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Dr. Sangeeta Ramteke
Associate Professor,
Department of Obstetrics and Gynaecology, GMCH, Nagpur, Maharashtra, India

Dr. Mansi Shrigiriwar
Associate Professor,
Department of Obstetrics and Gynaecology, GMCH, Nagpur, Maharashtra, India

Dr. Renu Singh
Junior Resident,
GMCH, Nagpur, Maharashtra, India

Dr. Ashwin Sontakke
Associate Professor,
Assistant Professor, GMCH, Nagpur, Maharashtra, India

Corresponding Author:
Dr. Renu Singh
Junior Resident,
GMCH, Nagpur, Maharashtra, India

Invasive mole mimicking as AV malformation

Dr. Sangeeta Ramteke, Dr. Mansi Shrigiriwar, Dr. Renu singh and Dr. Ashwin Sontakke

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Abstract
Invasive mole is a form of persistent or metastatic gestational trophoblastic disease presenting usually after hydatiform mole 6-10 times more common than choriocarcinoma. It is defined as molar gestation invading the myometrium or uterine vessels. Persistent vaginal bleeding after evacuation of molar pregnancy and persistent elevation of beta HCG [1]. The presentation of secondaries is after few months to years in choriocarcinoma but immediately in cases of invasive mole [2].
The following case is a spectrum showing a diagnostic dilemma between choriocarcinoma or invasive mole or a consistent finding of AV malformation in the ultrasonography because of lack of an initial histopathological evidence of molar pregnancy from the evacuated products of conception and also posed a difficulty in deciding the mode of treatment considering the age of the patient whether conservative or radical surgery.

Keywords: Invasive mole, vaginal secondaries, AV malformation, multi drug chemotherapy, radical approach, beta - HCG

Introduction
Gestational Trophoblastic neoplasia is a spectrum of trophoblastic disease that include invasive mole, placental site trophoblastic tumour, epithelial trophoblastic tumour and choriocarcinoma. Persistent elevation of beta HCG following a molar pregnancy is referred as gestational trophoblastic neoplasia. Asian women are more predisposed to the condition [4]. Metastasis to distant organs occurs mainly by hematogenous, lymphatic and transcoelomic spread. Invasive moles are mostly limited to the uterine cavity with a very high predilection of invasion of uterus and to the uterine vessels.
USG findings can be confusing showing a heterogeneous mass filling the uterus or infiltrating myometrium or hyperechoeic myometrial focal mass with cystic areas.
High flow and multiple flow voids present similar to an AV malformation can pose a diagnostic difficulty as both can present with life threatening torrential bleeding presenting an obstetric emergency [2].
This finding can be similar to retained products of conception or adenomyosis or placenta accreta.
Diagnosis is mainly formed by the histopathological examination of the products showing dysmorphic villi and extravillous trophoblast invading the myometrium and blood vessels with absence of intervening decidua in invasive mole.
GTD are characterised by trophoblastic proliferation with mild to severe atypia.
In cases of secondaries, diagnostic feature is presence of myometrium and vessels invaded by the villi confirmed by resecting the tissue and subjecting it to microscopic examination [1].
The point of differentiation from choriocarcinoma will be lack of villi and cytologic atypia, while placenta accreta shows normal placenta with villous implants invading into myometrium without any intervening decidua without any hydronic changes in the villi [1].
Role of beta HCG and the doubling time has been an important diagnostic tool in such cases however it does not always relate to the activity of the tumour. Despite the low beta HCG, there is high chances of uterine perforation, haemorrhage and infection.
Low beta HCG with increasing invasion can be seen in PSTT, ETT and quiescent tumour due to the hetrophile antibodies present. These hetrophile antibodies are not released in the urine. However, beta HCG is very useful in deciding the mode of treatment along with other factors as shown in the WHO modified Bagshaw scoring system.

