CASE REPORT

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Pelvic Congestion Syndrome with Enhanced Myometrial Vascularity Presenting as Abnormal Uterine Bleeding in A Nulli- Parous Women: Case Report

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ABSTRACT

Background: Pelvic Congestion Syndrome (PCS) is a chronic pelvic venous disorder commonly associated with chronic pelvic pain, dysmenorrhea, and dyspareunia. Presentation with abnormal uterine bleeding (AUB) is uncommon, particularly when accompanied by enhanced myometrial vascularity (EMV).

Case Presentation: Here, we report a rare case of Pelvic Congestion Syndrome with Enhanced Myometrial Vascularity in a young nulliparous woman presenting with prolonged AUB following misoprostol-induced expulsion of a nonviable twin pregnancy.

Conclusion: This case highlights an unusual presentation of PCS with concomitant enhanced myometrial vascularity, manifesting as prolonged abnormal uterine bleeding after pregnancy loss. Comprehensive evaluation using Doppler ultrasonography, MRI, and laparoscopy can facilitate accurate diagnosis, while progestin therapy may provide effective symptom control in selected cases.

Keywords: Pelvic congestion syndrome, Nulliparous, Abnormal uterine bleeding, Dysmenorrhea, Enhanced myometrial vascularity, Progestin

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INTRODUCTION

Pelvic congestion syndrome, also known as pelvic venous insufficiency, results from the incompetence of the internal iliac vein, ovarian vein, or a combination of pelvic venous structures.[1] Pelvic congestion syndrome can manifest in various forms, most commonly as chronic pelvic pain, dysmenorrhea, and dyspareunia, with some cases presenting as menstrual irregularities. The criteria for varices include an ovarian vein diameter of ≥6 mm, slow blood flow of <3 cm/s, and retrograde venous blood flow in the left ovarian vein combined with clinical vulval varicosities with the 3 Ds (dysmenorrhea, dysuria, and dyspareunia). The criteria for pelvic ultrasound diagnosis of varices include the visualization of dilated ovarian veins >6 mm, although 7 mm has been suggested as a cutoff. A previous study reported a positive predictive value of 83.3% for an ovarian vein diameter of 6 mm in diagnosing pelvic congestion syndrome.[2] Pelvic congestion syndrome mainly affects premenopausal multiparous women, but its incidence has also been reported in nulliparous women. To date, no cases of this syndrome have been reported in menopausal women.[3,4] In patients with chronic pelvic pain, the prevalence of the disease is nearly 30%.[5] Nearly 10% of women have isolated ovarian varices, and of this group, about 60% have pelvic congestion syndrome.[6] The overall prevalence of pelvic congestion syndrome ranges from 6% to 27% worldwide and remains a significant challenge for women's healthcare providers.[3,5] Recently, pelvic congestion syndrome was estimated to be present in up to 75.5% of patients with pelvic varicose veins, as reported in a 10-year retrospective analysis of a 600-female patient cohort conducted by Gavrilov et al. [7]

The exact etiology of pelvic congestion syndrome is unclear and is most likely dependent on multiple factors. Pelvic vein congestion can result from hormonal influences, venous valve insufficiency, venous obstruction, or secondary to concurrent medical conditions such as peripheral artery disease. The release of pain-inducing substances due to increased venous dilatation and stasis is a likely cause of pain in pelvic congestion syndrome.[8] There are various treatment options for pelvic congestion syndrome, including medical, surgical, and endovascular therapies.[9] This study aims to report an unusual case of Pelvic Congestion Syndrome with concomitant enhanced myometrial vascularity presenting as abnormal uterine bleeding in a young nulliparous woman after pregnancy loss, emphasizing the importance of considering pelvic congestion in the differential diagnosis of unexplained uterine bleeding and the role of multimodal imaging in diagnosis. The presentation of our case as isolated abnormal uterine bleeding (AUB), particularly in a young nulliparous woman following the medical expulsion of a nonviable pregnancy, is exceedingly rare. Furthermore, the coexistence of PCS with enhanced myometrial vascularity (EMV) poses a diagnostic challenge, as it may mimic retained products of conception, arteriovenous malformations, or other causes of abnormal uterine bleeding. This case highlights an atypical clinical presentation, demonstrates the value of Doppler ultrasonography, MRI, and laparoscopy in establishing the diagnosis, and illustrates successful management with progestin therapy. Reporting this case contributes to the limited literature on PCS-associated AUB and raises awareness among clinicians to consider pelvic venous disorders in the differential diagnosis of unexplained post-pregnancy bleeding.

