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Migration of Pediatric Pulmonary Artery Catheters

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Pulmonary hypertension commonly accompanies congenital cardiac anomalies with increased pulmonary blood flow.[1] The persistance of significant pulmonary artery (PA) hypertension following correction of left-to-right shunting is a major cause for increased postoperative mortality.[2] To guide therapeutic management of pulmonary hypertension postoperatively, PA catheters can be inserted directly by the surgeon at the time of repair. We describe two patients in whom pulmonary hypertension was transiently misdiagnosed when their PA catheters migrated from the PA into the aorta through an apparently closed ductus arteriosus.

REPORT OF TWO CASES

Patient 1. A 25-month-old, 8.9-kg, 79.5-cm girl with a ventricular septal defect (VSD) and anomalous abdominal venous drainage presented for VSD closure. A preoperative cardiac catheterization through the right femoral vein revealed abnormal development of the subcardinal vena cava with reabsorption on the right and persistance of the left venous drainage. A liver in the midline suggested the heterotaxia syndrome. Due to the tortuous course of the abdominal venous pathway, the catheter could not be manipulated into the right ventricle, although the right atrial pressure was 7/4 mmHg with 71% oxygen saturation. A catheter that passed through the right brachial vein could not be advanced past the axilla. Though the PA was never entered, the presence of right ventricular hypertrophy on the electrocardiogram and the chest roentgenogram, showing increased pulmonary vasculature suggested pulmonary hypertension. Catheterization through the right femoral artery revealed an aortic pressure of 81/35 mmHg with 93% oxygen saturation and a left ventricular pressure of 86/10 mmHg. The left ventriculogram showed a large subaortic VSD located high in the membranous septum with significant left-to-right shunting. An aortogram showed no evidence of a patent ductus arteriosus (PDA).

The patient underwent surgical correction of the VSD following induction of anesthesia with halothane and pancuronium and insertion of intravenous, left radial arterial, and left external jugular central venous pressure catheters. Prior to cardiopulmonary bypass (CPB), a PA pressure of 37/10 mmHg and a right ventricular pressure of 42/0 mmHg were obtained by direct puncture. The VSD was repaired during a 23-minute cross-clamp period. After weaning from CPB, size 4-French balloonless catheters¶ were inserted by the surgeon directly into both atria and the PA. The presumed PA pressure recorded at this time was systemic (96/66 mmHg) and hyperventilation was initiated while further clinical assessment was begun. Because the patient was clinically stable and the PA pressure did not feel systemic to the surgeon's palpation, the catheter was withdrawn slowly. A sudden decrease in the PA pressure to 42/24 mmHg occurred while the simultaneously recorded right ventricular pressure was 45/5 mmHg. Oxygen saturations with a $F_{I_{O_2}}$ of 1.0 were taken from the right atrium (76.3%), pulmonary artery (78.1%), and radial artery (98.7%). The remainder of the patient's hospital course was uneventful.

Patient 2. A 16-month-old, 7.5-kg, 69-cm boy with Down's syndrome and a complete atrioventricularis communis (AV canal) presented for surgical correction of his cardiac lesion. Cardiac catheterization at four months of age revealed a right atrial pressure of 7/6 mmHg with 69% oxygen saturation, right ventricular pressure of 97/8 mmHg with 80% oxygen saturation, pulmonary artery pressure of 89/36 mmHg with 80% oxygen saturation, a pulmonary venous saturation of 95%, a left atrial pressure of 6/6 mmHg with 85% oxygen saturation, a left ventricular pressure of 98/8 mmHg with 84% oxygen saturation, and an aortic oxygen saturation of 84%. The pulmonary-to-systemic blood flow ratio was 1.4:1, and a complete AV canal with left-to-right shunting at the atrial level and both left-to-right, and right-to-left shunting at the ventricular level were noted. Preoperative recatheterization revealed a right atrial pressure of 8/7 mmHg with 68% oxygen saturation, right ventricular pressure of 104/8 mmHg with 71% oxygen saturation, and left ventricular pressure of 98/7 mmHg with a 97% oxygen saturation. The pulmonary-to-systemic flow ratio was 1.12:1, and mild mitral regurgitation in addition to the complete AV canal were noted. No aortogram was obtained during either catheterization.

