



Extremely Dilated Pelvic Veins – The Answer May Lie Beyond the Pelvis

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Introduction

The dilation of ovarian veins plexi is one of the overlooked causes for chronic pelvic pain. Here we report a 52-year-old healthy woman who presented with asymptomatic huge pelvic varices, detected incidentally through routine pelvic ultrasound examination. A detailed evaluation led eventually to the diagnosis of an unexpected disease.

Case Presentation

A 52-year-old healthy woman was referred for evaluation of large asymptomatic pelvic varices that were diagnosed incidentally on routine pelvic ultrasound examination. Transvaginal sonography revealed an adenomatous uterus with normal appearance of the myometrial arcuate veins. The endometrial line was thin and regular. The ovaries were normal. The pelvic cavity, mainly the right side, was filled with extremely engorged and distended venous plexuses measuring 50 mm in maximal diameter (Figures 1, 2). No thrombi were visualized.

The patient was referred for further imaging work-up. However, before it was scheduled, pancytopenia developed (WBC 2470 K/micl, RBC 3660 M/micl, hemoglobin 8 g/dl, PLT 44 k/micl), and the patient was hospitalized.

Abdominal sonographic scan revealed an enlarged spleen with a span of 21 cm, a normal-sized

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Figure 1: Transverse view-engorgement of the pelvic veins in proximity to the right ovary.

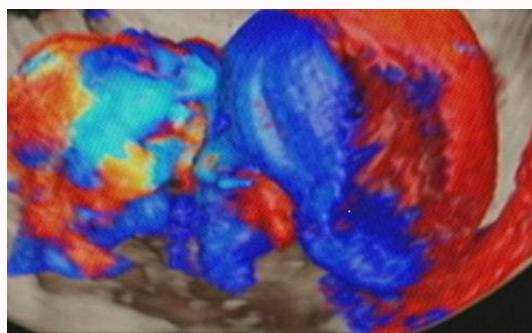


Figure 2: Transverse view- dilated pelvic veins depicted by color glass body 3D static filling the whole pelvic.



Figure 3: CT scan-transverse view-engorged veins to the right of the uterus.

liver with mild to moderate fatty infiltration, a normal portal vein (diameter 12 mm) and a large collateral vein connecting the splenic vein to the left iliac vein, compatible with portal hypertension. Subsequent Computed Tomography (CT) demonstrated an enlarged spleen (18.2 cm length), normal-sized liver, small gastric varices at the distal esophagus, varices between the splenic and right ovarian veins, and extremely dilated uterine and right adnexal varices with a maximal diameter of 46 mm (Figure 3, 4). Gastroscopy findings were consistent with the CT findings, demonstrating grade II varices in the distal esophagus with no active bleeding. The varices were ligated by endoligation. Liver biopsy performed *via* a transjugular approach revealed liver cirrhosis. Currently the patient has normal liver function and blood counts.

The venous vasculature of the pelvis drains *via* the ovarian veins and the internal, external, and common iliac veins to the inferior vena cava. Primary pelvic varicose veins are reported to be associated with pelvic congestion syndrome and may lead to chronic pelvic pain [1]. Possible etiologic factors include primary valvular deficiency, the nutcracker phenomenon, and May-Thurner syndrome. Valvular deficiency can result from absent or incompetent valves in ovarian veins resulting in reflux into the pelvic veins, which in turn dilate and become tortuous, forming pelvic varices [1-3]. The nutcracker phenomenon, also termed left renal vein entrapment syndrome, occurs when the left renal vein becomes compressed between the aorta and superior mesenteric artery. In May-Thurner syndrome, the left common iliac vein is compressed by the right common iliac artery [3]. The majority of cases of primary pelvic varices arise in the ovary, with left side predominance.

However, pelvic varices may also be secondary to non-gynecologic pathologies, such as inferior vena cava occlusion and vascular malformations. Inferior vena cava occlusion can result from thrombosis, tumor, or external compression [4]. Long-standing portal hypertension, usually due to cirrhosis, may also lead to the development of venous collaterals. Typically, these are paraumbilical collaterals, draining into the external iliac vein *via* the inferior epigastric vein or into the internal thoracic vein *via* the superior epigastric vein [5]. Occasionally, they drain into the internal iliac veins, causing pelvic varices [6]. In addition, pathologically increased blood flow due to large pelvic tumors and rare congenital disorders such as Klippel-Trenaunay-Weber syndrome (characterized by vascular nevas formation, deep venous thrombosis, varicosities) have also been reported to lead to pelvic varices.

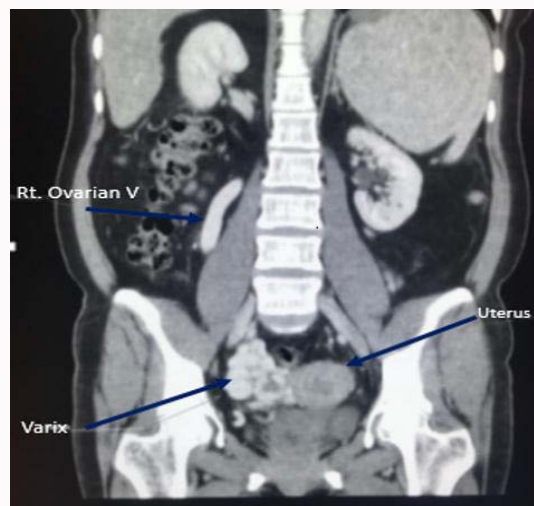


Figure 4: CT scan-Coronal view-engorged veins to the right of the uterus.

Transvaginal and transabdominal sonography are useful tools in the diagnosis of pelvic varices. Doppler flow provides dynamic visualization of venous blood flow [6] and can be used to distinguish varices from cystic adnexal masses [9-10]. The criteria for the sonographic diagnosis of varices include: 1) ovarian vein diameter greater than 4 mm; 2) dilated tortuous arcuate myometrial veins; 3) slow blood flow (less than 3 cm/s), and (4) retrograde venous blood flow [6,8]. However, to be defined as congestive pelvic syndrome, pelvic pain must be present.

CT and MRI, both noninvasive imaging modalities, may add diagnostic information in cases of secondary pelvic varices. Coakley et al. [8] proposed the following criteria for the diagnosis of pelvic varices by CT or MRI: Presence of at least four ipsilateral tortuous para-uterine veins of varying caliber of which at least one is wider than 4 mm or ovarian vein diameter greater than 8 mm.

There are reports in the literature of pelvic varices secondary to portal hypertension and portosystemic shunts and also associated with vaginal and/or vulvar varices that were described as an unusual cause of vaginal bleeding [9,10]. However, in these cases, the diagnosis of cirrhosis was already established.

To the best of our knowledge, this report is the first description of pelvic vein varices as the presenting sign of unknown underlying portal hypertension secondary to cirrhosis. Gynecologists and sonographers should be alert to this possibility when encountering patients with asymptomatic severely dilated pelvic veins. Multidisciplinary work-up and further imaging studies may lead to the diagnosis and appropriate medical care.

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