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Case report
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Postpartum Gynetresia

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Vaginal stenosis, or gynetresia, commonly results from a congenital defect, while acquired gynetresia is a rare condition. Reported contributors to acquired gynetresia include chronic graft-versus-host reaction, radiotherapy for gynecologic malignancies, female genital mutilation, postpartum foreign body insertion, or chemical insertion. We report a case of postpartum gynetresia, that was attributed to neither a foreign body nor chemicals. A 33-year-old Korean primigravid woman was referred to our hospital in hypovolemic shock due to postpartum bleeding caused by an actively bleeding vaginal laceration identified on pelvic examination. Primary repair of the vaginal laceration followed by embolization were performed. Four weeks later, postpartum gynetresia was identified on the pelvic examination. Adhesiolysis by blunt finger dissection was performed and a vaginal mold was inserted along with vaginal estrogen capsules. The vaginal mucosa was healed in four weeks.

Key Words: Postpartum period, Genitalia, Vagina, Constriction, Pathologic

Introduction

Acquired vaginal stenosis, or gynetresia, is a rare occurrence. In developed countries, it arises in association with chronic graft–versus–host reaction or radiotherapy treatment for gynecological malignancies. Acquired gynetresia is common in Africa, especially in Nigeria, with an incidence of 7:1,000. In this area, gynetresia is attributed to female genital mutilation, vaginal pessaries, birth injuries, and postpartum complications after vesicovaginal fistula repair. Some cases have been reported after exposure of the vaginal canal to chemicals such as rock salts, detergents used to tighten the vaginal mucosa, or postpartum infections from foreign bodies. Postpartum infections can lead to postpartum gynetresia, which is a rare complication. Herein, we present our experience with a patient who developed postpartum gynetresia unrelated to either infection or insertion of chemicals into the vaginal canal.

Case

A 33–year–old primigravid woman was referred to Gangnam Severance Hospital due to postpartum hemorrhage. She delivered a female baby vaginally in the primary care center and had no medico–surgical history. The baby's birthweight was 2,900 g. Postpartum hemorrhage occurred within two hours after the delivery and the estimated blood loss was approximately 2,000 to 3,000 mL prior to her transfer to our hospital. Upon arrival in the emergency room, she was in hypovolemic shock with active vaginal bleeding. A vaginal wall laceration with a perineal hematoma in the episiotomy site was identified. Fluid resuscitation and a norepinephrine infusion were initiated, followed by primary repair of the vaginal laceration. Approximately 2,000 mL of vaginal blood loss occurred during surgery. Subsequent embolization of the bilateral internal iliac arteries was performed due to conti-

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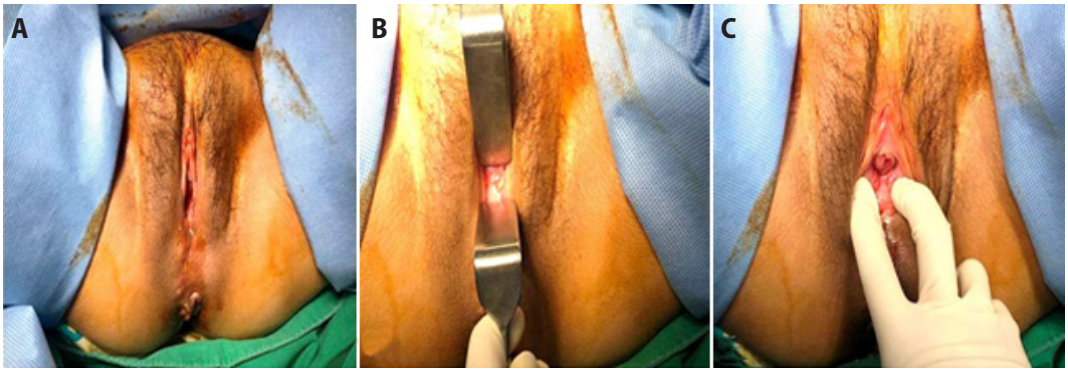

Fig. 1. A vaginal obstruction is identified on pelvic examination. (A) No specific findings are seen on the external genitalia. (B, C) The vaginal canal is completely obstructed at 10 mm from the vaginal orifice.

nued vaginal bleeding, suggestive of disseminated intravascular coagulation.

