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Foetal Intra-Abdominal Umbilical Vein Varix in a Pregnant Patient with Diabetes Mellitus Type 2: A Case Report

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Abstract

We present a case of a foetal intra-abdominal vein varix (FIUVV) diagnosed at the 26th week of gestation in a woman with untreated diabetes mellitus. FIUVV is a rare congenital anomaly that can be encountered alone or in association with other congenital conditions. It has yet to be proven whether it is related to specific maternal risk factors such as diabetes mellitus, although it is already known that hyperglycaemia is linked to vascular congenital abnormalities, playing an important role in the endothelial cells' development.

Keywords

Vein varix; congenital anomalies; diabetes mellitus; venous system development

Introduction

Foetal intra-abdominal umbilical vein varix (FIUVV) is defined as a dilation of the umbilical vein between its entry in the abdomen and the portal system. It is a rare entity, with a frequency of diagnosis ranging from 1 case per 2300 births [1]. FIUVV is defined when the diameter of the umbilical vein (UV) is greater than 9 mm or at least 50% wider than its non-dilated portion. FIUVV can be isolated (150 cases described in literature) or associated to other foetal anomalies and aneuploidies with a frequency ranging from 5 to 28% in different studies [1,2]. The aetiology is not certain: there may be an intrinsic weakness of the vessel wall due to an abnormal development during embryogenesis or it might be caused by an abnormal blood flow with consequent dilation in the weakest part of the venous circulation [2,3]. The clinical significance in still unclear: it is associated with a high risk of poor foetal outcome, including intrauterine foetal death [4]. For this reason the foetal monitoring to exclude signs of cardiac failure or to diagnose the appearance of turbulent flow, that may be associated to the formation of thrombus in umbilical vein varix is indicated [4].

Some pathological conditions, such as pre-pregnancy diabetes, can increase the risk of congenital anomalies [5]. Nevertheless there is not enough evidence to speculate whether FIUVV is a possible congenital anomaly of the cardiovascular system due to the teratogenic embryonic effect of pre-pregnancy diabetes.

Case Presentation

A 39 year-old woman at her third pregnancy was diagnosed with pregestational diabetes

(blood glucose 229 mg/dl and glycated haemoglobin 58 mmol/ml) at 26 weeks of gestation. She previously underwent two caesarean sections: the first one due to cervical dystocia during labour and the second one because of placenta abruption at 36 weeks of gestation.

She did not perform any kariotype analysis. A sonographic evaluation at the 21st week of gestation displayed normal foetal anatomy. When diabetes was diagnosed at 26 weeks of gestation glucose daily controls were laid out and an insulin therapy was started. An ultrasonographic examination was performed to assess foetal health and growth. Foetal growth was in range and the morphology was regular except for an oval-shaped enlargement of the umbilical vein detected between the abdominal wall and the edge of the liver, measuring 15 mm of maximum diameter [Figure 1]. It was diagnosed as a varix of the foetal intra-abdominal umbilical vein (FIUVV) in its extra hepatic portion. The pulsed and colour Doppler confirmed the venous origin and showed no signs of turbulence.

A foetal echocardiography, performed few days later, showed no signs of hemodynamic distress and confirmed the absence of other cardiovascular congenital anomalies.

Foetal monitoring was performed twice a week in order to check amniotic fluid's levels, foetal growth and blood flow in the umbilical vein varix. Signs of turbulence appeared starting from the 32nd week of gestation (maximum diameter of FIUVV was 17 mm). Foetal growth was still regular while the amniotic fluid's level was closed to the upper limit from the 34th week of gestation.

Elective caesarean section was performed at 38 weeks of gestation. A healthy 3550 g male infant was delivered with Apgar scores of 9 and 9 at 1 and 5 min, respectively, and umbilical cord pH of 7.32. Mother and baby were discharged healthy three days later with indication of obstetrics, endocrinologic and neonatal follow-up.

Discussion

FIUVV is considered a rare congenital anomaly with an increasing frequency of diagnosis in the last decades [1]. This is due not only to the more widespread use of ultrasonography and colour-coded Doppler technology, but also to the increasing awareness concerning this pathology.

The pathogenesis of the lesion is still unknown. The most spread hypothesis ascribes the varix to a congenital intrinsic weakness of the umbilical vein's wall but it is also possible that the varix develops from a lesion due to an abnormal blood flow [2,3].

Since there are few cases in the literature is nowadays not possible to identify any maternal or gestational risk factors associated to the presence of this specific congenital anomaly and even if there is an increased risk in future pregnencies.

It is a common knowledge that congenital anomalies have multiple causes that are largely unknown; several factors play a role such as maternal age, obesity, genetic factors, folate levels, pregestational diabetes, etc. Among them diabetes mellitus (DM) is a risk factor with an OR of 2,1 for congenital anomalies, mainly concerning the urinary tract and the cardiovascular system [6].

Children born from women with pregestational diabetes (type 1 or 2) have a 2 to 10 times higher risk of congenital anomalies, with a rate of 93,4 per 1000 live births and no differences between type 1 and type 2 (general population rate of 41,9 per 1000 live births) [5].

Going into details, hyperglycaemia is related to a teratogenic effect on the foetal heart and vessels development caused by its capacity to down-regulate some angiogenetic factors, such as the vascular endothelial growth factor (VEGF), involved in myocardiocytes and endothelial cells' maturation [7] In vitro studies have shown that high glucose levels seem to inhibit the transcription of the nuclear factor through which VEGF genes are activated, reducing VEGF levels [8].

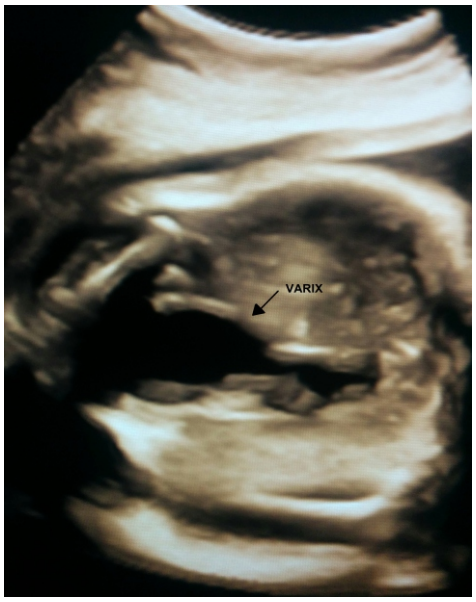
According to the "critical anastomosis theory" by White et al, most of the UV anomalies are due to an unsuccessful anastomosis between the umbilical and the vitelline veins, an early embriogenic step during the venous system development, between the 3rd and 5th week of gestation [9]).

Although this pathogenetic mechanism is plausible, up to now there is no literature available concerning the relation between FIUVV and pre-pregnancy diabetes as a risk factor.

FIUVV is associated with adverse foetal outcomes, even though recent literature is more reassuring. It could be associated to multiple abnormalities or to chromosomal anomalies (up to one third of the cases) [4-10]. When isolated it could bring to intra-uterine foetal death (4,5% according to recent literature) with two possible mechanisms: the formation of a thrombus due to the turbulent flow in the varix that may enter the foetal blood flow or a volume overload in the varix with a consequent hypovolemia that may lead to a cardiac insufficiency [1].

There is not a general consensus about the clinical management concerning the office follow-up and the delivery time. Most authors suggest a weekly follow up of foetal parameters, amniotic fluid level and ultrasonographic control of a possible turbulent flow in the varix and an expectant behaviour until spontaneous labour and vaginal birth [1,2,4,10].

Figures



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