CASE REPORT

A 25-year-old nulliparous woman with a history of two previous spontaneous abortions (no history of dilation and curettage) presented with a history of intermittent bleeding and abdominal pain for 25 days following misoprostol-induced expulsion of twin fetuses, as no cardiac activity was observed on ultrasound. There was no history of instrumentation. The post-expulsion period was unremarkable. Menstruation resumed two weeks after expulsion and continued for 30 days. The flow was moderate to heavy for 15 days and mild to moderate for the next 10 days, which was not relieved by medication. Her cycles were previously regular, every 28–30 days, with an average flow and duration of 3–5 days. The patient was otherwise healthy, with no similar history. There was no history of trauma, oral contraceptive use, mechanical symptoms, urinary retention or bladder incontinence, dysmenorrhea, dyspareunia, hematuria, or polycystic ovarian disease. There was no family history of varicosity or thrombo-occlusive disease. Her vital signs were as follows: blood pressure, 110/74 mmHg; temperature, 36.8°C; pulse rate, 80 beats/min; respiratory rate, 17 breaths/min; and oxygen saturation, 98% on room air. The physical examination was unremarkable, with no signs of superficial venous thrombosis or adnexal tenderness. The cervix and vaginal walls were normal. Her body mass index (BMI) was 20 kg/m². The laboratory results showed no evidence of inflammation.

Table 1: Laboratory report

Lab parameters	Values	Reference range
HB	10 gm/dl	12-15gm/dl
TLC	8000 cells/μL	4500-11000cells/μL
Platelet count	210000 cells/μL	150000-450000 cells/μL
CRP	3 mg/L	<3 mg/L
APTT	28 sec	25-35 seconds
INR	1.1	0.8-1.2
S.LDH	150 U/L	135-214 U/L
S.TSH	3.1 miu/ml	0.4-4.5 miu/l
S.PRL	10 ng/ml	3-27 ng/ml

Transvaginal ultrasonography revealed an ante-flexed and anteverted uterus with normal dimensions, no retained products of conception, and endometrial thickness within normal limits (4 mm), although it was heterogeneous in some areas. Both ovaries were within normal limits.

