Abstract: Introduction Uterine Artery malformation (AVM) is a rare but potentially life threatening complication with less than 100 cases reported in the literature. It may be congenital or acquired. Materials and Methods We report a case of a 19 year old lady referred with irregular bleeding following evacuation of vesicular mole. Her serum beta hCG levels were in the rising trend, after an initial fall, after the evacuation, with CT chest showing multiple discrete nodules in bilateral lower lobe of lung, giving a picture of metastatic trophoblastic disease. Ultrasound showed multiple cystic spaces with increased vascularity in posterior myometrium. MRI of the pelvis showed multiple dilated aneurysmal vessels of 5-18 mm seen at the uterine fundus and posterior myometrium suggesting uterine A-V malformation. Repeat CT of the chest showed non calcified nodule in the posterior basal segment of the right lower lobe with arterial feeder from right pulmonary artery suggesting pulmonary AV malformation. As the patient was a nullipara, uterine artery embolisation was done for preservation of future fertility. Conclusion Acquired uterine artery malformation is rare, even more, in our case there is a suspicion of associated pulmonary AV malformation. DC causes life threatening bleeding and high index of suspicion is needed in such cases.

Keywords: Vesicular mole, beta hCG, Uterine Arteriovenous Malformation, Embolisation

INTRODUCTION:

Uterine arteriovenous malformation (AVM) is a rare condition, with fewer than 100 cases reported in the literature.1 It may be congenital or acquired. Uterine arteriovenous malformation following molar pregnancy is a very uncommon condition which can be diagnosed by arteriography or by non invasive method like Colour Doppler. The most common diagnosis of irregular bleeding and elevated hCG post evacuation of molar pregnancy is, persistent trophoblastic disease. We report a case of congenital uterine arteriovenous malformation with associated pulmonary arteriovenous malformation who presented with a suspected diagnosis of persistent trophoblastic disease with lung metastasis.

CASE REPORT:

A 19 year old female, A2 was admitted to our hospital with recurrent episodes of bleeding per vagina. She had vesicular mole 2 months back being treated by suction and evacuation outside our hospital. Post evacuation ultrasonography findings were negative for remnants of conception. hCG levels prior to evacuation was >2,00,000 mIU/ml. Tissues were sent for histopathological analysis after suction and evacuation, revealing complete mole. Follow up with hCG showed post evacuation initial fall, with levels in rising trend after 12 weeks following evacuation. A private practitioner had recommended higher institutional care when the thoracic computerized tomography showed suspected metastatic disease. At the time of admission her vitals were stable. She was showing minimal bleeding per vaginum. On per vaginal examination, external os was closed and her uterus was normal sized. Her hemoglobin was 11g/dl, bleeding and clotting time were normal. Initial transvaginal ultrasonography with Colour Doppler imaging was done followed by Magnetic resonance imaging of the Pelvis which showed Multiple dilated aneurysmal vessels of 5-18 mm seen at the posterior myometrium and fundus and bilateral parametrium with no residual disease. These findings were suggestive of arteriovenous malformation.

The thoracic computerized tomography revealed a nodule in the posterior basal segment of the right lower lobe with a direct arterial feeder from the segmental branch of the right lower lobar pulmonary artery - giving a finding of pulmonary AV malformation. Cytological analysis of the bronchial washings ruled out metastatic tumour cells further confirming the diagnosis. She was asymptomatic for this pulmonary AV malformation as she has never had hemoptysis or dyspnoea in her life. She was treated for her uterine AV malformation with bilateral Uterine artery embolisation. Her pulmonary AV malformation was left alone as she was asymptomatic.

Oncologist’s opinion was sought for the rising levels of hCG which she was referred to us and she was kept on regular follow up with weekly hCG which showed decreasing trend until it reached undetectable levels without any chemotherapy. Computerised tomography of the brain and whole body
screening CT did not show AVM in any other region ruling out associated syndromes.

DISCUSSION:
The first case of uterine AVM was reported in 1926.1 To date, there are fewer than 100 cases reported in the literature. AVMs are characterised by multiple communications between arteries and veins centered at a vascular nidus, differentiating it from an AV fistula which has fewer or only one abnormal direct passage between an artery and adjoining vein. Uterine AVMs may either be congenital or acquired. Congenital AVMs occur due to an abnormality in the embryological development of primitive vascular structures resulting in abnormal vascular connections. Acquired AVMs are multiple small arteriovenous communications between intramural arterial branches and myometrial venous plexus, appearing as a vascular tangle. Acquired AVMs do not have a nidus, have only single or bilateral feeding uterine arteries and no supply from extramural vessels are implicated in endometrial carcinoma, cervical carcinoma, gestational trophoblastic disease, maternal diethylstilbesterol exposure and also occur due to trauma as in D&C, uterine surgeries.5In this case, it is possible that a congenital form of uterine AVM was present in the patient given the occurrence at other site – pulmonary AVM. However, if it was truly congenital rather than acquired, it is unclear why the patient did not present earlier in life. In order for the endometrium to bleed, endometrial blood vessels and surface epithelium must both breakdown. We can only postulate that the AVM was not large or superficial enough for the vessels to be exposed at the endometrium and subsequently bleed prior to evacuation.

Management of uterine AVM depends on the hemodynamic status, degree of bleeding, patient age, and desire for future fertility. Acute treatment involves stabilising the patient’s hemodynamic status, and stopping blood loss. Traditionally, a hysterectomy was the treatment of choice. However, the patient’s desire for future pregnancy is an important consideration, as there are now options available to avoid a hysterectomy.

CONCLUSION:
This case report highlights the importance of high index of suspicion for uterine AV malformations in patient who present with symptoms and signs of persistent trophoblastic disease. Dilatation and Curettage procedures would end up in life threatening bleeding. Unnecessary chemotherapy can be avoided if proper diagnosis is made by non invasive techniques like Colour Doppler imaging. Selective embolisation as a uterus preserving procedure is indicated in such patients nowadays.

REFERENCES:
4. Crofton Textbook of Pulmonology
5. Uterine artery embolisation and Gynaecological embolotherapy – James Spies