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Letters to the Editor - Brief communication

Placental mesenchymal dysplasia, a rare pathology

Dear Editor,

We report a case of a 16 year old primigravida referred to our center with a previous diagnosis of molar pregnancy and a 160/100 mmHg blood pressure at 28 weeks of gestation. On examination, she was noted to have 41 cm symphysis-fundal height, abdominal tenderness, diffuse bilateral edema in her lower extremities and hyperreflexia.

The ultrasound displayed duodenal atresia and intestinal duplication in the fetus and a 196x157x192 mm large irregular placenta, with multiple 2–3 millimeters cysts (Fig. 1a). Laboratory data revealed leukocytes 5.2 103/μl, platelets 116 103/mm3, human chorionic gonadotropin beta fraction (β-hCG) 201,258 IU/ml, thyroid stimulating hormone (TSH) 9.33 ulU/ml, thyroxine (T4) 0.914 ng/dL and lactic dehydrogenase (LDH) 822 U/L.

Despite pharmacological treatment, the patient remained hypertensive, therefore we decided to perform an emergency cesarean delivery. A viable female infant with APGAR score of 7 and 9 was delivered, the pediatric team reported weight of 1170 g, stature of 37.5 cm, cranium perimeter 26.5 cm, thoracic perimeter of 33.5 cm, abdominal circumference 24 cm and foot length 5.5 cm. Unfortunately, a few days later the infant died due to respiratory complications.

The placenta was fully extracted (Fig. 1b). Grossly examination showed a remarkable large specimen with 30 x 27 cm dimensions and weight 2940 g, in addition, multiple, irregular and confluent cysts were observed. Microscopy examination revealed enlarged villi, mesenchymal hyperplasia, hypervascularity of small vessels, chorioangiomas with alternated myxoid spots (Fig. 1c–d). Trophoblast abnormalities were not founded.

