Unexplained desaturation following a Glenn shunt

The Editor,

A 2-year-old child weighing 9 kg presented with cyanosis since birth. The peripheral saturation on room air was 45%. Echocardiogram revealed situs solitus, levocardia, unbalanced atrio-ventricular septal defect, malposed great arteries and severe pulmonary stenosis with a peak gradient of 70 mmHg. The pulmonary veins were draining normally. Angiography showed borderline right pulmonary artery, good size left pulmonary artery, right superior vena cava (RSVC) and innominate vein. There was no left superior vena cava (LSVC). Patient was scheduled for bi-directional Glenn (BDG) shunt.

After median sternotomy, right lobe of thymus was excised and pericardiotomy done. Intraoperatively, pulmonary arteries were about 6 mm each, but with uniform caliber throughout, with good sized innominate vein and RSVC. Mean pulmonary artery pressure was 15 mmHg. A BDG (cavopulmonary anastomosis) was performed on cardiopulmonary bypass. Following surgery, the Glenn pressure was 19 mmHg with antegrade flow preserved and 17 mmHg with main pulmonary artery (MPA) ligated. However, unexpectedly, patient's saturation was only 50%. In view of low saturation, MPA was not ligated and patient was shifted to intensive care unit. Post-operatively, over few hours, saturation dipped to 25%. However, clinically the patient improved with good urine output and decrease in facial puffiness. Chest X-ray showed non-oligemic lung fields. Echocardiogram showed good Glenn flow and contrast echocardiogram performed by injecting agitated saline through the left brachial vein showed no LSVC. The options considered were to perform a cardiac catheterization with oximetry studies, Glenn takedown with Blalock-Taussig shunt or re-exploration with oximetry performed on the operating table by the surgeon to locate the possible site of a right to left shunt. It was decided to take up the patient for re-exploration. On re-exploration, the left lobe of thymus was removed and to our surprise, a big LSVC was noted [Figure 1]. This was ligated following which systemic saturation immediately

rose to 89%. The Glenn pressure, however, became 30 mmHg. At this time, the left atrial pressure was 8 mmHg. The MPA was ligated and some volume was let off following, which the Glenn pressure decreased to 25 mmHg with left atrial pressure of 2 mmHg. Post operatively patient did well and was extubated on the first post-operative day.

Normally, left internal jugular vein (IJV) and axillary vein join the innominate vein and drain via SVC to right atrium, and to the pulmonary circulation after BDG. Occasionally, left IJV and axillary vein join and drain separately through LSVC into coronary sinus. In that case Bilateral BDG is required. The left IJV and axillary vein may also drain into both innominate as well as LSVC (as in this case). In such a situation, following BDG, the LSVC provides an alternative path and steals blood from BDG and divert flow into RA and systemic circulation as the mean RA pressure is lower than the mean pulmonary artery pressure (Glenn pressure), which cause desaturation, as in this patient. Therefore, as soon as the LSVC was interrupted, the stealing stopped, the saturation improved and the Glenn pressure increased.

The LSVC develops from failure of obliteration of left anterior cardinal vein and the innominate vein develops from anastomotic connections between right and left anterior cardinal veins. Normally, the LSVC regresses leaving the ligament of Marshall.[1] Usually, LSVC exists in the absence of innominate vein. However, a small LSVC may occur in the presence of innominate vein along the fold of Marshall.[2] It is certainly very unusual to find a large innominate vein, in the presence of a large LSVC, causing significant desaturation, as in our patient. Increasing cyanosis after a BDG has been described due to development of abnormal venous channels, which decrease effective pulmonary blood flow.[3] These abnormal vessels connect SVC with inferior vena cava, atrium and pulmonary veins and could be *de novo* angiogenesis or from re-opening of pre-existing channels.[3] The reason for development of abnormal venous channels are alteration in hemodynamics following establishment of BDG and increased pressure gradient between pulmonary and systemic venous circuits which promote their development.[3] It is pertinent to note that the presence of LSVC was missed in pre-operative angiogram as well as in post-operative echocardiogram. Not dissecting the left lobe of thymus also caused us to miss it. The LSVC should always be looked for in patients undergoing BDG even though pre-operative investigations do not point to its existence. The presence of bilateral SVC mandates a bilateral Glenn, but in this case, it was not required because of the presence of a good-sized innominate vein.

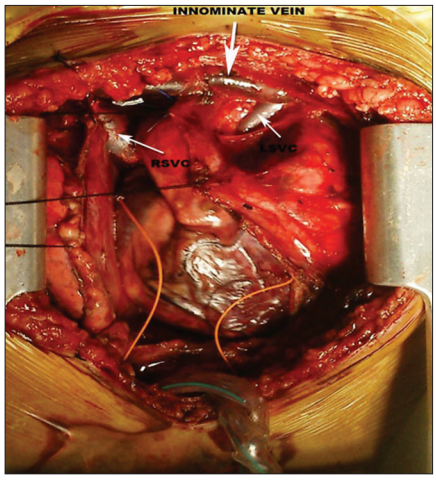

Figure 1: Large left superior vena cava, innominate vein and right superior vena cava identified intra-opeartively after left lobe of thymus removed

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