Serendipitous Discovery During Jugular Catheterization

Partial Anomalous Pulmonary Venous Connection

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This report describes the identification of a partial anomalous pulmonary venous connection during routine central venous catheterization in a man with pulmonary hypertension. To our knowledge, this is the first report in the English literature to describe this occurrence during central line placement. (Chest 1990; 98:493-95)

PAPVC = partial anomalous pulmonary venous connection

Placement of central venous catheters has become an increasingly common accompaniment of modern medicine. The frequent need for central pressure monitoring, hyperalimentation, and long-term venous access leads large numbers of patients to undergo this invasive procedure. It is surprising that the incidence of complications is relatively low and that malposition of catheters is rare. This case describes the first English-language report (to our knowledge) of the serendipitous discovery of an anomalous pulmonary venous connection during placement of a jugular catheter. Of particular interest, this anomaly may have contributed, at least in part, to our patient's pulmonary hypertension.

CASE REPORT

A 58-year-old man with a 40-pack-year history of cigarette smoking and ten-year history of asthma was admitted to the hospital for treatment of acute bronchospasm. He had been treated with theophylline and an inhaled β-agonist and had required hospital admission on three occasions for exacerbations. He had rarely been placed on a regimen of corticosteroids and had never been intubated. On the day of hospital admission he developed severe respiratory distress and he was intubated in the emergency room. On admission the electrocardiogram showed right axis deviation. Echocardiography revealed right ventricular hypertrophy. On the fifth hospital day a pulmonary artery catheter was placed to exclude a diagnosis of cardiogenic pulmonary edema. This revealed moderate pulmonary hypertension (68/44 mm Hg with pulmonary capillary wedge pressure of 10 mm Hg), a mixed venous oxygen saturation of 80 percent, and a thermodilution cardiac output of 9 L/min consistent with evolving Pseudomonas sepsis. A complicated and stormy course followed, with myocardial infarction, refractory bacterial and candidal sepsis, massive gastrointestinal hemorrhage, and acute renal failure.

On the 30th hospital day a venous introducer sheath and venous catheter were percutaneously placed through the left internal jugular vein without difficulty. A pressure tracing from the tip of the catheter is shown in Figure 1, and this led to concern about the catheter's position. The patient was being mechanically ventilated and was on an inspired oxygen fraction of 0.6.

Blood from the side arm of the introducer sheath, from the tip of the catheter, and from an arterial line were sent for analysis of blood gases as follows:

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<tbody>
<tr>
<td>Introducer sheath</td>
<td>73</td>
<td>53</td>
<td>7.31</td>
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<tr>
<td>Catheter tip</td>
<td>307</td>
<td>33</td>
<td>7.50</td>
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<tr>
<td>Arterial line</td>
<td>70</td>
<td>50</td>
<td>7.37</td>
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A posteroanterior and lateral chest roentgenogram showed the tip of the catheter to be within the left lung in three dimensions (Fig 2). Injection of intravenous contrast was performed to confirm the position of the catheter and showed filling of an anomalous pulmonary vein. This large vein drained into the left brachiocephalic vein just as it was joined superiorly by the jugular vein. Our catheter has passed down the jugular vein and directly into the anomalous vein. Under fluoroscopic guidance the catheter was redirected over a wire into the superior vena cava. Eventually the patient died secondary to multiple organ failure and sepsis. An autopsy was not performed.

DISCUSSION

The frequency of aberrant placement of central venous catheters using the internal jugular vein as the insertion site varies between 0.8 percent and 5.7 percent.1 The oxygen saturation of blood from the catheter tip in our patient is consistent with its position in a pulmonary vein draining a lung ventilated with 60 percent oxygen. The pulsatile nature of the waveform is explained on the basis of a catheter whose tip is wedged into a pulmonary venous branch. This converts
the pressure tracing from that of the pulmonary vein to that of the upstream pulmonary artery, as has been demonstrated in animal experiments and human studies.\(^4\) The tracing in Figure 1 no longer demonstrates pulmonary hypertension, probably due to the patient's preterminal, severe hypoperfusion state. It is interesting that, in retrospect, the high thermodilution cardiac output and elevated mixed venous oxygen saturation seen at pulmonary artery catheterization and attributed to sepsis, may have been partly due to his left-to-right shunt.