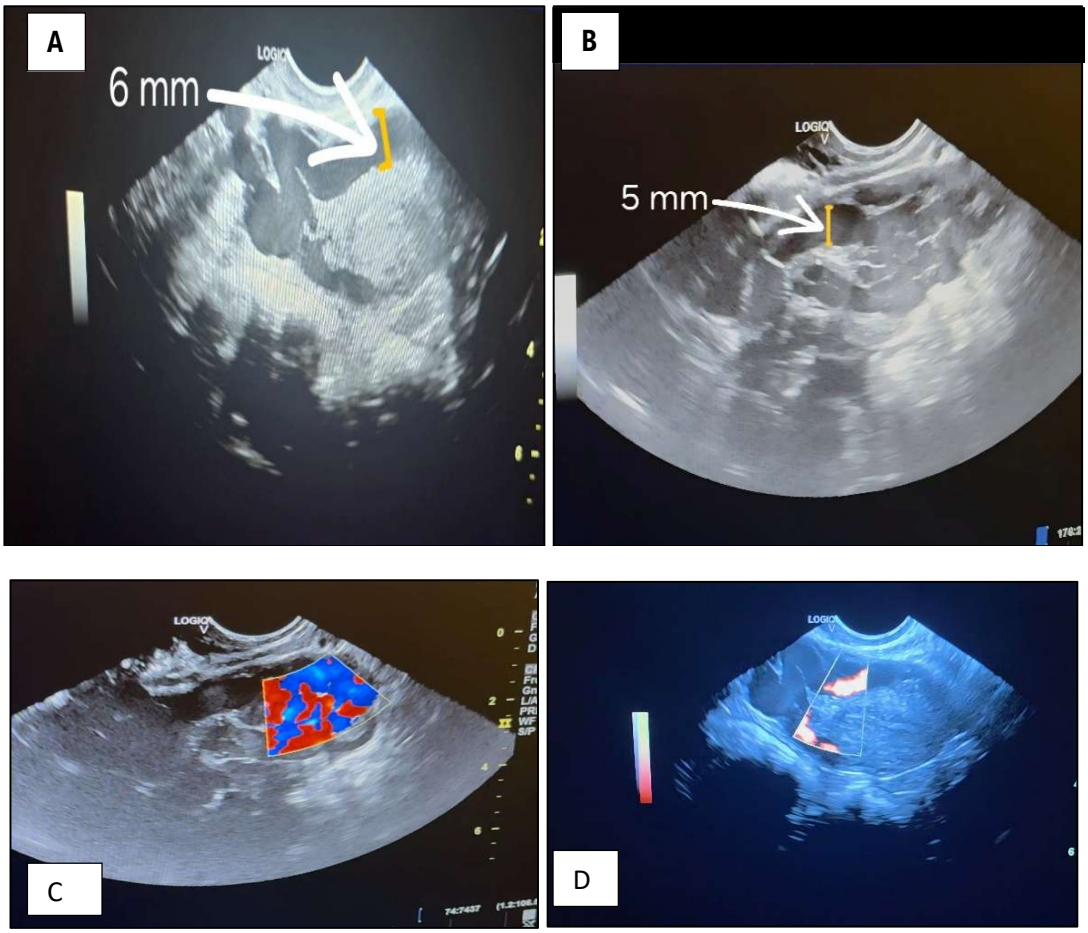

Figure1: **USG finding showing [A]enhanced myometrial vascularity of diameter 6mm,[B] multiple dilated vascular channels in both adnexa (5 mm diameter) and,[C],[D] increase perimetrial and myometrial vascularity on doppler and pulsed doppler respectively**