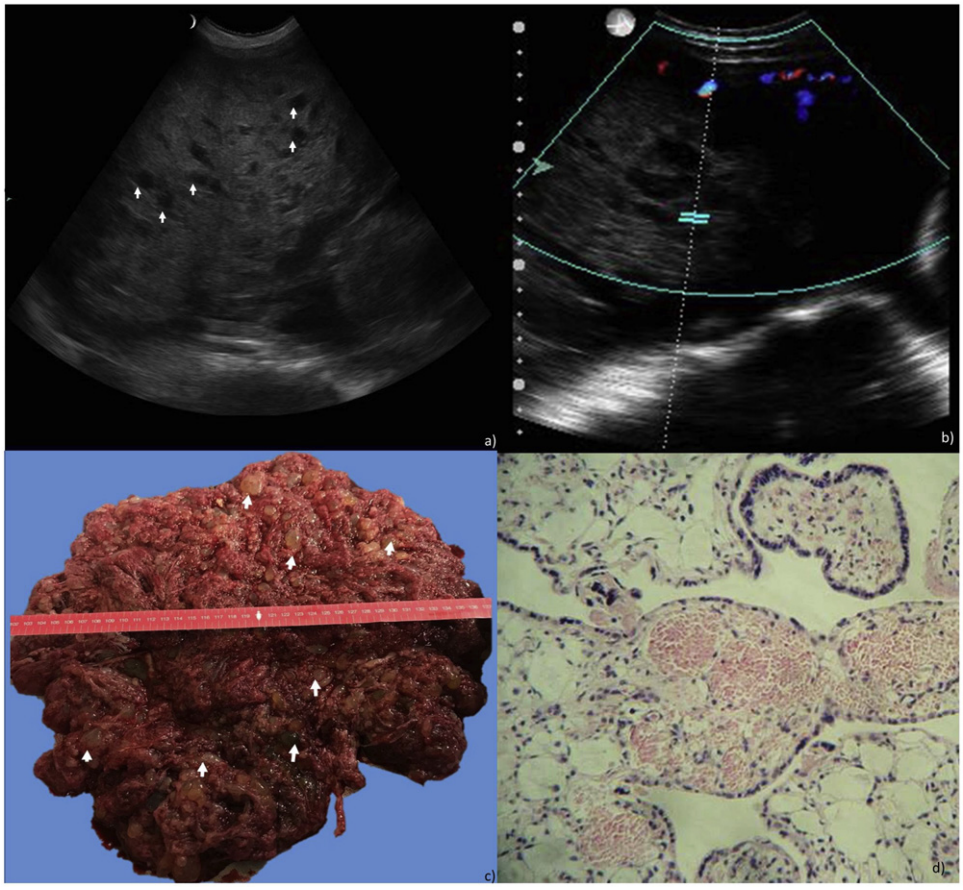

Fig. 1. a) Ultrasonographic image at 28 weeks' gestation, showing placental parenchyma with a multi-cystic pattern (arrows). b) Transabdominal color Doppler image at 28 weeks' gestation showing no-high velocity signal detection on the placenta c) Gross placental aspect, multicystic lesions (arrows). d) Microscopic placental aspect, hematoxylin and eosin x20 showing enlarged villi, mesenchymal hyperplasia, hypervascularity of small vessels, and absence of trophoblast proliferation.

After surgery, blood pressure came back to normal levels. The patient was translated to the Intensive Care Unit, five days later was discharged home without additional complications.

Placental mesenchymal dysplasia (PMD) is a rare placental stromal lesion, characterized by placentomegaly, cysts lesions, ectasia, tortuosity and thrombosis of chorionic plate and stem villous vessels [1]. Pathogenesis is not well elucidated, however, some theories have been proposed, including the mesoderm congenital malformation, molecular disruption of the imprinting genes of chromosome 11p15.5 associated with Beckwith-Wiedemann Syndrome (BWS) and androgenetic/biparental mosaicism [2]. The definitive diagnosis of PMD is made by histological examination, which shows mesenchymal hyperplasia, enlarged edematous stem-cell villi, dilated stem vessels with thickened vasculature and absence of trophoblastic proliferation of the placenta [3].

Ultrasound is the most effective diagnostic tool in prenatal monitoring. The multicystic placental lesion can be detected after the thirteenth week of pregnancy. The aneurysmal and varicose dilatation of the fetal chorionic vessels is another key sign that appears until the late second trimester [4].On the other hand, the ultrasonography can be useful to detect the structural anomalies associated with this pathology such as fetal skeletal dysplasia, fetal tumors (mostly hamartomas), Beckwith-Wiedemann syndrome, intrauterine growth restriction, and intrauterine fetal death [3].

The differentiation of PMD from molar pregnancy is crucial to prevent unneeded termination of pregnancy [4]. Heterogeneity and partially solid sonographic areas, finding by 2D ultrasonography, as well as high-velocity, and low impedance flow with low resistance, represent key findings to differentiate partial mole placenta [5].

Declaration of Competing Interest

The authors certify that they have no involvement in any organization with any financial or non-financial interest, in the subject matter discussed in the manuscript.

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Proposal for a new ICD-11 coding classification system for endometriosis

Dear Editor,

We report on work from our international taskforce established to improve the documentation of endometriosis in the upcoming 11th edition of the International Classification of Diseases (ICD-11). Our taskforce comprised representation from the World Endometriosis Society, World Endometriosis Research Foundation, European Society of Human Reproduction and Embryology, American Society for Reproductive Medicine, Society for Endometriosis and Uterine Disorders, European Society for Gynaecological Endoscopy, and British Society for Gynaecological Endoscopy.

The goal of ICD is to leverage information collected through health care systems to better understand predictors of clinical outcomes, implement data-driven strategies to improve outcomes and cost-effectiveness, and evaluate the changing impact of these strategies as health care evolves. Although new methodologies have been developed, and computing power has grown exponentially, the fundamental ingredient required for these analyses is high quality data, of which ICD coding remains a key component. For women with endometriosis, systematic ICD coding is clearly crucial to advance clinical care: it is a condition that has a high prevalence and is associated with a considerable diagnostic delay, high societal/personal burden, and no known cure.

Our taskforce noted the urgent need for improvement in the mechanics/focus of the coding process and in the structure/content of the codes for endometriosis. Classification systems for endometriosis exist including the revised American Society for Reproductive Medicine (r-ASRM) classification [1] and the Enzian classification [2,3]. There is also one validated outcomes prediction tool – the Endometriosis Fertility Index [4]. At present, no one single classification system has fully encompassed anatomic distribution and disease phenotypes nor a staging system that predicts or directs clinical outcomes such as symptom management, response to therapy, lesion and symptom recurrence, association with other disorders, or quality of life.

The nature of ICD coding lends itself to anatomical classification but even within this scope there were significant limitations with the ICD-10 classification. First and foremost, endometriosis was coded within ‘noninflammatory disorders of female genital tract’,