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Management of Biventricular Assist Device Implantation in Patients with Necrotic Pancreatitis

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ABSTRACT

This report describes the management of biventricular assist device (BIVAD) implantation in a patient with necrotic pancreatitis. BIVADs provide mechanical support for ventricular ejection in the failing heart and have become an accepted treatment for end-stage heart failure. They also have proved to be a successful bridge to heart transplantation. As their popularity has grown, the number of patients with BIVADs presenting for noncardiac surgery is increasing. We report the successful management of an implanted extracorporeal BIVAD in a patient with end-stage heart failure and with pancreatic stents in a case of necrotic pancreatitis. Historical, physical, laboratory, and imaging data allowed conservative management leading to a favorable outcome.

CASE DESCRIPTION

A 54-year-old white man was transported from another hospital that had given the diagnosis of pancreatitis and where 2 ductus hepaticus commcunis (DHC) stents had been implanted. The patient had a critically low left ventricular function (ejection fraction, 18%) and several multivessel diseases. He had experienced relapsing ventricular tachycardia and had been revived several times. Placement of an intraaortic balloon pump was necessary after maximal medical treatment was no longer helpful. After consultation in our center, a biventricular assist device (BIVAD) system (Excor; Berlin Heart, Berlin, Germany) was implanted as a bridge to transplantation.

A physical examination revealed a chronically ill–appearing white man of average constitution with mild icteric sclera and jaundice. The results of heart and lung examinations were normal. The abdomen was soft and not tender. There was no hepatosplenomegaly, palpable masses, or ascites. Cutaneous fat necrosis and lymphadenopathy were absent. The patient had no history of pancreaticobiliary disease or alcohol use, and no family history of pancreatitis.

Victor Gertner and Viktor Bordel contributed equally to this work.

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The patient's medications included 25 mg carvediol, 32 mg candesartan plus hydrochlorothiazide, 20 mg enalapril, 30 mg Lasix, 50 mg spironolactone, 20 mg simvastatin, 850 mg metformin, 300 mg allopurinol, 100 mg aspirin, and total parenteral nutrition.

A laboratory evaluation revealed elevations in serum P-amylase (87 IU/L; reference interval, 8-53 IU/L) and lipase (602 IU/L; reference interval, 23-300 IU/L). Total serum

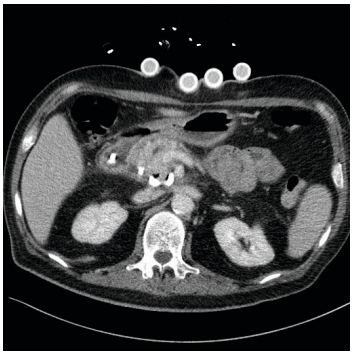


Figure 1. Abdominal computed tomography (CT) scan showing pancreatic stents in place, as well as the inflow and outflow canulas of the biventricular assist device (BIVAD) located above the epigastrium.



Figure 2. Abdominal computed tomography (CT) scan showing the pancreatic stents (coronary section).

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bilirubin was also elevated (1.8 mg/dL; normal, 1.2 mg/dL) with a direct fraction of 0.66 mg/dL (normal, 0.3 mg/dL). Alkaline phosphatase was also elevated (152 U/L; reference interval, 40-130 U/L). Liver transaminases were also mildly elevated with an aspartate aminotransferase value of 98 U/L (normal, 50 U/L). The alanine aminotransferase value was 17 U/L (normal, 50 U/L). Serum calcium and triglycerides were normal. Blood and urine cultures for bacteria and fungi were negative.

A computed tomography scan of the abdomen revealed a cystic change in the pancreatic corpus. The Wirsung duct was normal. A groove pancreatitis was in the tail despite stents and the corpus. Also observed were minimal free pancreatic liquid and an increase in the lymph nodes of the pancreas (Figure 1).

The hepatobiliary ultrasound examination found no stones or ductal dilatation. An endoscopic retrograde cholangiopancreatography evaluation supported the diagnosis of pancreatic rupture, and DHC stents were positioned. Furthermore, a transduodenal puncture of the pseudocysts with bulb dilatation was carried out and was followed by placing a double digital stent to drain the transduodenal necrosis (Figure 2).

After admission, the patient's deteriorating cardiac function required placement of a BIVAD in the operating room, which was an uncomplicated procedure.

Anticoagulation treatment was started on the second postoperative day with intravenous heparinization. The stents were removed by endoscopy on the fourth postoperative day. The patient presented with 2 episodes of melena, which were stopped by lowering the dose of intravenous heparinization. In addition, we continued the oral anticoagulation therapy with Coumadin and aspirin administration.

After 14 uneventful days in the surgical intensive care unit and 5 days in the intermediate care station (IMC) station, the patient was discharged home with instructions to continue his medications until he received a call back for heart transplantation.

DISCUSSION

This report describes the management of BIVAD implantation in patients with necrotic pancreatitis. Chronic BIVAD therapy has been found to reverse the progression of heart failure [McCarthy 1995; Mueller 1999; Nicolosi 2003; Swanson 2003]. It restores both the arterial pressure and cardiac output to near normal values and relieves the symptoms of heart failure [Swanson 2003]. It also produces improvements in hepatic, renal, and pancreas functions, which reflect better end-organ perfusion [McCarthy 1994; Swanson 2003]. We conclude that implantation of a pulsatile BIVAD can improve the course of chronic pancreatitis. The management of the anticoagulation therapy was clearly a challenging step in this patient, especially considering that there were 2 episodes of upper gastrointestinal bleeding, which were managed conservatively. The paracorporeal pulsatile BIVAD system (Excor) was successfully used in this case, for which aggressive anticoagulation therapy could not be used during the first 2 postoperative weeks. We were able to manage anticoagulation adjustment with our paracorporeal pump by minimal heparinization until the gastrointestinal bleeding was stopped.

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