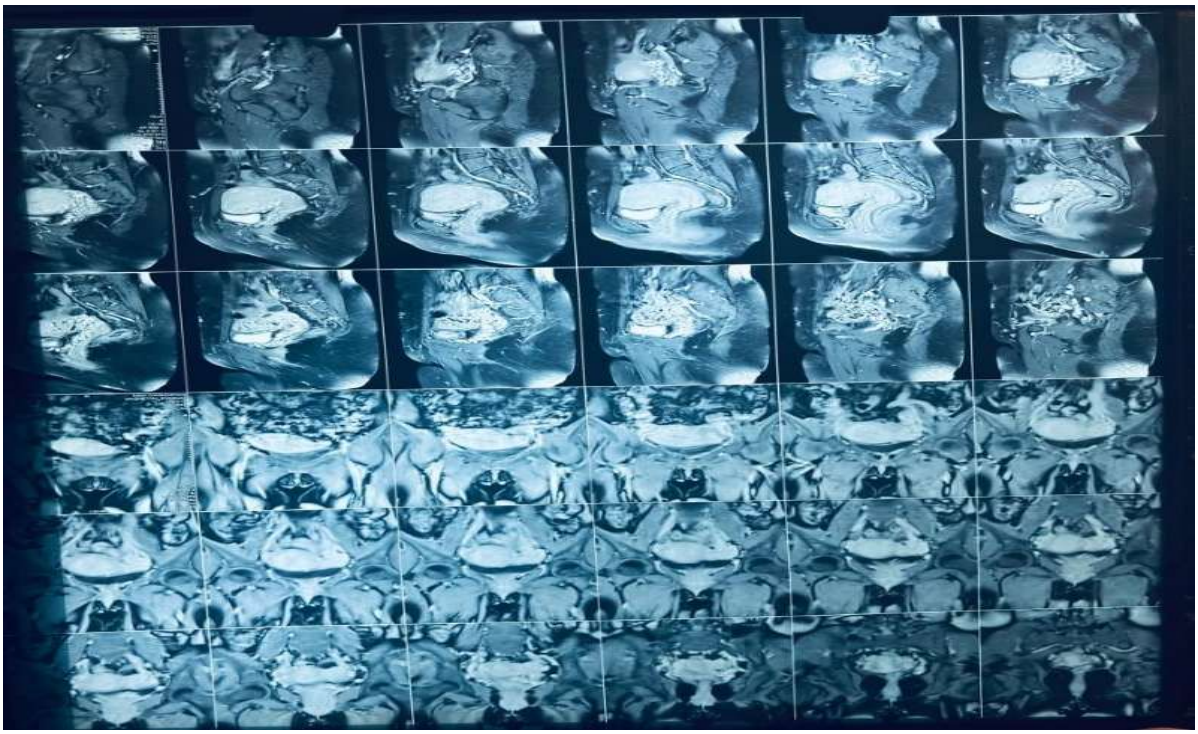

Figure 2: MRI report showing a diffusely inhomogeneous myometrial signal and multiple vascular channels with increased vascularity. Multiple dilated vascular channels with increased vascularity are seen in the bilateral adnexal regions

Increased peripheral vascularity was observed in the perimetrium and myometrial areas of the uterus. On both sides, multiple dilated tortuous veins of maximum diameter ~6 mm were observed in the adnexa (ovarian vein diameter>4 mm required for pelvic congestion). On the left side, venous flow was more prominent than that in the right adnexa, with reversed venous flow noted on color Doppler imaging.

The MRI report showed a bulky uterus with a diffusely inhomogeneous myometrial signal and multiple vascular channels with increased vascularity. Multiple dilated vascular channels with increased vascularity were observed in the bilateral adnexal regions (left greater than right), suggestive of pelvic congestion syndrome.

Because the patient could not afford CT pelvic venography, laparoscopy was performed. Laparoscopy revealed mild-to-moderate dilation of a small number of veins in the pouch of Douglas. Laparoscopic inspection of the left and right paracolic spaces revealed no obvious pathologies. No evidence of endometriosis was observed. The left and right diaphragmatic spaces were normal. The liver and gallbladder were also examined and found to be normal. The final diagnosis from the laparoscopy procedure was pelvic congestion syndrome (PCS). The patient was administered an intramuscular injection of depot medroxyprogesterone acetate(150 mg every 3 months for a total of 6 months). At the follow-up appointment in the gynecology outpatient clinic at 1 month3 month and six months interval, the patient gradually improved, and after 6 months, she was symptom-free.

