our patient had recurrent molar pregnancy without positive family history and consanguinity probably androgenetic in origin. Donar insemination, artificial reproductive technologies may help to avoid further molar pregnancies in these patients. Artificial reproductive techniques would be appropriate for recurrent molarpregnancies where dispermy can be prevented by ICSI and monospermy can be prevented by selection of male embryos for implantation(14), in contrast ovum donation may be the option in familial recurrent complete molar pregnancies.

CONCLUSION:

Clinicians diagnosing and managing recurrent molar pregnancies should be aware of the potential complications like malignant transformation and genetic predisposition. Early detection, proper referral, management and follow up is very essential in these patients. These patients should have a genetic counselling regarding future pregnancies. Our patient had recurrent molar pregnancy with invasive mole. She is not willing for artificial reproductive techniques since she could not afford it and she completed chemotherapy, and resumed her menstruation and she is under regular follow up.

REFERENCES

1.Lorigan PC, Sharma S, BrightN,ColemanRE, Hancock BW.Characteristics of women with recurrent molar pregnancies.GynaecolOncol 2007;78;288-92.

2.SebireNJ, FisherRA, FoskettM, Rees H, Sekl M, Newslands E. Risk of recurrent hydatidiform mole and

subsequent pregnancy outcome following complete or partial hydatidiform molar pregnancy.BJOG2003:110;
3.Michel, J. And S. Edwards. 2003. Gestational trophoblastic tumours. In: R. W.Shaw.(editor). Shaw's text book of gynaecoiogy. 3rd ed. London: Black well Sci.,pp: 117- 123.
4.Berkowitz RS, IM SS, BernsteinMR,Goldstein DP. Gestational trophoblastic disease.Subsequent pregnancy outcome,including repeat molar pregnancy. J Reprod Med1998;43;81-6.
5.SebireNJ, Seckl MJ. Gestational trophoblastic disease: Current management of hydatidiform mole 2008;337:452-8.
6.Pour Reza M,Agheli N, BaghefiSB.Serumcreatinine and urea and protein level changes in hydatidiform mole JAMA 1974;230;580-1.
7.PalmerJR, Driscoll SG, Rosenberg L, Berkowitz RS, Lurain JR, Soper J, et al. Oral contraceptive pills in gestational trophoblastic tumours. J Natl Cancer Inst 1999; 91:635-40.
8.Moglabey YB, Kircheisen R, Seoudd M, El m ogharbel N, Van den VeyverI,Slim R. Genetic map maternal locus responsibie fir familial hydatidiform moles. Hum Mol Genet 1999;8:667-71
9.Zhao J, Moss J, Sebire NJ, Cui QC; Sekl MJ Xiang Y, Fisher RA. Analysis of the chromosomal region 19q 13.4 in two Chinese families with recurrent hydatidiform mole. Hum Reprod 2006; 21:536-41.
10.Berkowitz RS, Goldstein DP. Current management of gestational trophoblastic disease.Gynaecol Oncol2009;112:654-652.
11.Kohorn EI. Negotiating a staging and risk factor scoring system for gestational trophoblastic neoplasia J Reprod Med 2002; 47:445-450.
12.GoldsteinDP, Vzanten –Przybysz I, BernsteinMR,etal.Rivised FIGO staging system for gestational disease recommendations regarding therapy.JReprod Med1998;43:37-4,
13.Homesly HD, Blessing JA, Rettenmaier M, et al: Weekly intramuscular methotrexate for nonmetastatic gestational trophoblastic disease.Obstet Gynaecol72:413,1988.
14Reubinoff BE, Lewin A, Verner M, Safran A, Schenker JG, AbeliovichD. Intracytoplasmic sperm Injection combined with periimplantation genetic diagnosis for the recurrent gestational trophoblastic disease. Hum Reprod 1997;12:805-8.
15.BerkowitzRS,Goldstein DP. Current management of gestational trophoblastic disease.GynaecolOncol 2009;112:654-662.

An Initiative of The Tamil Nadu Dr. M.G.R. Medical University
University Journal of Surgery and Surgical Specialities