Initial blood pressure was 74/46 mmHg with 102 bpm of heart rate and initial blood hemoglobin, platelet count, and serum albumin were 6.2 g/dL, 59,000/μL, and 1.8 g/dL, respectively. Prothrombin time, activated Partial Thromboplastin Time, and D-dimer were 1.97 international normalized ratio (INR), 67.0 seconds, and 41.28 μg/mL and consumptive coagulopathy could not be ruled out. After transfusion of seven units of packed red blood cells (pRBCs) and six units of fresh frozen plasma during the operation, blood pressure was still 88/46 mmHg due to consumptive coagulopathy despite of fluid resuscitation and norepinephrine infusion. However, vital sign was getting stabilized after internal iliac arteries embolization and two units of additional pRBC transfusion; blood pressure and pulse rate were recovered to 128/78 mmHg, and 103 bpm, respectively. Norepinephrine infusion was tapered off in the day after embolization. In the second day of hospitalization, she received additional transfusion of two units of pRBC due to 6.1 g/dL of blood hemoglobin level. Platelet level was 53,000/μL in the next day of operation followed by spontaneous recovery to 89,000/μL in 2 days. She was discharged on postoperative day 3 and visited our outpatient clinic four weeks later. At that time, a pelvic examination revealed a complete obstruction of the vaginal canal at 10 mm from the vaginal orifice (Fig. 1). On a gynecological ultrasound, postpartum myometrial ischemic changes were observed (Fig. 2). Vaginal adhesiolysis was planned. A vaginal culture was obtained prior to cleansing the vaginal area with betadine. Vaginal adhesiolysis was performed using blunt finger dissection, and adhesions at 20 to 50 mm from the vaginal orifice were identified. The inner vaginal wall and uterine cervix were intact (Fig. 3). A fibrotic ring was dissected using electrocautery and then dilated using two fingers which were passed through the vaginal orifice. A vaginal mold made from a 30 mL disposable syringe covered with Vaseline gauze (Fig. 4) was inserted into the vaginal cavity. Vaginal mold was removed and reinserted daily with the estrogen suppositories for 6 weeks. The vaginal mucosa was healed in 6 weeks (Fig. 5).

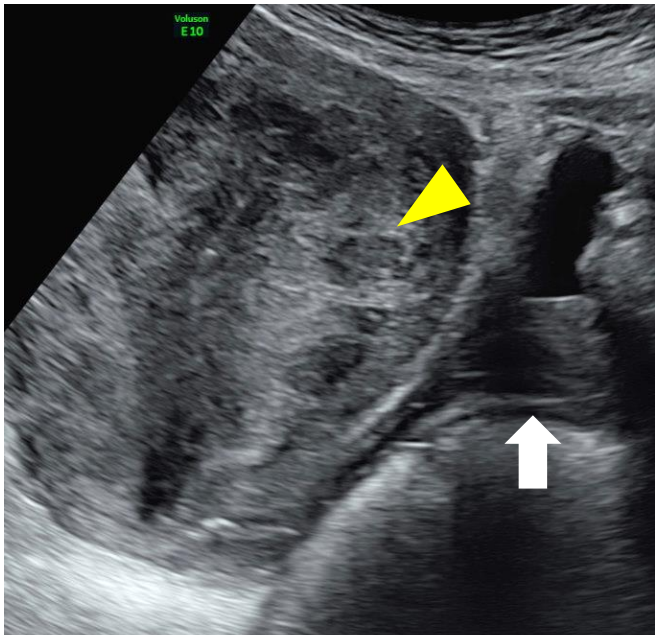

Fig. 2. A gynecologic ultrasound shows the gross anatomical configuration. Gynecological ultrasonography shows postpartum ischemic changes in the myometrium of the uterine body (yellow pointer) and a thin, but intact endometrium; however, the vaginal cavity cannot be identified because the uterine body has caused it to collapse (white arrow).

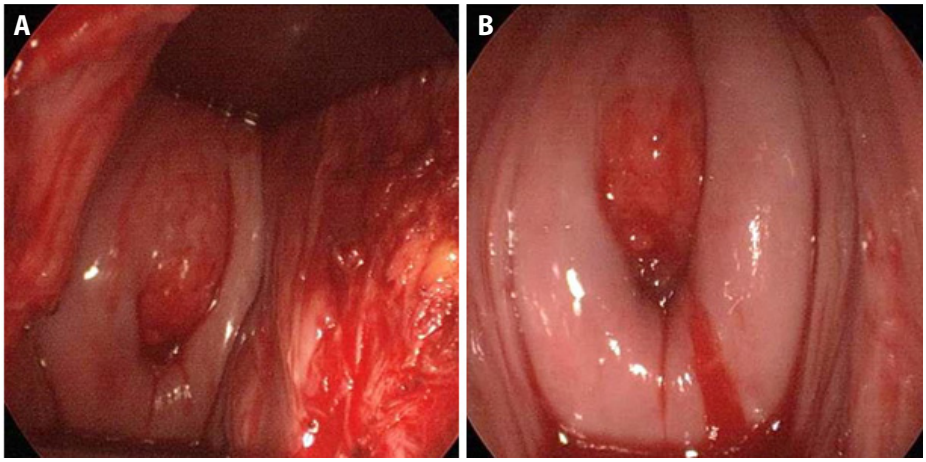

Fig. 3. An intact cervix and inner vaginal cavity are visible after adhesiolysis. (A, B) The inner vaginal canal and uterine cervix are intact, although an adherent lesion with an erosion is present.

