An Unusual Case of Cannula Obstruction Resulting from Venous Thromboembolism

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Abstract: Cerebral strokes of unknown origin frequently present with a patent foramen ovale (PFO), a common atrial septal defect occurring in approximately 25% of the adult population. Deep vein thrombosis (DVT) or pulmonary embolism (PE) in the presence of a PFO can produce paradoxical systemic embolization subsequent to an increase in pulmonary pressure, permitting entry of thrombi into the arterial circulation. Diagnosis of an impending paradoxical embolism (IPDE) involves the detection of DVT or PE in the presence of an abnormal communication between left and right circulations and may include a right-to-left shunt. Treatment includes oral anticoagulation, antiplatelet agents, thrombolysis, transcatheter closure of the defect, or surgical embolectomy and PFO closure. As a result of risks of intracranial hemorrhage with fibrinolysis, pulmonary embolectomy using cardiopulmonary bypass (CPB) and deep hypothermia is a primary treatment with a surgical mortality rate at approximately 5%. Despite optimal management, IPDE is associated with a mortality rate of 18%. Prompt diagnosis and treatment is critical in avoiding systemic thromboembolization and strokes in these patients.

We report a case of superior vena cava cannula obstruction resulting from a paradoxical embolus traversing a PFO during surgery. Warning signs and management during CPB are discussed.

DESCRIPTION

A usually active, retired 80-year-old white man presented to the emergency department with shortness of breath on exertion, low arterial oxygen saturations (91%), and diaphoresis. He reported bilateral calf pain 1 day before admission and was noted to have experienced a long period of immobility during a recent journey. His build was tall and slender, measuring 187 cm and 81 kg. Medical history included hypertension, pleural plaques, and cigarette smoking (ceased 30 years ago), hematocrit 46%, low platelet count of $148 \times 10^3$ m$^-3$, increased international normalized ratio (1.4) and prothrombin time (16.2 seconds), and normal activated partial thromboplastin time (36.6 seconds).

Computed tomography of the pulmonary arteries showed evidence of multiple bilateral pulmonary emboli...
throughout the distal pulmonary artery branches and lobes. Bilateral lower limb venous duplex ultrasound revealed occlusive DVT in the right calf. Transthoracic echocardiography revealed a PFO with a large, highly mobile, serpiginous thrombus traversing the atrial septum (Figure 1). Mild pulmonary hypertension with normal left ventricular size and function was noted. Anterior T-wave inversions were noted on the electrocardiogram. Coronary angiography revealed ostial and proximal stenosis in the left anterior descending artery, ostial disease in the intermediate artery, and irregularities in the circumflex and right coronary arteries. Emergent surgery was scheduled for removal of the intra-atrial thrombus, closure of the PFO, pulmonary embolectomy, and coronary artery bypass grafting.

The operative procedure required bicaval cannulation and CPB for the exposure of the thrombus and PFO. The patient body surface area and calculated flow rates were 2.06 m² and 4.95 L/min, respectively. After heparinization and cannulation of the ascending aorta, the SVC was cannulated using a 28-Fr DLP® Right Angle Single Stage one-piece polyvinyl chloride venous cannula (Medtronic, Inc., Minneapolis, MN). Activated clotting time was maintained at >450 seconds throughout the procedure. Partial CPB was instituted using a gravity drainage technique to reduce the risk of thrombus migration during inferior vena caval (IVC) manipulation. The IVC was then cannulated using a 32-Fr venous cannula of the same model to establish complete CPB. At this point the anesthesiologist noted that the thrombus was no longer visible on transesophageal echo. The surgical team was concerned about possible systemic embolization of the thrombus. The aorta was cross-clamped and the cavae were snared. Patient cooling to a nasopharyngeal temperature of 30°C was commenced. Inspection through a right atriotomy revealed a large PFO. The thrombus could not be located in the right or left atria. The main pulmonary artery was opened and found to be thrombus-free. Throughout the procedure there was reduced venous drainage to the reservoir of the heart–lung machine, requiring additional fluid administration to maintain a safe operating venous reservoir level. A total of 2000 mL Hartmann’s solution was administered in addition to the circuit priming fluid during the 113-minute bypass period. After coronary artery grafting, the heart was deaired and the cross-clamp removed. The heart spontaneously reverted to sinus rhythm and two bipolar ventricular pacing wires were inserted. The patient was rewarmed to a nasopharyngeal temperature of 37°C and was uneventfully weaned from CPB.

On SVC decannulation, a 16-cm long thrombus was found inside the tip having traversed the side holes of the venous cannula (Figures 2 and 3). Protamine was administered and the sternotomy was closed in the routine fashion. Postoperative anticoagulant therapy continued with the patient making a full recovery before discharge on day 8.

COMMENT

Paradoxical emboli are rare, potentially fatal events requiring immediate treatment to minimize the risk of stroke.
from cerebral embolization. In this reported case, the clinical significance of DVT with a PFO warranted emergent surgery. The disappearance of thrombus at the initiation of CPB was alarming considering the potential for cerebral embolism through entry into the systemic circulation.

At the time, various theories were hypothesized as to the location of the thrombus with the most threatening scenario involving systemic entry. Alternatively, the thrombus may have been siphoned into the venous reservoir filter and been unable to be detected. The other possibility was that the thrombus had lodged in the SVC cannula. It was apparent to the perfusionist that a venous obstruction was present, causing a reduction in the blood volume in the hard-shell venous reservoir combined with an elevated central venous pressure during the procedure. No venous line pressures were measured and no visible signs of venous distension were noticed by the anesthetic team (e.g., head color change, distension of head and neck veins). Correct head position of the patient was ensured, ruling out a positional SVC drainage issue. The venous cannulae were manipulated by the surgeon in an attempt to improve venous drainage, to no avail. The discovery of thrombus in the SVC cannula on decannulation explained this finding. In retrospect, suboptimal venous return should have alerted the team to the cannula obstruction. Clamping of each venous cannula independently may have been a useful diagnostic method in determining which cannula was obstructed, and change-out on CPB could have been undertaken.

The use of near-infrared spectroscopy (NIRS) to measure the regional oxygen saturation (rSO₂%) in the frontal cortex capillary system may have been useful in identifying a declining rSO₂%, suggesting inadequate cerebral venous drainage secondary to SVC obstruction. A recent study demonstrated the benefit of NIRS in detecting SVC obstruction when all other perfusion parameters were normal (5). The application of vacuum-assisted venous drainage (VAVD) during CPB may also be valuable, because augmenting the venous return on initiation of CPB would increase the likelihood of thrombus draining into the venous reservoir rather than traversing the left heart if right-sided pressures were to increase. VAVD was not used in this instance; however, it would be used in the future if a similar case arose.

This case study highlights the issues with venous cannulation and obstruction secondary to thrombus dislodgement in the setting of a high-risk paradoxical venous thromboembolism. Any impediment to venous flow in this clinical setting should alert the perfusionist to the possibility of cannula obstruction secondary to thrombus dislodgement. Luminal venous cannula obstruction must be a consideration to the surgical team when other causes of decreased venous return have been ruled out (6).

REFERENCES