Repeated variceal bleeding in a patient with advanced urothelial cell carcinoma

Yu-Guang Chen¹, Wei-Chou Chang², Te-Pao Lin², Ping-Ying Chang¹, Ming-Shen Dai¹

¹ Division of Hematology/Oncology, Department of Internal Medicine, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan
² Department of Radiology, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan

A 54-year-old woman with uremia who was undergoing maintenance hemodialysis was hospitalized because of a 3-month history of intermittent hematochezia. She was also currently undergoing palliative chemotherapy for advanced renal urothelial cell carcinoma, which was diagnosed 5 months earlier. One month before hospital admission, she presented to another hospital with intermittent mild hematemesis. A gastroscopy revealed varices in the lower esophagus and red wale markings. An ultrasound revealed hepatosplenomegaly but no evidence of a hepatic space lesion, liver cirrhosis, or portal vein thrombosis. On serial workup, markers of viral hepatitis, serological markers of autoimmune and metabolic diseases, and markers of alcoholic and nonalcoholic steatohepatitis were negative. Thereafter, the patient received a conservative treatment for suspected idiopathic portal hypertension. After 2 weeks, she experienced massive hematemesis of approximately 400 ml of fresh blood. On her arrival at the emergency room, we observed the following vital signs: body temperature, 36.7°C; pulse rate, 125 bpm; respiratory rate, 22 breaths/min; and blood pressure, 69/30 mmHg. A physical examination revealed dry skin turgor, mild ascites, and bilateral edema of the lower extremities, with a dilated engorged abdominal superficial vein. Her hemoglobin level was very low (4.6 g/dl), and other serial laboratory test results were unremarkable. A Sengstaken–Blakemore (SB) tube was inserted to stop variceal bleeding. Contrast-enhanced abdominal computed tomography (CT) angiography revealed hepatosplenic megaly, diffused paraaortic and paracaval lymphadenopathy, and compression of the inferior vena cava (IVC) by a large necrotic node (FIGURE 1A). Subsequently, the patient received supportive care with fluid and salt restriction for chronic IVC syndrome. A right anterior oblique 3-dimensional reformatted image obtained by direct CT angiography and venography showed engorged portal and splenic veins with multiple collateral branches between the IVC and portal veins (FIGURE 1B). The findings indicated the recurrent variceal hemorrhage caused by chronic IVC syndrome with caval-portal circulation related to noncirrhotic portal hypertension (NCPH; FIGURE 1C). Bleeding ceased gradually with conservative management, tight salt and fluid intake restrictions, and supportive care. Thereafter, the SB tube was removed. However, the patient died of cancer 3 weeks later.

Generally, the major causes of NCPH include vascular lesions, schistosomiasis, regenerative nodule hyperplasia, idiopathic portal hypertension, and chronic human immunodeficiency virus infection.¹,² The most frequent vascular causes are posthepatic venous outflow obstruction or intrahepatic portal/splenic venous obstruction.³ However, infrarenal IVC syndrome-related retrograde caval-portal circulation is a rare cause of NCPH. In patients with advanced malignancies, IVC syndrome caused by regional lymphadenopathy is a common manifestation. The symptoms of malignant IVC syndrome vary from no symptoms to lower leg edema, refractory ascites, and even acute distal thrombosis. However, our patient underwent long-term hemodialysis and had a relatively decreased systemic blood flow compared with the general population. The common manifestations mentioned above were usually unapparent in our patient. In comparison with the common manifestations of IVC syndrome, the life-threatening complications of recurrent variceal hemorrhaging are a rare presentation and have been rarely reported.
FIGURE 1 A – a coronal view of an abdominal contrast-enhanced computed tomography scan showing a large metastatic lymph node (arrow) over the paracaval space with inferior vena cava compression and massive splenic infarctions; B – a 3-dimensional volume-rendered computed tomography scan showing the multiple caval-portal collateral pathways exhibited after severe inferior vena cava obstruction; the arrowhead denotes the caval-superficial-umbilical-portal pathway; the arrow, the caval-inferior mesenteric portal pathway; and the asterisk, the caval-retroperitoneal-portal pathway; C – a schematic representative diagram showing the altered blood flow after inferior vena cava obstruction, and the splenic infarction could be considered the cause of the turbulent flow via the inferior mesenteric vein

Abbreviations: A, descending aorta; ALV, ascending lumbar vein; DV, duodenal vein; HVP, hemorrhoidal venous plexus; IMV, inferior mesenteric vein; L, liver; PV, portal vein; PU, paraumbilical vein; SEV, superficial epigastric vein; SMV, superior mesenteric vein; SP, spleen; SV, splenic vein; UA, umbilical anastomosis; V, inferior vena cava

REFERENCES