CASE REPORTS

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Upper Extremity Veno-arterial Shunting during Pediatric Orthotopic Liver Transplantation

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ORTHOTOPIC liver transplantation is a definitive therapy for various acute and chronic liver diseases, including congenital abnormalities and inherited metabolic diseases in children. The procedure may involve significant blood loss and transfusion, and invasive monitoring is used to maintain close hemodynamic control. We report a case of spurious arterial blood gas findings and abnormal pulse oximetry readings during pediatric liver transplantation.

Case Report

The patient was a 14-month-old 8.7-kg boy with biliary atresia who underwent portoenterostomy (Kasai procedure) at 10 weeks of age and a revision at 4 months of age who had been living at home for 10 months. He was scheduled for urgent orthotopic liver transplantation because of deteriorating liver function, which occurred over a 2-week period.

Anesthesia was induced using thiopental (50 mg) and fentanyl (25 μg intravenous bolus) and maintained with isoflurane, 0.2–0.6%, with air/oxygen and boluses of fentanyl intermittently. Muscle relaxation was maintained using pancuronium. Monitoring included electrocardiography, blood pressure, end-tidal carbon dioxide (ET_{CO_2}), and pulse oximetry (Sp_{O_2}). Central venous pressure was monitored intermittently *via* a double-lumen left internal jugular vein catheter placed before transplant for plasmapheresis. Twenty-two-gauge catheters were placed in the both the right and the left radial arteries to monitor blood pressure continuously and to allow frequent arterial sampling. Large-bore peripheral venous access was secured using an 18-gauge intravenous catheter at the base of each thumb, lateral to the arterial catheters.

Massive hemorrhage was encountered during dissection of the diseased liver. The patient was immediately administered rapid pressurized transfusion by pressurized bag (inflated to approximately 250 mmHg) and manual syringe injection *via* the right 18-gauge peripheral intravenous line. Intermittent 50–100-mg boluses of calcium chloride also were administered through this line. Within moments of the initiation of pressurized transfusion, it was noted that the Sp_{O_2} reading from the right-hand probe decreased from 100% to 66%, although breath sounds were good and ET_{CO_2} waveform was normal. An arterial blood sample drawn from the right radial catheter reflected values similar to those of packed erythrocytes. Sp_{O_2} from the left-hand and right-foot probes was 100%, whereas a simultaneous reading from the right hand was 66%. Arterial blood sampling from the left radial catheter (with the left peripheral intravenous line running at keep vein open) showed more normal values (table 1). The right hand was examined and was found to be dusky and slightly edematous, but not taut, after infusion of 6,000 ml fluid. The blood pressure tracings from the right and left radial arterial catheters remained identical, with a blood pressure of 70/40 mmHg.

All infusions through the right peripheral intravenous line were immediately discontinued and begun in the left peripheral intravenous line. The Sp_{O_2} in the right hand returned to 100% within 2 min. However, during rapid transfusion *via* the left intravenous catheter the Sp_{O_2} of the left hand decreased to 80%, and subsequent arterial sampling from the left radial catheter reflected values similar to those of packed erythrocytes. All infusions were subsequently administered *via* the central venous catheter. The Sp_{O_2} of the left hand quickly returned to 100% and a subsequent arterial sample from the left radial artery reflected more normal values.

Consultation was obtained from a hand surgeon at the completion of the liver transplant because the right hand continued to appear swollen and dusky. Fasciotomy of the right distal forearm was performed for increased compartment pressure of 35 mmHg and concern of decreased venous return. Subsequently, the color of the hand improved, and ultimately there was no loss of function.

Postoperative arteriography and venography of the left hand were obtained to evaluate the suspected arteriovenous shunting. The venogram (fig. 1) showed evidence of distal arteriovenous connection.