The child underwent repair of the AV canal following induction of anesthesia with fentanyl, halothane, and pancuronium and insertion of an intravenous, left radial arterial, and right external jugular catheters. Following a 60-min cross-clamp time and weaning from CPB, size 4-French balloonless catheters were inserted directly by the surgeons into the PA, left atrium, and right atrium. The PA pressure was 32/20 mmHg with an aortic pressure of 92/58 mmHg. Following transport to the ICU, the PA pressure had increased to 64/57 mmHg with an arterial pressure of 100/40 mmHg, left atrial pressure of 10 mmHg, and a central venous pressure of 13 mmHg. Hyperventilation with 100% oxygen and nitroprusside infusion through the PA catheter were initiated, but nitroprusside was soon discontinued because of systemic arterial hypotension. Shortly thereafter, the presumed PA

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pressure rose to systemic levels (93/60 mmHg). Suspicion of PA catheter migration arose when the right atrial pressure failed to increase above 8 mmHg, and the patient's clinical condition remained stable. A chest roentgenogram (fig. 1) showed that the PA catheter had passed from the PA through the ductus arteriosus into the ascending aorta. Simultaneous oxygen saturations at this time taken from the right atrium, presumed PA, and the aorta were 52.6%, 97.9%, 97.7%, respectively. As the PA catheter was withdrawn to its proper location, the waveform changed from an aortic trace of 100/57 mmHg to a dampened trace of 60/35 mmHg (presumably with the catheter tip in the ductus), and finally to a PA trace of 25/18 mmHg. The PA pressure eventually stabilized at 44/30 mmHg. The oxygen saturation in the PA blood at this time was 58.8%.

Discussion

Closure of the ductus arteriosus normally occurs within the first two weeks of life, but persistant reactivity of the vascular wall allows periodic reopening during the early months of life.[3] Eventually, the vascular wall reactivity is lost, and fibrotic obliteration of the lumen leads to formation of the ligamentum arteriosus. Until full fibrosis occurs, however, a potential opening is present through the ligamentum. In patients with congenital heart disease, a higher incidence of persistant ductus patency is noted.[4]

Both reported cases represent instances where directly placed PA catheters were unintentionally introduced through a presumably closed ductus arteriosus. In Case 1, a preoperative aortogram did not show a PDA despite a pressure differential of 37/10 mmHg in the PA compared with the 100/50 mmHg in the aorta. The relative desaturation of the aortic blood (93%) does argue for the presence of either significant intrapulmonary shunting or some right-to-left cardiac shunting, perhaps through a PDA, though the large VSD is the most likely site. Also, both a comparison of postoperative right atrial and PA blood samples revealing no major step up in oxygen saturation and the normal aortic oxygen saturation argue against the presence of a PDA.

In Case 2, the argument for the ductus arteriosus being closed preoperatively is less complete since a preoperative aortogram was not performed. The similarity of the PA, left ventricular, and aortic pressures in this case may have prevented visualization of a patent ductus even with an aortogram. The cardiac catheterization had shown no step up in oxygen saturation between the right ventricle and PA arguing against left-to-right shunting through a PDA and no step down in oxygen saturation between the left ventricle and aorta, arguing against right-to-left shunting through a PDA. Following the discovery of the PA catheter migration and adjustment of its position, a slight step up in the PA oxygen saturation compared with the right atrial saturation (58.8% and 52.6%, respectively) was noted which argues that some left-to-right flow through the ductus was occurring at this time.

