Successful Hepatectomy Using Venovenous Bypass in a Patient With Carcinoid Heart Disease and Severe Tricuspid Regurgitation

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GASTROINTESTINAL NEUROENDOCRINE tumor (GINET) is an uncommon, slow-growing tumor arising from neuroendocrine cells. Its incidence, about 2.5 to 5 cases per 100,000, has been increasing up to 10% per year.¹ Its ability to synthesize and secrete active peptides and other neuroamines can lead to carcinoid syndrome, which may manifest as cutaneous flushing, gastrointestinal (GI) hypermobility, and bronchospasm. Carcinoid heart disease is a process of endomyocardial fibroelastosis usually affecting the right heart valves (tricuspid and pulmonic) with involvement of subvalvular apparatus. The degeneration of one or both right heart valves leads to dilation of the right heart chambers and right ventricular (RV) dysfunction, and the resulting increase in right-sided pressures and liver congestion can result in significant hemorrhage during liver resection.

The authors present the successful management of right hepatectomy using venovenous bypass in a patient with severe tricuspid regurgitation and moderate pulmonic regurgitation due to carcinoid heart disease. They also present a review of the literature regarding the appropriate sequence of surgeries in this unique situation.

CASE REPORT

A 54-year-old woman (height: 163 cm, weight: 79 kg) diagnosed with GINET and liver metastases as well as RV dysfunction from carcinoid heart disease was scheduled for right hepatectomy. One year before, she had undergone an octreotide scan and cross-sectional imaging and received a diagnosis of carcinoid tumor originating at the ileocecal valve. The lesion was suspected to be in the terminal ileum, but the patient had not undergone excision of the primary lesion at that time.

She presented with occasional flushing and shortness of breath. She was a former smoker with a 15-pack-year smoking history. She had quit 15 years earlier and denied having any drug or food allergies. Transthoracic echocardiogram revealed a significantly dilated right ventricle, severe tricuspid regurgitation (TR), mild pulmonary stenosis (PS), and moderate

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pulmonary regurgitation (PR). The patient had undergone chemoembolization of the hepatic artery 3 months before surgery, but her symptoms had remained unchanged. Her medications were furosemide, slow-release potassium chloride, and octreotide acetate for injectable suspension (60 mg subcutaneously every 21 days).

On physical examination, she was not in acute distress. She had stable vital signs, no peripheral edema, and a pan-systolic murmur best heard over the right parasternal area. Her airway assessment was normal. She had a normal complete blood count and electrolyte levels. Her international normalized ratio (INR) was 1.2. Partial thromboplastin time was 22 seconds. Liver and kidney function tests were unremarkable. Chest x-ray and electrocardiogram were within normal limits. Preoperative urinary 5-hydroxyindoleacetic acid (5-HIAA) ranged from 7.7 to 11.06 mg/24 h.

Because the patient had significant cardiac involvement in the form of severe TR and RV dilatation, multidisciplinary team meetings involving cardiac surgery, hepatobiliary surgery, and anesthesiology were held. Discussions mainly concerned the appropriate sequence of the 2 required surgeries, hepatectomy and tricuspid valve replacement. The cardiac surgeons believed the carcinoid disease would affect the new bioprosthesis and were, therefore, reluctant to replace the tricuspid valve first. After much discussion, the team decided to proceed with hepatectomy first. It was decided that venovenous bypass (VVB) would be performed to prevent excessive hepatic congestion and blood loss from high right-sided cardiac pressure because of the planned total vascular exclusion. The risks and benefits of the surgery were discussed extensively with the patient, and she provided written informed consent.

