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1

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Portal vein thrombosis in noncirrhosis patients after hemihepatectomy: A case report and review of literature

Zhang SB *et al.* Portal vein thrombosis after hemihepatectomy

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Abstract

BACKGROUND

Portal vein thrombosis (PVT) is a lesion caused by hemodynamic disorders. It may be noted in the portal vein system when there is an inflammatory stimulus in the abdominal cavity. However, PVT is rarely reported after hepatectomy. At present, related guidelines and major expert opinions tend to consider vitamin K antagonists (VKAs) or low-molecular weight heparin (LMWH) as standard treatment. But based on research, direct oral anticoagulants may be more effective and safe for noncirrhotic PVT and are also beneficial by reducing the recurrence rate of PVT.

CASE SUMMARY

A 51-year-old woman without any history of disease felt discomfort in her right upper abdomen for 20 d, with worsening for 7 d. Contrast-enhanced computed tomography (CECT) of the upper abdomen showed right liver intrahepatic cholangiocarcinoma with multiple intrahepatic metastases but not to the left liver. Therefore, she underwent right hepatic and caudate lobectomy. One week after surgery, the patient underwent a CECT scan, due to nausea, vomiting and abdominal distension. Thrombosis in the left branch and main trunk of the portal vein and near the confluence of the splenic vein was found. After using LMWH for 22 d, CECT showed no filling defect in the portal vein system.

CONCLUSION

Although PVT after hepatectomy is rare, it needs to be prevented during the perioperative period.

Key Words: Portal vein thrombosis, Hemihepatectomy; Anticoagulation; Noncirrhosis; Low-molecular weight heparin; Case report

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Core Tip: We present a case of portal vein thrombosis (PVT) after hemihepatectomy. It is most common in liver cirrhosis, postsplenectomy and liver transplantation, but is relatively rare after hepatectomy. This case verified the safety and effectiveness of low-molecular weight heparin. In the perioperative period, attention should be given to the prevention, early diagnosis and systemic treatment of PVT.

INTRODUCTION

Portal vein thrombosis (PVT) is often detected in the extrahepatic portal vein, such as the superior mentraic vein and spleen vein. Vitamin K antagonists (VKAs) or low-molecular weight heparin (LMWH) are considered as the standard treatment^[1,2]. But direct oral anticoagulants (DOACs) may be more effective and safe for noncirrhotic PVT (ncPVT) and are also beneficial by reducing the recurrence rate of PVT^[3,4]. Portal vein tumor thrombosis is significantly different from PVT^[5,6]. Studies show that 2.1%-9.1%^[7,8] patients have PVT after hepatectomy, with a rate of 10%-40%^[9-11] among patients with hepatocellular carcinoma. Anticoagulation is widely accepted as the standard treatment option for ncPVT^[12-14]. According to the Child-Pugh classification principle, thrombectomy and interventional therapy may have great disadvantages.

CASE PRESENTATION

Chief complaints

Discomfort in the right upper abdomen for 20 d with worsening for 7 d.

History of present illness

The symptoms were unrelated to feeding behavior, and she had not undergone any treatment.

1

History of past illness

The patient was in good health.

Personal and family history

The patient had no medical history or family history of malignant tumors.

Physical examination

The physical examinations were toughly normal.

Laboratory examinations

CA-199 was over 1000 U/mL on admission.

Imaging examinations

Contrast-enhanced computed tomography (CECT) of the upper abdomen showed right liver intrahepatic cholangiocarcinoma (ICC) with multiple intrahepatic metastases, which were confined to the right liver and caudate lobe.

FINAL DIAGNOSIS

ICC with multiple intrahepatic metastases in the right liver and caudate lobe

TREATMENT

After three-dimensional reconstruction of the liver, we found that the middle hepatic vein (Figure 1) and the right branch of the portal vein (Figure 2) were located close to the tumor. Therefore, we decided to perform laparoscopic left-liver-first anterior radical modular orthotopic right hemihepatectomy (Lap-larmorh) (Figure 3). The right liver and caudate lobe, which accounted for 62% of the liver volume, were resected. After the operation, in addition to antibiotics, hepatic protectants and proton pump inhibitors, we also intermittently administered fibrinogen, human prothrombin complex concentrates

and ordinary frozen plasma to avoid hemorrhage from the liver wounds. However, one week after surgery, nausea, vomiting and abdominal distension occurred, and she underwent a CECT scan. Thrombosis was distributed in the left branch and main trunk of the portal vein and near the confluence of the splenic vein. Moreover, the main trunk was almost occluded (Figure 4A). To prevent further progression, we began to inject 6400 IU LMWH twice a day until the PVT disappeared and stopped procoagulant therapy. After the above treatments, the patient's discomfort was significantly relieved. In addition, CECT and color Doppler ultrasound indicated that the thrombosis was smaller than before (Figure 4B and C). On day 22, CECT showed that there was no filling defect in the portal vein (Figure 4D).

OUTCOME AND FOLLOW-UP

This patient was discharged uneventfully one month after the operation. Nine months of follow-up by telephone showed that there were no recurrent thromboembolic events without anticoagulation therapy. Moreover, there were no symptoms of digestive system discomfort.

DISCUSSION

As a deep vascular complication, PVT is not common in clinical work. It usually occurs in patients with liver cirrhosis, with an incidence of 10-25%^[15-17]. The incidence is closely related to surgery, ranging from 1% to 3%^[18] after liver transplantation, and from 1.6 to 11%^[19,20] after splenectomy. At present, our department does not provide preventive treatment for thrombosis during the perioperative period. Anticoagulation therapy has no significant effect on the incidence of thrombosis^[21].