Table 1: Shows in Bagshaw scoring system

Factors	0	1	2	4
Age (year)	</= 40	>40		
Antecedent pregnancy	Mole	abortion	Term pregnancy	
Interval between pregnancy and start of chemotherapy	<4	4-6	7-12	>12
HCG levels pretreatment	<1000	1000- 10000	10000-100000	>100000
Largest tumour size (cm)	<3	3-5	>5	
Site of metastasis	Lung	Spleen , kidney	GIT	Brain , liver
Number of metastasis		1-3	4-8	>8
Prior chemotherapy			I drug	>/= 2 drugs

Role of ultrasonography and doppler is also important in diagnosis with usg showing 56% detection rate [4]. However on usg majority of complete moles can be diagnosed as delayed miscarriage / an embryonic pregnancy. And partial mole will be seen as cystic spaces in placenta with ratio of transverse to AP dimension of g sac >1.5 [4].

Av malformation presents as unexplained intermittent, vaginal bleeding with or without pelvic pain or dyspareunia, profuse bleeding and can lead to life threatening haemorrhage. Can be congenital and acquired following uterine trauma such as dilatation and curettage, pelvic surgery, GTD or choriocarcinoma. Usg features are suggestive of multiple cystic spaces with flow voids with high PSV intervening nidus with multiple feeding vessels.

This case deals with a delimma of diagnosis in a patient presenting with uterine bleeding with an inconsistent and unreliable history given and to establish an accurate mode of treatment modality.

Case report

A 16YR old presented to the emergency department with complaints of per vaginal bleeding, minimal spotting since 1 month and amenorrhea since 2 months. UPT test was positive. She was 8weeks by dates the outside USG reports were suggestive of a left tuboovarian mass and features of complete hydatiform mole. Repeat ultrasonography was also suggestive of the same. Patient was hemodynamically unstable with BP of 90/60mmhg and pulse rate of 130/min, patient had torrential bleeding, thus was shifted for an examination under anaesthesia to the operation theatre with 2 pint blood arranged and issued. The bleeding could be described as torrential with fresh blood gushing through the vagina and source could not be identified whether through the os or else where.

With difficulty the bleeding was identified to be coming from a vaginal nodule, a friable bluish nodule on the anterior fornix more on the left side. The bleeding was controlled by taking homeostatic suture bleeding controlled. Further proceeded with suction evacuation. The bleeding was immense and very few vesicles could be obtained. Patient was transfused with 3 pint PRC. Patient was started on higher antibiotics and beta HCG was done on day 3 which was 54000 and chest X-ray was normal. Repeat beta HCG on day 7 was found to increase by 51%. The post op period was uneventful but on day 10 she reported bleeding per vaginum with soakage of 2 pads per day. Repeat USG and MRI was done suggestive of an ill defined mass lesion , heterogeneous with multiple cystic spaces in the uterine cavity with complete loss of endomyometrial junction involving the anterior uterine wall. Suggestive of a neoplastic etiology ? Choriocarcinoma with ? AV malformation.

While the HPE report was awaited the patient was monitored by serial beta HCG levels as her initial risk score was 6 which was borderline ans the source of the uterine was yet not confirmed to be a secondary or an incidental finding of the AV malformation.

However on day 15 patient had a bleeding episode with blood loss of about 600-700cc which subsided on giving injection tranexamic acid and her tachycardia subsided eventually after correction of the blood deficit.

However her beta HCG was rising with a day 14 value of 90% of the initial value. Hence, decision to proceed with methotrexate therapy was taken. While a repeat bleeding episode made us question whether the AV malformation was causing the bleeding episodes so a uterine artery embolisation was decided upon and the b/l uterine arteries were emboli zed.

Meanwhile the HPE report came out to be inconclusive with only blood clots been visualised and the sample of the nodule was suggestive of RPOC, S. Both the findings were causing a diagnostic delimma and making the radio oncologist reluctant to start multi drug regimen.

On day 5 of embolisation the condition of the patient deteriorated with immense bleeding leading to shock and patient was shifted to OT for a repeat exploration.

The repeat exploration revealed an increase in the size of nodule and this time even more friable and vascular. Thus, making it difficult to achieve homeostasis.

