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Uterine Ischemia Secondary to Chronic Procidentia: A Rare Complication of Advanced Pelvic Organ Prolapse

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ABSTRACT

Background:
Uterine ischemia is an uncommon condition typically associated with uterine artery embolization or malignancy.

Case Report:
We report a rare case of uterine ischemia secondary to complete uterine prolapse in a generally healthy 53-year-old woman with fourth degree prolapse (procidentia). The patient had undergone significant weight loss prior to elective pelvic reconstructive surgery. At the time of the planned abdominal subtotal hysterectomy and sacrocervicopexy unexpected findings included uterine ischemia with necrotic areas and diffuse peritoneal inflammation. Due to concerns about tissue integrity the intended procedure was modified.

Conclusion:
This case highlights a previously unreported complication of advanced uterine prolapse and raises the possibility of vascular compromise exacerbated by mechanical factors and rapid weight loss. Awareness of this rare complication may aid surgical planning in similar cases.

Keywords: Uterine ischemia, pelvic organ prolapse, procidentia, sacrocervicopexy

INTRODUCTION

Pelvic organ prolapse (POP) is a common condition affecting up to 40% of women aged 50-79 years.[1] The prevalence of uterine prolapse has been shown to be as high as 14% among women.[2] Common symptoms include pelvic pressure, vaginal bulge, and urinary complaints. While certain complications of POP such as hydronephrosis are well described in the literature,[3] vascular compromise of the uterus is not a recognized sequela of POP. Uterine ischemia has been described in the context of malignancy, postpartum complications, and following uterine artery embolization. To our knowledge, this is the first reported case of uterine ischemia associated with chronic uterine prolapse in the absence of an acute vascular event. This case may inform surgical decision-making in patients with long-standing procidentia.

CASE REPORT

A 53-year-old married mother of two with a history of fourth degree uterine prolapse was scheduled for elective abdominal subtotal hysterectomy and sacrocervicopexy. She had no significant past medical history, was a non-smoker and was not on any chronic medications. Her obstetric history included two normal vaginal deliveries. Her surgical history consisted of a rectoenterocoele repair performed 10 years prior and laparoscopic appendectomy and cholecystectomy. The patient had been suffering from uterine prolapse for a few years. She had been symptomatic with a dragging pelvic sensation and discomfort and had not tolerated pessary use, prompting her desire for surgical intervention. She denied acute pelvic pain, vaginal bleeding or any systemic symptoms. To optimize surgical outcomes and reduce the risk of complications she was advised to lose weight and she successfully lost approximately 32 kg over a period of six months through diet and exercise, bringing her body mass index (BMI) down from 38 kg/m^2 to 25. The patient was then deemed suitable for surgery and the case was presented and approved at a multidisciplinary team meeting in accordance with departmental protocol. Preoperative pelvic ultrasound showed a normal uterus with heterogenous echotexture and multiple small fibroids. The endometrium was regular, Both ovaries were normal in shape and size, and there were no masses or free fluid in the pelvic cavity (Figure 1).

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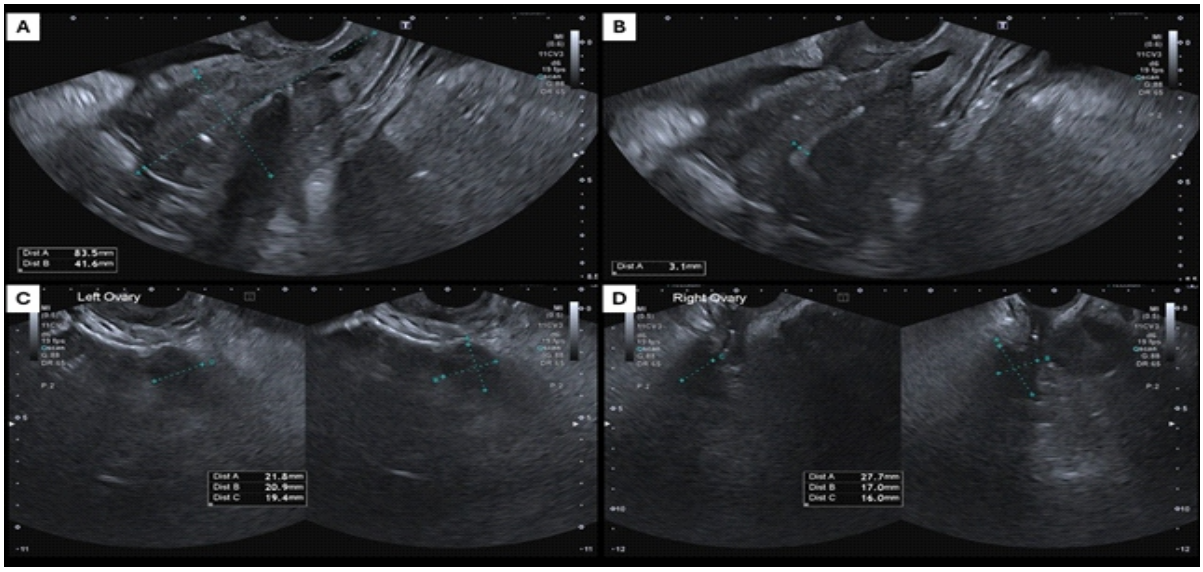