DISCUSSION

Pelvic Congestion Syndrome (PCS) is a chronic pelvic venous disorder resulting from venous insufficiency of the ovarian and/or internal iliac veins. It is classically described in multiparous women of reproductive age and most commonly presents with chronic pelvic pain, dysmenorrhea, dyspareunia, and postcoital discomfort. Although PCS accounts for a significant proportion of chronic pelvic pain cases, its diagnosis remains challenging because of its nonspecific symptoms and overlap with other gynecological conditions. The present case is noteworthy because the patient did not present with the classical presentation of PCS. Unlike most reported cases, she presented primarily with prolonged abnormal uterine bleeding following misoprostol-induced expulsion of a nonviable twin pregnancy without chronic pelvic pain, dyspareunia, or dysmenorrhea. This atypical presentation contributed to the diagnostic challenge and initially raised concerns regarding more common causes of post-pregnancy bleeding, such as retained products of conception, gestational trophoblastic disease, or uterine vascular malformations. Previous studies by Bałabuszek et al. and Koo and Fan have emphasized chronic pelvic pain as the hallmark manifestation of PCS, often associated with multiparity and venous valvular incompetence.[8,10]. In contrast, our patient was nulliparous and had no prior history suggestive of pelvic or venous disease. Although pregnancy-related hormonal and hemodynamic changes are recognized contributors to pelvic venous dilatation, reports describing the development or clinical evident after pregnancy loss are scarce. The temporal relationship between fetal expulsion and symptom onset in our case suggests that pregnancy-associated venous remodeling and elevated estrogen levels may have contributed to the development or unmasking of venous insufficiency. An additional unusual feature was the coexistence of PCS with enhanced myometrial vascularity (EMV). Elagwany described the diagnostic confusion that may arise when EMV is identified on imaging, particularly in differentiating it from uterine arteriovenous malformations and retained products of conception.[11] Similar to the findings of this report, our patient demonstrated increased myometrial and perimetrial vascularity on Doppler ultrasonography and MRI. However, unlike true arteriovenous malformations, the vascular channels showed low-velocity venous flow associated with prominent adnexal varicosities and reversed venous flow, which is more consistent with pelvic venous congestion. This observation supports the hypothesis that venous hypertension associated with PCS may contribute to secondary myometrial vascular changes and abnormal uterine bleeding. Doppler ultrasonography demonstrated multiple dilated adnexal veins greater than 4 mm in diameter with reversed venous flow, while MRI confirmed extensive pelvic venous dilatation and increased myometrial vascularity. Although CT or conventional venography is often considered the diagnostic gold standard, financial constraints preclude these investigations. Diagnostic laparoscopy subsequently confirmed pelvic venous congestion and excluded alternative pelvic pathologies, such as endometriosis, pelvic inflammatory disease, and adnexal pathology. This highlights the value of combining noninvasive imaging modalities with minimally invasive surgical assessments when definitive radiological studies are unavailable. The management of PCS remains controversial, with treatment options including hormonal therapy, ovarian vein embolization, and surgical interventions. Medical management is typically the first-line treatment for pelvic congestion syndrome because of its noninvasive nature and lower associated risks. This approach is often combined with pelvic floor physical and cognitive behavioral therapy.[12] Up to 70% of pelvic congestion syndrome-affected females may be adequately managed through these conservative treatment approaches, although few studies assess long-term efficacy. Pharmacological options for managing pelvic congestion syndrome include gonadotropin-releasing hormone (GnRH) agonists, danazol, combined oral contraceptives, progestins, phlebotonics, and nonsteroidal anti-inflammatory drugs.[13] Symptomatic relief is observed when medroxyprogesterone is combined with psychotherapy. [15] Goserelin, a GnRH agonist, has better results in controlling pain than medroxyprogesterone

acetate. However, treatment cannot be continued beyond 1 year because it is a GnRH agonist.[14]

Most contemporary studies favor endovascular embolization because of its high success rate and minimally invasive nature. However, access to interventional radiology may be limited in resource-constrained environments. In our patient, treatment with medroxyprogesterone acetate resulted in complete symptom resolution and sustained clinical improvement at six months. This favorable outcome supports previous observations that hormonal suppression may be an effective therapeutic option in selected patients, particularly when fertility preservation and non-invasive management are desired. This case broadens the recognized clinical spectrum of PCS by demonstrating that the syndrome may present predominantly as abnormal uterine bleeding rather than as chronic pelvic pain. Furthermore, it highlights the potential association between PCS and enhanced myometrial vascularity following pregnancy loss, a relationship that has received limited attention in the literature to date. Increased awareness of this atypical presentation may facilitate early diagnosis, prevent unnecessary interventions, and improve patient outcomes.

CONCLUSION

This case highlights the importance of a multidisciplinary approach in managing PCS. Clinicians should maintain a high level of suspicion for PCS in women with chronic pelvic pain, conduct thorough evaluations, and utilize appropriate diagnostic imaging, endoscopic surgery, and pharmaceutical treatments. PCS typically affects multigravida and premenopausal women; however, it can also occur in premenopausal nulligravida women, as demonstrated in this case report. PCS generally presents as chronic pelvic pain; however, it can also present as an unusual finding after expulsion of the fetus. One probable cause of this is excess estrogen during pregnancy. Due to financial restraints, limited resource settings, unavailability of CT venography, and sequential follow-up imaging, the study has some limitations. Therefore, further research is necessary to understand the precise pathological mechanisms involved and establish a gold-standard treatment method that effectively addresses this syndrome.

Declaration of patient consent: The authors certify that they have obtained all the appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published, and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Declaration of non-use of generative AI Tools: This article was prepared without the use of generative AI tools for content creation, analysis, or data generation. All findings and interpretations are based solely on the authors' independent work and expertise.

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