According to the location of the thrombus, the degree of obstruction and the speed of obstruction formation, PVT can be divided into the acute phase, subacute phase and chronic phase. At the onset of the acute phase, the patient will suddenly show symptoms such as nausea, vomiting, abdominal pain, or fever. After hepatectomy, without undergoing portal vein color Doppler ultrasound and CECT, it is difficult to

associate these symptoms with acute PVT and make an early diagnosis or start treatment early. If the portal vein is not completely blocked in the acute phase, these symptoms will continue for several weeks and progress to the subacute phase, eventually entering the chronic phase after the formation of venous collateral circulation. The patient gradually developed gastrointestinal symptoms 1 wk after right hepatectomy, which proved that the PVT did not suddenly block most of the main portal vein, which created the possibility for subsequent treatment.

The three basic risk factors for the formation of PVT are vascular endothelial cell damage, blood hypercoagulability and portal vein blood flow disorder. Reviewing the patient's perioperative treatment, there were the following risk factors that may have led to PVT: (1) Surgical factors, such as intraoperative laparoscopic equipment pulling the portal vein and damaging the vascular endothelial cells, 20 min of treatment with the first hepatic portal blood flow occlusion method causing prolonged hypoxia and high pneumoperitoneal pressure (15 mmHg)^[22,23] causing a reduction in portal blood flow velocity; and (2) After the operation, the patient's effective circulating blood volume was insufficient, and the blood hypercoagulable state was caused by an inflammatory reaction. In addition, an insufficient remaining liver volume after hepatectomy resulted in a fragile balance of the coagulation system. Frozen plasma and other procoagulant drugs infused to prevent hemorrhage from the wound may also have promoted the development of PVT.

At present, the early diagnosis of PVT is based on clinical symptoms and the detection of thrombi through imaging examination. However, approximately 67%^[24] of patients have no special clinical manifestations after the formation of PVT. CECT is still the first choice for a clear diagnosis^[21,25]. The diagnosis of this patient was achieved through CECT, and in the follow-up process, the combination of CECT and ultrasound also achieved good results.

The purpose of PVT treatment is to prevent the further development of the thrombus and recanalization of blood vessels. Generally, there are three treatment methods. First, thrombectomy can immediately relieve blood vessel obstruction and prevent acute

intestinal necrosis or related complications. However, new portal vein injuries and other surgical trauma often lead to the recurrence of PVT. In particular, patients cannot withstand the traumatic stimulation of reoperation. Therefore, this method is only suitable for patients where there are signs of peritonitis or where the thrombus has been confirmed to come from the portal venous system. Second, interventional therapy can accurately deliver thrombolytic drugs to the obstruction with less damage and fewer complications. In addition, it provides an option for patients in whom liver function is decompensated, the PVT lasts for a long time and conservative treatment was not effective. Although nonsurgical treatment is minimally invasive, effective and reproducible, its risks still require our attention. For example, percutaneous portal vein thrombolysis can induce bleeding in the puncture tract. During the process of thrombolysis, it is necessary to monitor coagulation function and blood cell count over time. Therefore, it is contraindicated in patients with Child-Pugh class C who have large amounts of ascites and poor coagulation. Finally, anticoagulation and thrombolysis are the basic principles of noninvasive treatment^[26]. Patients with primary blood hypercoagulability and early thrombosis will benefit more from anticoagulation therapy. Eighty percent of acute PVT can be completely or partially recanalized after anticoagulation therapy^[27]. Compared with heparin, LMWH is the best anticoagulant. Because there is no obvious difference between the two anticoagulant drugs in terms of causing bleeding, LMWH can better improve the survival rate^[25,28]. Studies have shown that LMWH is safe and effective in the treatment of PVT caused by liver cirrhosis^[29], and changes in thrombus volume can be seen on imaging after 2 wk of anticoagulation therapy^[28]. This is also consistent with the therapeutic effect of this patient after hepatectomy. If anticoagulation is not continued after recanalization, approximately 38%^[30] of patients will develop PVT again. Some experts suggest that after 20 d of intravenous infusion of anticoagulant, it is best for patients to continue oral anticoagulant for 3 to 6 mo instead of subcutaneous LMWH^[19,21]. Because long-term subcutaneous injection of LMWH after discharge can lead to decreased patient compliance, and standard treatment represented by VKAs and LMWH require

continuous monitoring of renal function and INR to adjust the dosage^[31]. However, related foundational trials included no patients with PVT^[32-35]. DOACs for the treatment of PVT are still not widely accepted, and further research is needed^[36].

In addition to the treatment of PVT and prevention of recurrence mentioned above, prevention during the perioperative period is crucial. First, adequate portal blood flow should be ensured. Ultrasound can be used to measure portal vein blood flow, and less than 15 cm/s is an important risk factor^[37]; Second, damage to the vascular intima should be avoided, for example, preventing violent pulling of the portal vein system during surgery, and reducing the time and frequency of portal occlusion. Finally, if symptoms such as nausea and vomiting occur after surgery, it is necessary to promptly confirm whether PVT occurred^[21].

CONCLUSION

PVT is common in patients with liver cirrhosis or after liver transplantation and splenectomy. Surgeons should improve their understanding of this complication and use color Doppler ultrasound and CECT to confirm the appearance of PVT in a timely manner.

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