Figure 1:The uterus was normal in shape and size, with no structural abnormalities (A). Endometrial thickness measured 3 mm (B), The adnexa appear normal, both left (C) and right (D).

The uterus was normal in shape and size, with no structural abnormalities. Endometrial thickness measured 3 mm, The adnexa appear normal, both. There were no masses or free fluid in the pelvic cavity. Preoperative blood count showed borderline anemia (hemoglobin 119 g/L) and was otherwise normal, with no leukocytosis.

Under general anesthesia, the patient was found to have a complete procidentia with the cervix protruding approximately 20 cm beyond the introitus. The cervix was engorged but showed no ulceration or signs of infection. An intrauterine device was removed preoperatively without complication. A Pfannenstiel incision was performed. Upon entry into the peritoneal cavity, a large volume of clear fluid was encountered. The peritoneum appeared thickened and diffusely inflamed. The uterus, enlarged to approximately the size of a 12-week pregnancy, appeared pale, with a uniform white appearance and an area of dark necrosis noted on the posterior uterine wall. The uterine and pelvic tissues appeared markedly edematous and friable, easily tearing with minimal manipulation. The fallopian tubes were dilated and adherent to the pelvic sidewalls along with the ovaries.

A decision was made to proceed with a subtotal hysterectomy and adnexectomy but to abort the planned sacrocervicopexy. This was due to concerns that mesh fixation to the friable cervix would not be durable and that the peritoneum was too inflamed and immobile to provide appropriate coverage for the mesh. The left ovary was not removed due to adhesions. Her postoperative recovery was uneventful. She was given 24 hours of intravenous cefalexin and metronidazole, followed by five days of oral antibiotic therapy. Vaginal estrogen was initiated postoperatively.

Histological analysis showed a normal endometrium. There were signs of possible adenomyosis, and the presence of two fibroids smaller than 10 mm. The uterine serosa was focally edematous, and there was chronic salpingitis affecting both fallopian tubes. There were no features suggestive of malignancy, and no structural lesion identified to explain the ischemic changes (Figure 2).

