

POSTPARTUM COLLAPSE CAUSED BY EXTENSIVE MESENTERIC VENOUS THROMBOSIS

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Early postpartum maternal shock is a grave obstetric emergency which calls for immediate attention if maternal mortality is to be prevented. Fortunately, in the majority of patients, such common causes of shock as postpartum hemorrhage, ruptured uterus and eclampsia are immediately evident. In the course of management of a patient presenting with severe postpartum shock, the authors recently encountered a most unusual cause: extensive mesenteric venous thrombosis.

Case Report

A 25-year-old Saudi woman, para 5, all normal-term deliveries, was referred to the emergency room of King Fahad Central Hospital (KFCH), Gizan, from a peripheral general hospital where she had had her fifth spontaneous vaginal delivery seven hours before presentation. She had had unsupervised antenatal care at home, and was admitted at term with vague abdominal pain, and after about 12 hours, went into active labor and delivered a healthy, average-sized male infant after two hours. Three hours later, she went into shock. After active resuscitation in the general hospital, she was transferred to KFCH. There was no history of any major obstetric complication except repeated blood transfusions for anemia in the second, third, and fourth pregnancies. She had no other significant medical or surgical history, and no history suggestive of thromboembolism. She had never used any oral contraceptives or other hormonal preparations.

On examination, the patient was semi-conscious, afebrile, markedly pale with unrecordable diastolic blood pressure, and a thready pulse of 128 per minute. The abdomen was moderately distended with generalized guarding and rebound tenderness. The uterus was of 18-weeks' gestational size and was firmly contracted. Pelvic examination revealed normal lochia, and a healthy cervix

with fullness in the Pouch of Douglas. A tentative diagnosis of postpartum shock caused by a ruptured uterus with possible broad ligament hematoma was made. She was immediately transferred to the intensive care unit and actively resuscitated with intravenous fluids, while central venous pressure monitoring was commenced via a right internal jugular cannula. Urgent investigations were conducted. Her blood group was O rhesus positive, hemoglobin genotype was AA, and hemoglobin was 10.5 g/dL. Apart from leucocytosis ($38,000/\text{mm}^3$) and elevated liver enzymes, other blood investigations including coagulation studies exhibited normal profiles. Urine culture yielded no growth of organisms. Abdominal ultrasound revealed the presence of free fluid in the peritoneal cavity. Crossmatched blood was made available and the patient was transferred to the operating room for exploratory laparotomy.

The abdomen was opened via a midline subumbilical incision, and approximately 1500 mL of exudate was found in the peritoneal cavity. The uterus, ovaries and fallopian tubes were normal. The broad ligaments were intact with no gross pathology. There was extensive gangrene of colon extending from the cecum down to the rectosigmoid junction, and hemorrhagic clots covering the colon and mesocolon. All the veins in the affected area were thrombosed. There was some urine smell emanating from the right renal area with no detectable gross renal tract pathology. She underwent subtotal colectomy and ileosigmoidostomy. The abdomen was rinsed with normal saline and a Redi-Vac drain was inserted to drain the right colic gutter.

Postoperatively, the patient had a total of 2000 mL of blood transfused, intravenous fluids to correct dehydration, and antibiotics. Intravenous heparin, 15,000 U every 12 hours, was commenced. After a week's treatment, this was tailed off to prophylactic warfarin. She had a short period of loose bowel motion which resolved spontaneously. The urine leakage dried up, and intravenous urography performed on the eighth postoperative day showed normal renal function. The patient was discharged 13 days post-surgery for follow-up at the surgical, medical, and gynecological clinics. Warfarin therapy (5 mg daily) was

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discontinued after three months' medication. She now has recurrent bouts of mild diarrhea which respond to symptomatic treatment.

Gross histopathology revealed a resected segment of bowel containing 120 mm of terminal ileum and 715 mm of large bowel. There were large and multiple areas which were dark red brown and necrotic. The mesenteric border was dark red. The wall of the bowel was thickened and dark grey red. The cecal wall had a similar appearance. The appendix could not be identified. Microscopy of several sections from the colon and cecum showed necrosis accompanied by edema, hemorrhage and neutrophilic exudation. The veins in the mesentery were dilated and filled with clotted blood. The mesentery also showed hemorrhage, necrosis, and neutrophilic exudation. Both surgical margins showed viable tissue. Microscopic diagnosis showed hemorrhagic necrosis of the colon and cecum, which is consistent with mesenteric venous thrombosis.

Discussion

Mesenteric vascular thrombosis in this patient affected mainly the drainage of the ileocolic, right colic, and middle colic venous tributaries of the superior mesenteric vein, and to some extent, the ascending branch of the left colic venous tributary of the inferior mesenteric vein. This may have been precipitated by local venous congestion and stasis,¹ but this does not explain the extent of pathological sequelae.

This case represents what can be considered one of the enigmas of obstetrics. Here was a moribund patient with all features pointing to intraperitoneal catastrophe for which a ruptured uterus was presumed, only to turn out to be a most unexpected surgical pathology. It is remarkable that the patient had no relevant history of thromboembolism or other cardiovascular risk factors. Nevertheless, the hypercoagulable state of pregnancy² should be seen as an important background etiological factor, notwithstanding the nonavailability of details of the patient's index obstetric history, especially the antenatal course. Apart from occurrence in the puerperium, a number of reports of mesenteric venous thrombosis during pregnancy have been recorded, with consequent additional risks to mother as well as to the fetus.²⁻⁵

Several articles document thrombotic events that can follow estrogenic stimulation. Appleberg in 1971 reported on mesenteric venous thrombosis following the use of stilbestrol to suppress lactation.⁶ Early thrombosis is considered a major risk in relation to in-vitro fertilization

programs as a result of the high endogenous plasma estrogen concentrations subsequent to ovarian stimulation.⁷ The use of estrogenic oral contraceptives (often inadvertently) during pregnancy, or their use in early puerperium, are known to precipitate mesenteric venous infarcts and massive colonic bleeding.^{2,4,8,9}

There was no evident precipitating factor, such as infection, dystocic labor, or assisted delivery to shed light on the cause of this patient's condition. This made the process of diagnosis more difficult, especially as she had a normal hemoglobin genotype (sickle-cell disease does give rise to mesenteric vascular infarcts and abdominal pain crisis). It is suggested that a patient with a clinical presentation of this type of acute abdomen, notably in the puerperium, should arouse a high index of suspicion, especially with the presence of other noted high-risk factors.¹⁰

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