During embryologic development a common pulmonary vein evaginates from the left atrium and joins the splanchnic plexus of veins in the region of the developing lung buds. Occasionally some or all of the pulmonary veins fail to establish connection with the left atrium. Pulmonary venous blood may then return to the right atrium directly or via a derivative of the primitive cardinal venous system, usually the coronary sinus, vena cava, or one of its branches. Total anomalous pulmonary venous return typically presents in infancy with congestive heart failure. In contrast, partial anomalous pulmonary venous connection (PAPVC) may be asymptomatic and found incidentally on chest roentgenogram or during evaluation of a murmur, or even be an incidental finding at autopsy; PAPVC is often associated with atrial septal defect and occasionally with complex cardiac defects. Isolated PAPVC in association with intact atrial septum remains unusual and may join the venous circulation at various locations.\(^3\) Drainage of the left upper lobe via a vertical vein to the brachiocephalic vein, as in our patient, is the most common pattern.\(^4\)

The clinical significance of PAPVC relates to the magnitude of the left-to-right shunt. The number of anomalous pulmonary veins, the presence and degree of pulmonary venous obstruction, and the presence of concomitant lung disease affect the risk for the development of pulmonary hypertension. It has generally been assumed that if less than 50 percent of the pulmonary circuit drains into the right side of the heart, pulmonary hypertension would not ensue. Recent reports by Saalouke et al.\(^5\) and Babb et al.\(^6\) have challenged this notion and suggest that pulmonary hypertension can develop even with small shunts due to PAPVC, although mechanisms to explain this are speculative.

We are unable to estimate the magnitude of left-to-right shunt in our patient since at the time of PAPVC discovery it was not possible to obtain a true mixed venous oxygen saturation antegrade to the site of the shunt. It is not fully satisfying to attribute our patient's pulmonary hypertension and right ventricular hypertrophy to his asthma. It is at least possible that his life-long shunt played a role. Recognition of the significance of a malpositioned central venous catheter therefore contributed to our synthesis of this patient's findings.

**REFERENCES**

This cardiogenic fashion, if Thrombolytic TST with Clinical Lecturer, William Centre, University an anomalous vascular anomalous venous connection: a congenital defect with late and serious complications. Ann Thorac Surg 1981; 31:540-43

Threatened Reinfarction*
Effective Therapy Using Streptokinase with Reversal of Cardiogenic Shock

Thrombolytic therapy has an established niche in the treatment of acute myocardial infarction. One view, often rigidly held, is that this therapy should be attempted only if ischemic pain is present for less than four hours. Additionally, treatment is often reserved for those with an anterior infarction, a subgroup that did well in early reports. This case report demonstrates an impressive reversal of cardiogenic shock after streptokinase therapy in a patient who experienced an inferior infarction. His chest pain and ST elevations were present for more than nine hours at the commencement of treatment. (Chest 1990; 98:495-97)

Thrombolytic therapy has been practiced in several areas, but efficacy is most convincing in acute myocardial infarction. Previous studies have demonstrated increased rates of recanalization, improved left ventricular function, and reduced mortality. Because of its large size, the GISSI study was able to conclude that certain groups of patients were more likely to benefit from thrombolytic therapy than others. Mortality was significantly reduced in patients aged younger than 65 years, in those experiencing a first infarction, or in those in Killip class 1 or 2. The most striking reduction in mortality, 47 percent, occurred in patients administered streptokinase within one hour of the onset of symptoms. Recent data, however, suggest that benefit may be realized up to 24 hours after the onset of infarction. While successful recanalization is commonplace, and reversal of cardiogenic shock has been reported, we report the case of a patient with cardiogenic shock in whom streptokinase therapy was begun nine hours after the onset of chest pain and was associated with an impressive reversal of the shock state.

Case Report
The patient is a 69-year-old man who presented to a referring hospital with three hours of chest tightness that was associated with nausea, diaphoresis, and dyspnea. He had been experiencing a stable anginal syndrome for about four years prior to hospital admission, consisting of intermittent chest discomfort on exertion that was relieved by sublingual nitroglycerine or rest. In the referring hospital, the patient had a slow junctional rhythm that responded to atropine and resulted in transfer to our hospital. On admission to our Cardiac Care Unit, he was hemodynamically stable, and physical examination was remarkable only for bibasilar crackles on auscultation of the lungs. His electrocardiogram revealed acute inferolateral ischemia and first-degree atrioventricular block. Intravenous streptokinase, 1.5 million units, was administered, resulting in partial resolution of the inferior ST elevations. Follow-up therapy consisted of aspirin 325 mg daily, but no intravenous heparin. Subsequently, the peak creatine phosphokinase value was 7,200 U/L, and the MB fraction was more than 5 percent. For the next few days the patient experienced mild postinfarction angina that was always relieved by sublingual nitroglycerine. A baseline

Figure 1. Electrocardiogram prior to streptokinase administration. Note marked ST elevations inferiorly.