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EDITORIAL COMMENTARY

A vicious circle

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Venovenous collaterals are commonly encountered after bidirectional cavopulmonary shunt (BCPS) or Fontan procedures. Up to one-third of patients after BCPS procedures have been diagnosed with collateral channels, more than half of them draining below the diaphragm.¹

Krishnamoorthy and colleagues² report a very unique scenario in a patient with tricuspid atresia, ventricular septal defect, and pulmonary stenosis. After uneventful BCPS, the patient developed cyanosis caused by a decompressing left superior vena cava (LSVC) draining to the coronary sinus. This resulted in a circular shunt from the pulmonary artery via BCPS, innominate vein, and LSVC to the atria and ventricles back into the pulmonary artery.

Recanalization of an occluded LSVC has been reported after BCPS and Fontan procedures.³⁻⁵ Girisch and colleagues⁵ reported on 2 patients with double inlet left ventricle and pulmonary stenosis who experienced cyanosis after Fontan due to a recanalized LSVC.⁵ A patient with unbalanced atrioventricular septal defect, hypoplasia of the right ventricle, and ventricular septal defect showed desaturation caused by reopening of an LSVC pathway.⁶ Nevertheless, recirculation was never mentioned despite the persistent connection of the right ventricle to the pulmonary arteries. A case series at our institution described a patient with tricuspid atresia, ventricular septal defect, and recanalized LSVC that resulted in a circular shunt across an initially ligated pulmonary artery after Fontan completion.³

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Central Message

Venous collaterals after bidirectional cavopulmonary shunt with a subpulmonary ventricle may cause a progressive, self-maintaining circular shunt.

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Although the LSVC in the presented patient can be seen as one of many possible collateral channels, the physiology is quite unique. Opposed to the solely decompressing effect of a systemic venous collateral after BCPS without right ventricle to pulmonary artery connection, the reopening of the LSVC in the setting of a subpulmonary ventricle initiates a vicious circle. The pulmonary blood flow caused by the subpulmonary ventricle leads to higher pressure in the BCPS.^{7,8} This in turn increases the pressure difference between the upper and lower venous compartment and the decompressing blood flow through the LSVC. The increased direct return to the atrium not only reduces the transpulmonary gradient, but also increases ventricular preload and right ventricular stroke volume, thereby further reducing the driving force for antegrade BCPS flow.

Unfortunately, some details are not reported. It remains unclear if the systemic venous anatomy was assessed before BCPS. Therefore, we can only assume that the LSVC was indeed functionally occluded. The age at BCPS and the time between initial operation and cyanosis is not mentioned. The report also does not state whether the right ventricle to pulmonary artery connection is part of the authors' routine strategy or if late BCPS necessitated additional pulmonary blood flow.

The report highlights that it is crucial to assess systemic venous anatomy before BCPS. Negative imaging may provide proof of functional obliteration of collateral channels but does not exclude their existence. A subpulmonary ventricle may increase the risk of venovenous collaterals that reduce the efficiency of BCPS and cause a redistribution of blood flow to the lower body.⁷ Nevertheless, the numerous reports of patients with a decompressing LSVC have nicely shown that catheter occlusion either with coils or other devices is feasible, very efficient, and can help avoid reoperation in these patients.

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