Discussion

Venoarterial admixture has been shown in patients with several types of chronic liver failure.[1–5] Several sites of arteriovenous shunting have been shown morphologically in patients with chronic liver disease; these include intrapulmonary, portopulmonary, and pleural shunts.[3,5]

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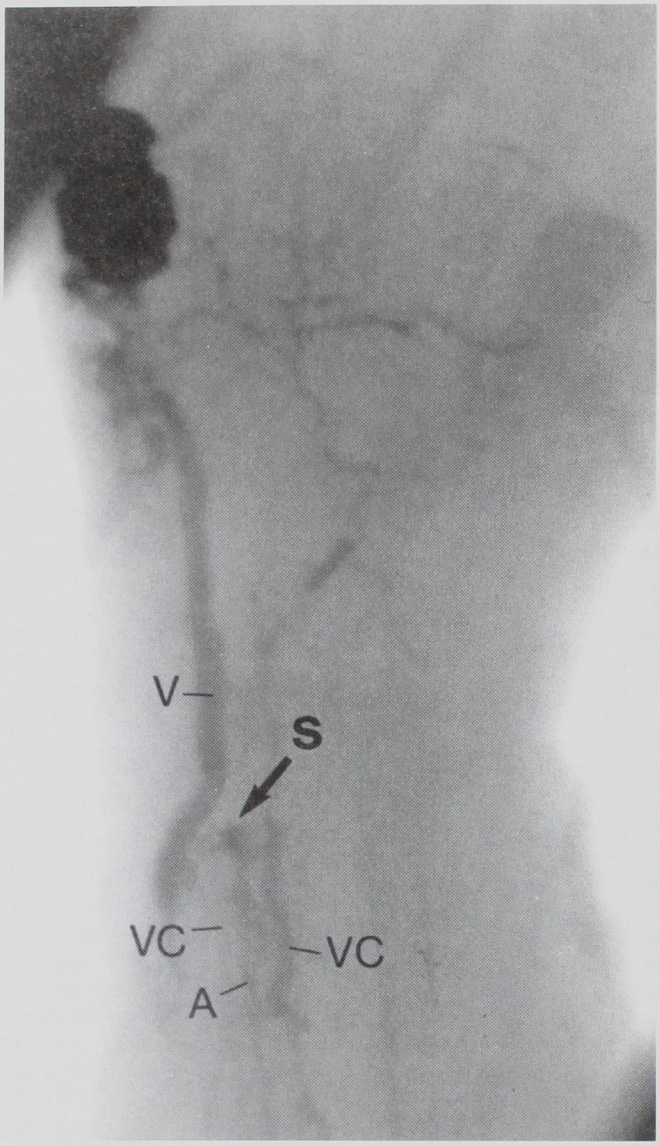

Fig. 1. Selective injection of the venous catheter (V) shows a tortuous loop and early fill of the artery (A) with the venous injection. The communication or shunt is represented by S. The venae communitantes (VC) are also delineated after venous contrast injection.

Shunting can occur by three different mechanisms: anatomic communications between pulmonary arterioles and venules, dilated capillary beds decreasing effective oxygen diffusion, or ventilation-perfusion mismatching.[5] There are few published data available describing arteriovenous shunting occurring at more distal sites.

Possible explanations for the observed venoarterial admixture are anatomic and pressure related. The anatomic basis would include the presence of a congenital vascular anomaly (fig. 2), the presence of a posttraumatic venoarterial shunt, or a catheter tip traversing a new communication. The pressure-related origin allows retrograde flow through capillary beds. Vascular relaxation of pre- and postcapillary sphincters is enhanced by high levels of endogenous vasodilators (including estrogen) in hepatic failure,[6] by general anesthesia, and by extremely high venous pressures that might occur during pressurized infusion. Dilation of the capillary and precapillary beds have been described in the pulmonary system.[7,8] Such shunting may be trivial during normal, awake circumstances but could become significant during the physiologic stress of surgery, massive blood loss, decreased arterial blood pressure, and with pressurized transfusion.

Our physical examination, arterial blood gas data, angiographic findings, and experience with other patients[9] support the existence of an established arteriovenous

