

Thrombosis of Splanchnic Veins Following Splenectomy in a Factor V Leiden Heterozygote

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Citation

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Abstract

Most patients who develop thromboses of the splanchnic veins following splenectomies have underlying illnesses — most commonly, myeloproliferative disorders, hemolytic anemia or liver cirrhosis. Only rarely do splanchnic thromboses present in patients undergoing splenectomies because of traumatic rupture or inadvertent splenic injury during abdominal surgery. This case report presents a 45 year old female who thrombosed her splenic, superior mesenteric, left renal and portal veins post splenectomy for a benign mass even though there were no apparent pre-disposing patient characteristics that would have predicted these thrombotic complications. Upon closer examination, the patient was found to be a Factor V Leiden heterozygote which may have been the underlying genetic basis predisposing this patient to thrombose in the absence of any additional risk factors. The patient was successfully treated with heparin and coumadin anticoagulation. This case report suggests that patients who are heterozygous for Factor V Leiden may be at higher risk for splanchnic venous thrombosis following splenectomies.

CASE REPORT

A 45-year-old white woman presented to her physician with vague abdominal pain. She had a history of asthma, hypothyroidism, arthritis, borderline hypertension and irritable bowel syndrome. She had been on Questran™ for the past three years. Liver function tests were completed and found to be abnormal. A computed tomography (CT) scan indicated a 6 cm splenic mass. The abdominal ultrasound indicated a solid mass with no evidence of a cystic splenic mass. The liver was noted to be normal. A splenectomy was performed and the patient was discharged 4 days later without evidence of complications. The mass was benign.

Fourteen days later she presented to the emergency room with complaints of burning, stabbing, and epigastric abdominal pain. She also had complaints of nausea and vomiting. Her white blood count was 23.3, platelets 716, and liver function tests were normal. Urinalysis was positive for nitrites, ketones, white blood cells, red blood cells, many bacteria and 6-10 epithelial cells. She complained of tenderness in the epigastric area with some guarding but no rebound or distention.

An abnormal CT scan identified thromboses within the splenic, superior mesenteric, left renal and portal veins. The left renal vein was only partially thrombosed, but the remaining veins were completely thrombosed (Figure 1)

Figure 1

Figure 1: CT scan of abdomen showing (1) hepatic vein thrombosis and (2) superior mesenteric vein thrombosis forming (3) portal vein thrombosis.



A heparin drip was initiated. Coagulation studies were obtained; the patient was tested for factor V Leiden and found to be heterozygous.

The patient was discharged home in seven days on Coumadin™ therapy. A repeat CT scan a year later showed resolution of the thromboses and no further complications were noted.

DISCUSSION

The incidence of splanchnic vein thromboses after splenectomy may be higher than previously thought, since recent imaging studies have identified asymptomatic cases. Petit et al₂ found 13 of 119 patients who underwent ultrasound following splenectomy had splenic vein thromboses. Seven of the 13 were asymptomatic, while six were symptomatic. In another study, 60 splenectomies were done for hematological disorders₁. Four of these patients had portal vein thromboses diagnosed by Doppler color flow imaging. One was symptomatic but three were asymptomatic. In 1993, Gertsch et al₃ reviewed 64 cases of acute thromboses of the splanchnic veins which were published in 45 publications over the preceding 22 years. The mortality rate was as high as 76%, depending on the extent of venous obstruction.

The association between patient histories of venous thromboses and high prevalences of activated protein C resistance was first reported in 1994₄. Subsequent studies have shown that most of the resistance is caused by a single point mutation in the gene for coagulation factor V called factor V Leiden. This mutation occurs in the heterozygous state in about 3% to 15% of the general Caucasian population, thus making it the most common inherited risk factor for thrombosis₅.

Because we had no obvious a priori indications from our patient of the potential for thrombotic issues post-operatively, we further questioned our patient, seeking factors that might have alerted the surgeon to the potential for thrombotic complications. The patient denied any family history of clotting or bleeding disorders. Her past surgical history was significant for multiple reconstructive surgeries as an infant, child and young woman for a birth defect, but she denied coagulation or bleeding problems associated with those procedures. She had taken oral contraceptive pills for three years, but her physician asked her to discontinue birth control pills after she complained of "black-outs and ringing in her ears". Subsequently, she became pregnant 7 times with 4 miscarriages and all three live births were delivered via Caesarean sections. The patient indicated that she had never taken hormone replacement therapy.

Clearly the incidence of thromboembolic events in

individuals homozygous for the Factor V Leiden mutation is much higher than in heterozygous carriers (80 fold vs 7 fold)₆. These investigators surmised that since the absolute risk in homozygotes increases several percentage points per year, most homozygous carriers will have at least one thrombotic event in their lifetime although they may repeatedly experience risk situations without thromboses occurring. Our patient led a fully functional life as a Factor V Leiden heterozygote prior to the resection of her benign splenic mass. Only her history of multiple miscarriages hints at the possibility of a coagulation disorder. While we cannot definitively assign blame for the thrombotic complications in this case, our patient's heterozygosity provides a physiologic basis that offers a potential explanation for the complication when all other possibilities failed to do so.

LEARNING POINTS

- Asymptomatic splanchnic vein thromboses are not uncommon.
- Factor V Leiden is a common mutation that may lead to hypercoagulable complications.

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