In the operating room, after attaching the standard American Society of Anesthesiologists-recommended monitors (which were the same as those recommended by the Canadian Anesthesiologists' Society) and before the induction of anesthesia, an arterial line was inserted. Anesthesia was induced with midazolam, fentanyl, propofol, and rocuronium. After uneventful induction and intubation, octreotide, 1,600 µg (20 µg/kg), was injected subcutaneously. Anesthesia was maintained with fentanyl, sevoflurane, and rocuronium. Bispectral index, esophageal temperature probe, and neuromuscular monitoring also were used. An introducer sheath and a venous bypass cannula were inserted under ultrasound guidance through the right internal jugular vein under strict aseptic conditions. Intravenous (IV) heparin infusion was started, keeping the activated coagulation time around 200 seconds to prevent clotting of the VVB cannula.³⁶ A transesophageal echocardiography (TEE) probe was introduced without complication, and a comprehensive TEE examination, performed by a board-certified echocardiographer, confirmed the preoperative findings of severe RV dilatation (Fig. 1) with

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Fig 1. Intraoperative transesophageal echocardiography showing severe right ventricle dilatation.

mild RV dysfunction, severe TR (Figs. 2 and 3), mild PS (Figs. 4 and 5), moderate PR (Fig. 4), and mild pulmonary hypertension (Figs. 1–5).

The patient was stable during the first 5 hours as the surgeons mobilized and identified the major vessels of the liver. Some difficulties were encountered because of adhesions from previous cholecystectomy. Baseline central venous pressure (CVP) was 28 mmHg and was maintained between 16 and 25 mmHg. Mean arterial pressure (MAP) was maintained at around 60 to 70 mmHg by administering crystalloid solution and a small amount of colloid (5% albumin). Hourly arterial blood gas analyses were performed to monitor blood electrolytes, lactate, and hemoglobin concentrations. A Pringle maneuver (clamping of hepatic artery and portal vein at the level of the hepatoduodenal ligament) was performed, followed by trial clamping of the inferior vena cava (IVC).

Within 1 minute of IVC clamping, there was a precipitous drop in blood pressure to an MAP of 30 mmHg and a CVP of 6 mmHg, requiring release of the clamps. A 19-Fr venous cannula was introduced from the left femoral vein into the IVC. VVB was initiated and flow rate was >2 L/min. Even with VVB, MAP dropped to 40 to 50 mmHg after total vascular exclusion, but was manageable with IV infusions of norepinephrine at 3-to-5 μ g/min and octreotide, 100 μ g/h. Hemoglobin concentration was 8.2 g/dL; however, in view of the low blood pressure, blood loss of about 1,000 mL, and



Fig 3. Intraoperative transesophageal echocardiography. Hepatic vein flow depicting severe tricuspid regurgitation (systolic reversal flow).

more blood loss expected, packed red blood cells (RBCs) were transfused.

Approximately 90% of the entire tumor mass was successfully resected (Fig. 6). Despite total vascular exclusion, there was still significant blood loss during hepatic parenchymal transection (about 3,000 mL in 1 hour), so transfusion of packed RBCs was continued. Because point-ofcare tests of coagulation such as thromboelastography and rotational thromboelastometry were not available, fresh frozen plasma (FFP) was transfused based on an INR of 1.5. Immediately after the extended right hepatectomy (segments IV-VIII), the patient's blood pressure dropped to 50 mmHg systolic in the absence of further significant blood loss. Release of the vascular clamp increased the CVP to 15 mmHg but did not improve the blood pressure. TEE did not reveal any new abnormal changes at this stage, and carcinoid crisis was suspected. Epinephrine infusion at 2 µg/min was started, along with 2 IV boluses of octreotide, 100 µg each, and another 100 µg injected subcutaneously.

About 20 minutes after hepatectomy, blood pressure started to rise and remained stable thereafter. Epinephrine infusion was discontinued, but infusions of norepinephrine, 5 µg/min, and



Fig 2. Intraoperative transesophageal echocardiography showing severe tricuspid regurgitation.



Fig 4. Intraoperative transesophageal echocardiography showing mild pulmonary artery stenosis and moderate pulmonary regurgitation.

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