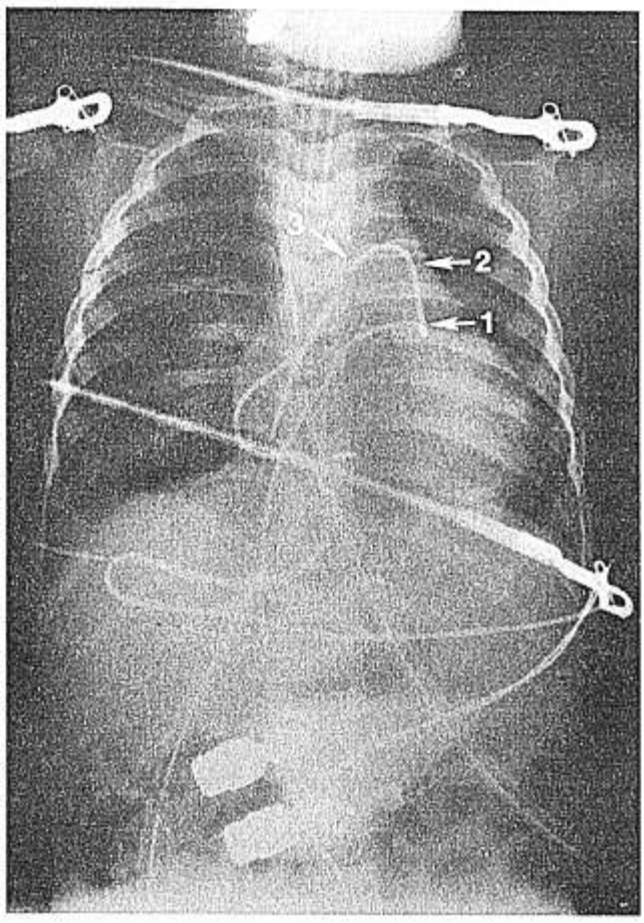

FIG. 1. Chest roentgenogram from Case 2 showing the catheter lying within the main pulmonary artery (Arrow 1), entering the ductus arteriosus (Arrow 2), and extending into the aorta (Arrow 3). The catheter tip lies in the ascending aorta.

In neither of the reported cases was there any evidence for the ductus being patent during CPB, including the presence of PA distension, increased left atrial pulmonary venous return, or systemic hypotension at normal bypass flow rates. Whether the migration of the PA catheter occurred through an existing patent ductus or was pushed through the potential opening of a constricted but not obliterated ductus is of secondary importance. Paramount is recognition that such migration can occur so that inappropriate medical therapy is avoided.

In Case 1, the pulmonary artery catheter was forced through the ductus arteriosus by the surgeon during the insertion. In Case 2, some redundant pulmonary artery catheter allowed catheter migration through the ductus upon changing the patient's position. To prevent this situation, overadvancement of the pulmonary artery catheter should be avoided and manipulation of

the catheter into a branch PA at the time of insertion should be attempted.

Suspicion of pulmonary artery catheter migration through a ductus arteriosus should exist whenever an unexpectedly high PA pressure or a sudden increase in PA pressure occurs. The diagnosis of catheter migration through a ductus arteriosus can be made by showing equal oxygen saturations when simultaneous systemic and "pulmonary" arterial blood gases are obtained. A chest roentgenogram can be of help in making the diagnosis, as was seen in Case 2. In addition, the similarity in waveform with superimposition of arterial and PA traces may suggest that migration has occurred. Unfortunately, since the pressure trace may be distorted through a size 4-French PA catheter, dampened when the catheter tip is lying within the ductus arteriosus proper, and calibrated on a different scale, comparisons of wave trace forms may not be as helpful as might be expected.

Since both these patients had preoperative PA hypertension and were at risk for persistant postoperative elevation of the PA pressure, the recorded pressures taken from their PA catheters placed under direct vision, were taken at face value. The initial lack of recognition of the true placement of the PA catheters in both these patients led to the contemplation or institution of inappropriate medical therapy. A high degree of suspicion that inadvertant PA migration can occur in conjunction with a lack of clinical substantiation of an increased PA pressure should lead to the rapid diagnosis and correction of this problem when it occurs.

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