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Pulmonary Artery Catheter Migration Causing Venous Cannula Obstruction during Cardiopulmonary Bypass

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EXTRACORPOREAL circulation during cardiopulmonary bypass (CPB) requires the intracardiac placement of large-bore cannulae for venous drainage and arterial return and generally is performed without incident. The presence of other intracardiac catheters (e.g., central venous, pacing, or pulmonary artery (PA)) may interfere with proper pump function. We report a case in which an oximetric PA catheter was siphoned into the venous cannula of the CPB apparatus, resulting in intermittent occlusion of venous return. No previous reports have detailed premonitory signs of this complication.

Case Report

A 64-yr-old man with known coronary artery disease and progressively unstable angina presented for urgent coronary revascularization.

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Preoperative chest radiography noted a normal cardiac silhouette without chamber dilation. Preinduction monitoring, including peripheral intravenous catheters and a radial artery catheter, were placed under local anesthesia with intravenous sedation. An 8.5-Fr introducer was placed via a right internal jugular approach, followed by flotation of a 110-cm 8-Fr balloon-tipped oximetric PA catheter (Opticath #50328, Abbott, Chicago, IL) to a wedged position at a depth of approximately 45 cm. Baseline measurements included PA and central venous pressures (27/18 and 12 mmHg, respectively), cardiac output (7.1 l/min), and mixed venous saturation (80%). Anesthetic induction and prebypass sternotomy proceeded uneventfully. A routine postinduction transesophageal echocardiographic (TEE) study confirmed appropriate right-heart passage of the PA catheter beyond the pulmonic valve.

After anticoagulation with heparin, a two-stage venous cannula (#TR3446L, Research Medical, Midvale, UT) was placed through a purse-string arteriotomy into the right atrial appendage without resistance. Just before the onset of CPB, the PA catheter was withdrawn 4 cm while continuously demonstrating an appropriate PA tracing. CPB with mild hypothermia (32°C) commenced at a flow of 4.5 l/min for 98 min. After cross-clamping of the aorta, the mixed venous saturation increased to 100%, reflecting stagnant arterialized blood in the PA. Cold antegrade cardioplegia resulted in diastolic arrest.

Early in the bypass period, an alternating, persistently negative deflection of the PA waveform between −4 and −22 mmHg was noted, at which time the mixed venous saturation had precipitously decreased to −80%. Later, during partial CPB (flow ~2.1 l/min), as ventricular ejection was evident on the radial arterial waveform, the PA waveform remained consistently negative and dampened. Therefore, the PA catheter was manipulated in an attempt to recover a normal tracing. During balloon inflation, sudden loss of venous return to the CPB reservoir was identified by the perfusist, requiring a substantial reduction in pump flow to prevent air entrainment. Inspection of the CPB apparatus revealed no obvious extracardiac occlusion. After balloon deflation, venous return was restored, but the PA catheter could no longer be withdrawn. Repeated inflation and deflation of the PA catheter balloon yielded similar, abrupt changes in venous return; therefore, further manipulations were
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abandoned, and CPB uneventfully was terminated. During right atrial decannulation, the PA catheter was removed through the atriotomy along with the venous cannula. The distal end of the catheter had become twisted around the venous sump and migrated into a 3 × 8-mm slot in the superior caval stage (fig. 1). When the balloon was inflated, its 2-ml volume occluded the entire 12-mm lumen of the cannula (fig. 1, inset). After reintroduction into the right atrium and atrial closure, the PA catheter was floated uneventfully into an appropriate wedged position.

Discussion

Flow-directed PA catheters are ubiquitous in the perioperative management of cardiac surgery patients, providing substantial hemodynamic information for patient management. Although complications are rare, migration of PA catheter in the peribypass period can increase their incidence. The PA catheter can be mechanically displaced by other intracardiac catheters or migrate distally from temperature-induced changes in catheter deformability. As is our standard practice, the PA catheter was withdrawn 3–4 cm before the onset of CPB to reduce the likelihood of distal migration and potential pulmonary arterial occlusion or perforation. Additional catheter manipulation during partial CPB must have withdrawn the catheter into the right atrium, where the venous sump sphinctor the catheter into its effluent flow. To our knowledge, this has been anecdotally reported only once, in which a standard PA catheter obstructed an undefined atrial cannula and no clinical signs heralded the onset of impaired venous return. Unique to this case, however, the use of an oximetric PA catheter, constant PA pressure monitoring and TEE each provided insight—albeit retrospectively—on the impending venous obstruction. The TEE confirmed appropriate placement before CPB but was not rechecked until after complete separation from CPB. TEE examination generally is limited during partial CPB, particularly if the chambers are underfilled. The occurrence of a persistent negative PA waveform suggested that blood flow was being directed away from the distal port but was improperly attributed to retrograde flow across a trivially incompetent tricuspid valve. The mixed-venous saturation of 80% during full CPB reflected blood proximal to the “arterialized” stagnant PA, which was likely the right atrium. Fortunately, inflation of the PA catheter balloon in the absence of an identifiable waveform resulted only in an impairment of CPB venous return; in retrospect, serious injury could have resulted if the PA catheter had been situated in a distal pulmonary vessel. Despite all these premonitory signs, not until the obstruction and entrapment of the catheter occurred was the problem correctly identified. The specific venous cannula employed during this procedure is not unique in design, and therefore, catheters by other suppliers manufactured with similarly sized orifices would appear capable of PA catheter entrainment.

As demonstrated by this case report, we would highlight the following suggestions to prevent similar occurrences: (1) continuously monitor all intracardiac pressures (and PA oximetry, if present) throughout CPB and investigate all suspicious changes suggestive of catheter migration, (2) avoid manipulating the PA catheter (particularly withdrawal) until separation from CPB is completed, (3) avoid distal balloon inflation when an inappropriate waveform is displayed, (4) include intraluminal cannula obstruction in the differential diagnosis of sudden loss of venous return to the CPB apparatus, and (5) confirm placement of the PA catheter with either chest radiography or TEE when clinical suspicion suggests the PA catheter may be improperly positioned.

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