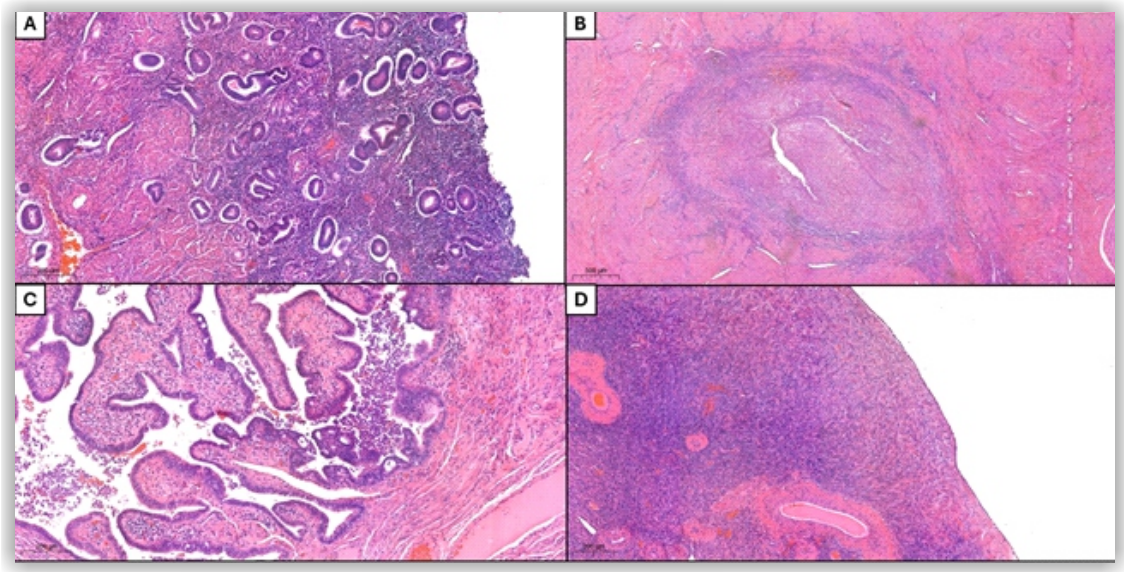

Figure 2: H&E staining of histopathology specimens demonstrates normal endometrium (A), myometrium with adenomyosis and focal ischemia (B), fallopian tube with evidence of salpingitis (C), and normal ovarian tissue (D).

Inflammatory markers were obtained three weeks postoperatively; C-Reactive Protein (CRP) was within normal limits (1 mg/L), while erythrocyte sedimentation rate (ESR) was found to be slightly elevated (32 mm/h). The patient is scheduled for a diagnostic laparoscopy at six months to reassess peritoneal inflammation and tissue viability, with the possibility of delayed sacrocervicopexy depending on intraoperative findings.

DISCUSSION

This case illustrates a unique and previously unreported complication of advanced uterine prolapse—uterine ischemia in the absence of an acute vascular event. We hypothesize that chronic traction on the uterine supports in the setting of complete procidentia may lead to kinking or stretching of the uterine arteries, compromising blood flow over time.

Uterine ischemia and necrosis are well documented complications of pelvic arterial embolization for post-partum hemorrhage and for treatment of symptomatic leiomyomas.[5,6] Rarer causes such as uterine torsion have also been described.[7] To the best of our knowledge there have been no previous reports of uterine ischemia caused by prolapse as a sole contributor.

Pelvic organ prolapse has been reported previously as a possible etiology for vaginal wall ischemia and necrosis. In one case report, Arabadzhieva et al describe a case of transvaginal intestinal evisceration in the presence of a long-standing uterine prolapse. The vagina was found to be distended and thickened in its upper third, with a defect in the posterior vaginal fornix, attributed to the prolonged prolapse. In this case, a hysterectomy with resection of the upper third of the vagina was performed.[8]

While our patient did not report any acute symptoms, the intraoperative findings suggest a prolonged ischemic process. Notably, this case occurred following rapid and significant weight loss, raising the possibility that loss of perivaginal and pelvic fat may have altered tissue support or vascular configuration, potentially exacerbating ischemia.

In a large study reviewing the natural history of pelvic organ prolapse following weight change, a borderline worsening of uterine prolapse was shown following a 10% weight loss.[9] Although this was a borderline finding that was observed during a five-year follow-up, perhaps this phenomenon was a contributing factor to worsening of uterine prolapse and the development of uterine ischemia in our patient.

Although the patient did not report acute symptoms, uterine ischemia may develop insidiously, especially in cases of mechanical compression or vascular stretching. This underscores the importance of intraoperative vigilance in patients with longstanding prolapse.

Further, the decision to forgo mesh placement was driven by both concern for the inflamed peritoneum's inability to integrate the mesh and the fragility of the remaining cervical tissue. This highlights the need for intraoperative flexibility in surgical planning and the value of reevaluation after initial surgical management.

CONCLUSION

In rare cases, chronic uterine prolapse may result in uterine ischemia, even without the presence of a structural anomaly or underlying malignancy. Longstanding procidentia can gradually lead to mechanical vascular compromise, which may be further exacerbated by significant and rapid weight loss that alters pelvic tissue support. These factors highlight the importance of careful preoperative assessment and vigilant intraoperative evaluation of tissue viability. Surgical planning in such complex cases should remain flexible, allowing for real-time adjustments based on intraoperative findings. Consideration should be given to the condition of uterine and peritoneal tissues when determining the appropriateness and timing of mesh-based repairs. In some instances, delayed reconstructive procedures may be a safer and more effective option following reassessment of tissue integrity.

Conflict of Interest : None
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