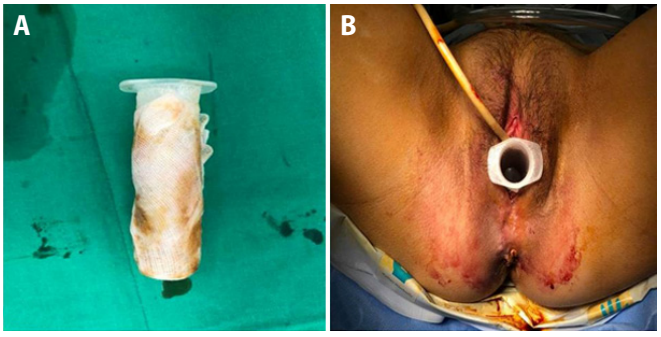

Fig. 4. A vaginal mold is inserted into the vaginal canal. (A) The temporary vaginal mold is crafted from a 30 mL syringe covered by Vaseline gauze. (B) The mold is inserted into the vaginal cavity.

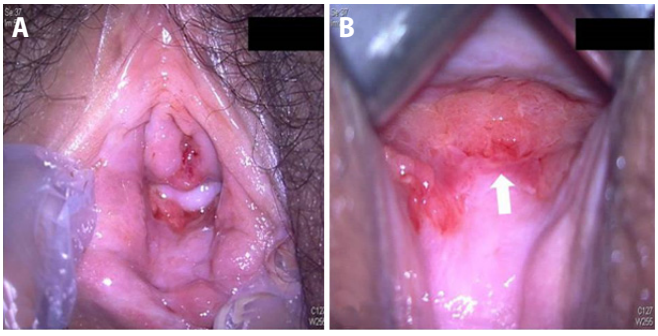

Fig. 5. A pelvic examination is performed at 4 weeks after the adhesiolysis. (A) The vaginal lumen shows no obstruction. (B) The vaginal mucosa shows an erosion (white arrow) with mild fibrotic changes in the posterior vaginal wall.

Discussion

Vaginal atresia is a condition in which the vagina is either abnormally closed or absent. Vaginal hypoplasia and vaginal obstruction are commonly associated with imperforate hymen and transverse vaginal septum. Acquired vaginal stenosis, or gynetresia, occurs frequently in African countries, especially in Nigeria, due to the practice of female genital mutilation. In other African countries and some rural areas in India, chemicals such as herbal products, caustic sodas, Dettol, and other substances may be inserted into the vaginal cavity to induce vaginal tightening and enhance sexual pleasure in postpartum women. The complications following vaginal insertion of chemicals may be acute or chronic in nature. Acute complications include burning, itching, abrasions, and fistula formation. Chronic extensive scaring causes fibrosis, stenosis, and obstruction/gynetresia. Howard and Lewicky-Gaupp[1] reported that the hypoestrogenic state during lactational amenorrhea caused vaginal agglutination. Vaginal stenosis can be attributed to sepsis in the postpartum period secondary to the retention of microbes[2] or foreign bodies in the vaginal cavity.[3] In our case, the patient was in an ischemic state due to hypovolemic shock and embolization. In addition, her norepinephrine infusion was continued for approximately 15 hours to manage the hypovolemic shock, which could have led to peripheral vasospasm and hypoxia. At 4 weeks after surgery, the fibrotic ring in her vaginal cavity had resolved. Fibrosis is an aberrant wound healing process wherein the repair of injured tissue is deregulated, causing myofibroblasts to deposit excessive amounts of extracellular matrix.[4] The excessive extracellular matrix results in an overgrowth of tissue with hardening and scar formation.[5] A characteristic of fibrotic tissue is reduced capillary density

within the tissue or organ, resulting in decreased oxygen delivery to cells under hypoxic conditions. Human fibroblasts exhibit pro-fibrotic features including activated transforming growth factor (TGF)-beta signaling, although the fibroblast was originated from cardiac tissue.[6] In brief, ischemic damage to the vaginal wall under hypoxic conditions could provoke adhesions and fibrotic scarring, which could have led to gynetresia in this patient. Hence, ischemia should be regarded as a contributor to postpartum gynetresia in patients with postpartum hemorrhage caused by vaginal wall or cervical lacerations.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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