Further 3 cycles of methotrexate were given which documented a falling trend of beta HCG and repeat USG showing a reduced mass size but the vascularity however persisted. Thus, pointing our diagnosis more toward a resolving uterine lesion with an AV malformation. This was not the end of her bleeding episodes. Further such episodes made us question the histopathology report and thus a review was ordered which was done after a thorough discussion explaining the clinical scenario and this time the accurate findings of chorionic villi along with trophoblastic tissue seen s/o invasive mole was reported. Also a repeat MRI was done showing a large lobulated mixed intensity lesion in fundus and upper body communicating with endometrial cavity and causing myometrial thinning, invasion along the anterior and superior wall of left side of fundus and upper body. Lesion extends upto the anterior serial surface of uterus measuring 4.3*3.4*3.2cm. Similar lesion in anterior wall of vagina just superior to the Introit-us measuring 3.2*2.5*2.3cm closely abutting the bladder neck and upper urethra, extending to the left paravaginal fat planes to indent the lower part of elevator and muscle.

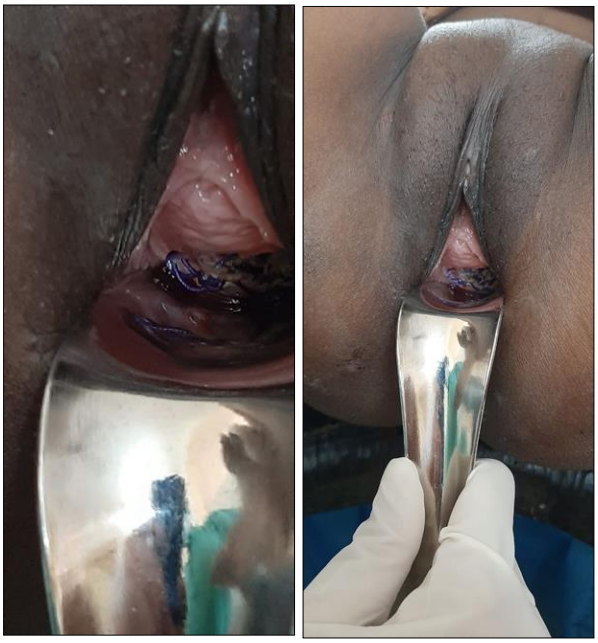

Image 1: Speculum finding of the friable bleeding nodule after taking homeostatic sutures.

The per speculum finding of the friable bleeding nodule after taking homeostatic sutures.
Multiple small vascular channels in the uterine wall and periuterine soft tissue around the lesion. Engorgement f right parametrical plexus s/o GTD invasive mole Vs choriocarcinoma.
Based on this the patient was started on EMA-CO regimen.
The recovery was hastened with rapid decline in beta HCG and no further bleeding episodes.

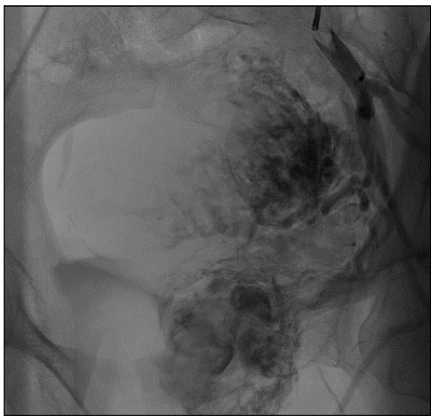

Image 2: Of the av communications prior to embolisation.

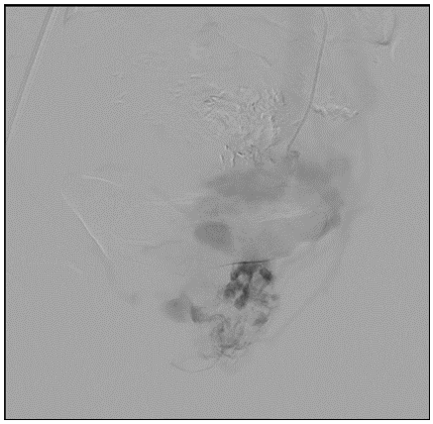

Image 3: After embolization.