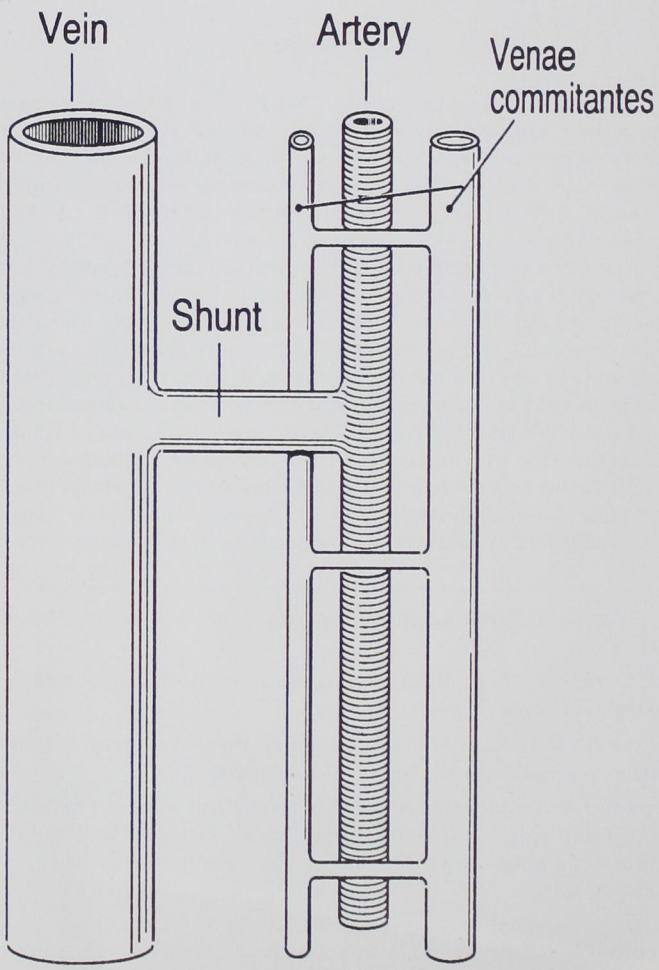

Fig. 2. * Diagramatic representation of the shunt that connects the radial artery (A), one venae communitantes (VC), and the larger cephalic vein (V).

Table 1. Serial Blood Gas and Serum Chemistry Results

	Time					
	1700	1707	1712	1737	1746	1800
Arterial sample site	Right	Right	Left	Right	Left	Left
Intravenous infusion site	Right	Right	Right	Left	Left	Central
$F_{I_{O_2}}$	0.5	1.0	1.0	1.0	1.0	1.0
*p*H	6.94	7.22	7.31	7.19	6.96	7.27
p_{O_2} (mmHg)	47	62	409	106	42	500
p_{CO_2} (mmHg)	70	64	30	29	85	49
BE	−19	−4	−10	−16	−16	−5
Hct (%)		32	31	26		30
Na^+ (mEq/L)	146	149	148	151	151	151
K^+ (mEq/L)	8.3	8.6	5.3	4.1	5.0	5.4
Ca^{2+} (mM)	5.7	4.7	2.1	0.33	0.09	1.2

shunt. Medium peripheral arteries, such as the radial and ulnar arteries at the wrist, are accompanied by paired venae commitantes that have frequent step-ladder connections on either surface of the central artery.[10] Subsequent puncture for arterial blood gas determinations or arterial catheter insertion for monitoring may result in such venoarterial communications that the catheter will often traverse venae commitante and artery simultaneously.[6] In normal situations, vasoconstriction of the arterial wall and low venous pressure in collapsed scarred veins prevents arteriovenous shunting. However, the clinical history of this patient did not support such a mechanism.

In the case presented, the use of pressurized venous infusion necessitated by ongoing blood loss, together with systemic arterial hypotension in the presence of an arteriovenous communication, produced the results observed. The presence of acidemia, hypercapnia, and hyperkalemia on arterial blood gas analysis is not surprising when the average properties of 7-day-old blood products stored in citrate phosphate dextrose (CPD) preservative are considered. The presence of hyper- or hypocalcemia was also a localized phenomenon reflecting sampling artifact depending on sampling site and substance being infused, calcium chloride (producing locally elevated Ca^{++} levels) *versus* CPD blood (decreasing Ca^{++} levels). The apparent extreme hypocalcemia observed (0.09 mmol/l) was inconsistent with the normal hemodynamic and electrocardiographic parameters observed, showing that it was a localized phenomenon.

This case shows the presence of distal arteriovenous connections in a patient with chronic liver disease and that such connections can become physiologically important shunts during surgical stress and changes in pressure gradients. It also emphasizes the importance of recognizing local causes of arterial blood gas and pulse oximetry abnormalities.

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