Portal Vein Thrombosis: An Unusual Complication of Laparoscopic Cholecystectomy

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ABSTRACT

Background: Complications following laparoscopic cholecystectomy are encountered infrequently due to increasing proficiency in laparoscopic surgery. The occurrence of portal venous thrombosis following laparoscopic cholecystectomy has not been previously described and forms the basis of this report.

Methods: A healthy, 32-year-old, female on oral contraceptives underwent an uneventful laparoscopic cholecystectomy for symptomatic gallbladder disease. Sequential compression devices and mini-dose unfractionated heparin were used before the procedure. The patient was discharged home on the first postoperative day without complaints. She returned 1 week later with nausea, bloating, and diffuse abdominal pain.

Results: Ultrasonography of the abdomen revealed thrombosis of the portal vein not seen in the preoperative ultrasound and the superior mesenteric vein. Computer tomography of the abdomen and pelvis on the same day confirmed this finding and showed a wedge-shaped infarction of the right lobe of the liver. The patient was anticoagulated with intravenous heparin. An extensive coagulation workup revealed elevation of the Immunoglobulin G anticardiolipin antibody. A percutaneous transhepatic portal vein thrombectomy was performed. A postprocedure duplex ultrasound of the abdomen demonstrated recannalization of the portal venous system with no flow voids. Anticoagulation therapy was continued, and the patient was discharged home with resolution of her ileus. She was maintained on a therapeutic dose of warfarin.

Conclusions: This case demonstrates an unusual complication of laparoscopic cholecystectomy. It may have resulted from the use of oral contraceptives, elevation of the Immunoglobulin G anticardiolipin antibody, unrecognized trauma, and was accentuated by the pneumoperitoneum generated for the performance of the laparoscopic cholecystectomy. Our case report provides insight and poses questions regarding necessary perioperative measures for thromboprophylaxis in young females on oral contraceptives undergoing elective laparoscopic abdominal surgery.

Key Words: Laparoscopy, Cholecystectomy, Portal vein thrombosis.

INTRODUCTION

Complications following laparoscopic cholecystectomy can be due to laparoscopy itself, or the cholecystectomy. Hemorrhage, bile leak, and injury to a major bile duct are complications related to removal of the gallbladder. Laparoscopic complications occur secondary to carbon dioxide pneumoperitoneum or the instruments inserted through the abdominal wall. The complications of the pneumoperitoneum, such as gas embolism, vagal reaction, ventricular arrhythmias, and hypercarbia with acidosis, are well known. The occurrence of portal venous thrombosis following laparoscopic cholecystectomy has not been previously reported and forms the basis of this report.

CASE REPORT

A healthy, 32-year-old female presented with a history of intermittent right upper quadrant pain for a few months. Physical examination was significant only for some mild abdominal tenderness over the right upper quadrant. The laboratory workup was normal. Past medical and surgical history was unremarkable except for the fact that the patient was on oral contraceptives. A diagnostic abdominal ultrasound study was performed that revealed multiple small polyps in the gallbladder, as well as a patent portal vein system. The patient underwent an uneventful
laparoscopic cholecystectomy. Sequential compression devices and mini-dose unfractionated heparin were used for prophylaxis of deep vein thrombosis because the patient was on oral contraceptives. Pneumoperitoneum was established by the open technique to a pressure of 15 mm Hg. The patient was placed in the reverse Trendelenburg position. Electrocautery was used only to detach the gallbladder from the liver bed.

The patient was discharged to home on the first postoperative day without complaints and on a regular diet. A few days later, the patient went to the emergency room with vague abdominal pain. The ultrasound at that point was normal, and the patient went home the same day. She returned 1 week later with complaints of diffuse abdominal pain, bloating, and nausea. Ultrasonography of the abdomen revealed a rounded area of echogenicity at the bifurcation of the splenic vein and superior mesenteric vein that extended along the course of the portal vein and the superior mesenteric vein. This finding was consistent with thrombosis of these vessels (Figure 1). Computed tomography of the abdomen and pelvis with intravenous and oral contrast confirmed the findings and in addition demonstrated a wedge-shaped zone of decreased attenuation involving the lateral aspect of the right lobe of the liver compatible with liver infarction (Figure 2). A mesenteric angiogram was performed and confirmed the diagnosis. Coagulation studies including prothrombin time, partial thromboplastin time, platelet count, protein C & S, antithrombin III, lupus anticoagulant, and platelet aggregation studies were normal. Anticardiolipin antibody Immunoglobulin G (IgG) was elevated to 19 g-phospholipid level (GPL) units (normal range, 0 to 10), with negative IgM and IgA. The patient was immediately started on intravenous heparin anticoagulation, with improvement of her symptoms. A percutaneous transhepatic portal vein thrombectomy was performed with a postprocedure duplex abdominal ultrasound that demonstrated recannalization of the portal venous system with no flow voids. Anticoagulation was continued, and the patient was discharged home on a regular diet and therapeutic doses of oral warfarin. The patient has since been asymptomatic for 2 years.