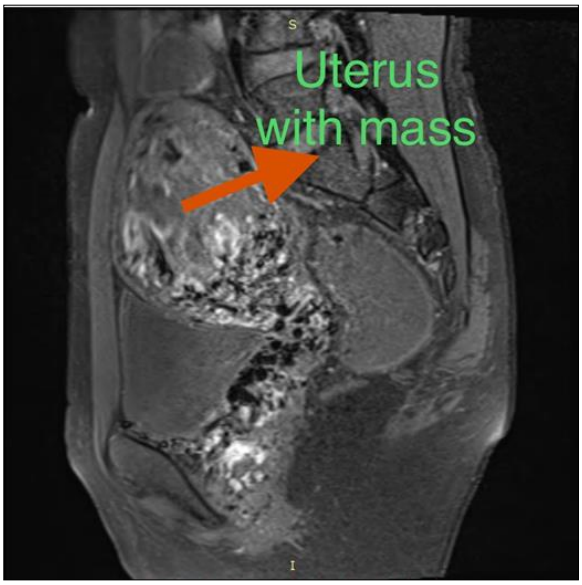

Image 4: MRI image showing the mass and the uterus with extension to the bladder and vagina.

Discussion

Persistent uterine bleeding per vaginum can be as a result of various causes including choriocarcinoma, AV malformations Uterine AVM can be congenital or acquired. GTD has been implicated as one of the causes of acquired AVM, [3]. Acquired AVM represent multiple small AV fistulas between intramural arterial branches and myometrial venous plexus, deep inside the myometrium [3]. Such hypervascularity can also be found in trophoblastic invasion that can mimic each other in doppler ultrasound. MRI is an excellent non- invasive method to determine the disease extent and to confirm the diagnosis of AVM [3].
Gestational Trophoblastic neoplasia comprises of both benign and malignant spectrum of disease and have propensity of local invasion and metastasis. Thus, USG with beta HCG forms a tool for diagnosis. This delimma lead us to consider various options like use of sclerosing agents, and also wide excision of the mass. But as the extent of the mass was doubtful and the fact that sclerosing agents would be washed off by the high flowing blood we finally went ahead with uterine artery embolisation.
Due to the repeated episodes of bleeding and rising trend of beta HCG levels hysterectomy to remove the source of tumor could also have been an option but considering the age of the female going ahead with conservative method was a the approach chosen. Multi drug regimen with 3 cycles of EMACO were given to the patient which showed a decline in the beta hcg levels and also no more bleeding episodes.
Thus we would conclude by saying that relying on our clinical diagnosis despite the fluctuating beta HCG levels, USG reports and adapting to a conservative approach should always be preferred before opting for a radical approach.

References

1. Bereck, Novak. Gestational trophoblastic disease Molar pregnancies Invasive hydatidiform mole Sonali Lanjewar, M.B.B.S., Raavi Gupta, M.D., 2017.
2. Diagnosis and Treatment of an Atypical Invasive Mole: I Nyoman Gede Budiana1 and Tjokorda Gede Astawa Pemayun 2 Oncogynaecology Consultant, 2 Obstetrics & Gynaecology Specialist, Obstetrics & Gynaecology Department, Udayana University, Sanglah General Hospital, Bali, Indonesia.

http://dx.doi.org/10.13005/bpj/1945 (Received: 15 February 2020; accepted: 09 April 2020)

3. Ruptured arteriovenous malformation secondary to placental site trophoblastic tumor: A diagnostic dilemma and its successful management, Egyptian journal of radiology, Avantika Gupta, Usha Manaktala, Nita Khurana 2018.
4. Uterine Arteriovenous Vascular Malformation Masked by Partial Molar Pregnancy: Diagnostic Challenge and Subsequent Embolic Treatment Sohail Contractor, Ryan Chung, [...], and Abhishek Kumar J Obstet Gynaecol India 2014;64(6):442-446. Published online 2014 Sep 26. Doi: 10.1007/s13224-014-0601-9
5. Uterine Choriocarcinoma: A Diagnostic Challenge (A Rare Case) KJ Jeevitha 1, P Devaki 2, Rathna Ramamurthy3, Raja Rajeshwari S4. Received on March 05/2015; accepted (revised) on Marh 11/2015.