**DISCUSSION**

The practice of laparoscopic cholecystectomy has increased rapidly over the last 14 years and has rapidly become the standard of care for the management of symptomatic gallbladder disease. Safety, efficacy, and patient benefits have been established. Complications following laparoscopic cholecystectomy are infrequent and include major bile duct injury, major vascular injury during trocar placement, cardiopulmonary failure, wound infections, and spillage of stones into the peritoneal cavity with subsequent abscess and or fistula formation. To our knowledge, the occurrence of isolated portal vein thrombosis and superior mesenteric vein thrombosis following laparoscopic cholecystectomy has not been previously described and forms the basis of this discussion. Portal vein thrombosis as a rare complication of laparoscopic splenectomy in patients with idiopathic thrombocytopenia was previously reported. Several factors could have contributed to portal vein thrombosis. Decreased splanchnic flow with insufflation of CO₂ has been shown.2,3 Hy-
percavaria produces vasodilation that can offset the reduced mesenteric flow from increased intraperitoneal pressure secondary to CO₂ insufflation. Hepatic microcirculation, portal venous blood flow, and renal blood flow are decreased with increased intraabdominal pressure as documented in experimental as well as in clinical studies. Takagi et al. showed that the diameter of the portal venous trunk and the mean portal blood flow was significantly decreased with an elevation of intraperitoneal pressure (IP) >10 mm Hg. The flow was less than one tenth of the baseline value when the IP was 16 mm Hg. Jakimowitcz et al. demonstrated a stepwise reduction of portal blood flow from 990 (100 mL/min) to 568 (81 mL/min) at 7 mm Hg of IP, compared with baseline flow measured at 0 mm Hg. In the same study, at an IP level of 14 mm Hg, which was used routinely for all laparoscopic procedures, the portal venous flow sustained a further drop to 440 (56 mL/min), corresponding to a 53% reduction in the portal venous flow compared with the initial baseline value. Junghans et al. in experimental studies, reported that the head up position in association with an elevation of IP greater than 12 mm Hg should be avoided during laparoscopic surgery because they compromise hepatic and renal blood flow. Infrarenal inferior vena caval flow was also been shown in experimental reports to decrease during insufflation with CO₂. The above-mentioned studies confirm the reduction of portal venous flow and also a relationship between the drop in portal venous flow and the increase in intraabdominal pressure.

With regard to the above observations, a selective setting of IP during laparoscopic surgery is advocated with low pressure or gasless abdominal wall lift approaches. In our case, other factors may have accentuated the effects of the pneumoperitoneum. For example, the presence of a preexisting portal vein thrombosis or trauma occurring during the procedure, even though we found no evidence of this. The use of oral contraceptives has been established as an independent risk factor for the development of deep vein thrombosis and approximately one quarter of idiopathic events among women of childbearing age have been attributed to oral contraceptives. The risk is increased when oral contraceptive use is combined with surgery and inherited inhibitor deficiencies. An alteration in the coagulation system with an increase in blood viscosity, fibrinogen, plasma levels of factors VII and X, and platelet adhesion and aggregation may contribute to this thrombotic tendency. No association with portal vein thrombosis has been reported. Elevated levels of cardiolipin antibody (IgG) with normal IgM and IgA also predisposes to portal vein thrombosis. The significance of an isolated, mild elevation of IgG in a patient is not clear. The presence of antiphospholipid antibodies predisposes to both arterial and venous thrombosis. Portal vein thrombosis was reported in one patient from a series of 800 in which the level of elevation of IgG, the partial thromboplastin time, the value of dilute Russell viper venom (d RVV), as well as the association with IgM was not very clear.

The diagnosis of portal vein thrombosis can be accurately established with an abdominal duplex scan or computed tomography. If their results are equivocal, then arterial portography may be performed. Treatment options in addition to anticoagulation therapy include open portomesenteric venous thrombectomy or percutaneous transhepatic procedures, including thrombectomy, infusion of fibrinolytic agents, balloon dilation, and stenting. The optimal duration of anticoagulation is unknown. For those with primary coagulation disorders, lifelong anticoagulation is necessary. As most recurrent thromboses occur within 6 weeks of portomesenteric thrombosis in patients with treatable secondary causes of the portal venous thrombosis, removal of the risk factor and a 6-month course of anticoagulation should be adequate.

CONCLUSION
The perioperative measures for thromboprophylaxis used in young, otherwise healthy females taking oral contraceptives and undergoing laparoscopic abdominal surgery needs to be reevaluated. The above case poses more questions than answers that the medical community needs to address to avoid such complications in the future.

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