Air Entrainment after De-Airing

To the Editor,

A 70-year-old lady presented with progressively increasing exertional dyspnea and on clinical work up her transthoracic echocardiography revealed that she had severe aortic stenosis with a mean gradient of 45 mm Hg, a left ventricular ejection fraction of

60%, severe tricuspid regurgitation, moderate aortic regurgitation (AR), and a calculated pulmonary artery systolic pressure of 55 mm Hg. She underwent conventional surgical aortic valve replacement with a 21-mm Medtronic Avalus 400 bioprosthetic valve and tricuspid annuloplasty with 32-mm Carpentier–Edwards Physio Tricuspid ring on cardiopulmonary bypass (CPB). The left ventricle was vented through a right upper pulmonary vein (RUPV) vent during CPB. While weaning from CPB, meticulous de-airing was done with continuous transesophageal echocardiography (TEE) monitoring. Later on the RUPV vent was removed and the heart was allowed to eject with continued aortic root venting. After ensuring complete deairing on TEE, and acceptable hemodynamics, the patient was separated from CPB and the aortic root vent was removed. Two to three minutes later, the TEE showed sudden gush of air bubbles in the left atrium; the origin of which was traced to the RUPV. When entrainment of air was seen to increase with each cardiac contraction [Video 1], the patient was put in the steep Trendelenburg position and aortic root venting was restarted with a suction flow of 250–300 ml/min. No changes in the ECG were observed. TEE retraced the direction and location of air entrainment to the right upper pulmonary vein and on further inspection, the surgeon found that the suture over the RUPV vent site had loosened and was bleeding. Extra purse string sutures were placed over that area and which controlled the air entrainment completely [Video 2].

In the immediate postoperative period, the patient was found to have dense left-sided hemiplegia and plain CT scan of brain on first postoperative day (POD) showed an ill-defined hypodense lesion in the right suprainsular area (above lateral sulcus) and basal ganglia. On the second POD, even though the dense hemiplegia persisted, the patient was weaned from ventilator and extubated because she was fully conscious, oriented, and breathing spontaneously with good protective airway reflexes. On the second POD night, she had an episode of generalized tonic clonic seizure which was controlled with intravenous midazolam and levetiracetam. On third POD, the patient's weakness improved remarkably with only a minimal weakness of the left upper limb. On fourth POD, she regained her power completely and was discharged from the postoperative intensive care unit. Repeat CT brain did not show any interval change. Eventually, she was discharged from the hospital without any neurological deficits on the eighth POD and advised to continue Tab levetiracetam 500 mg for 3 months.

TEE plays a crucial role in ensuring adequate deairing before the weaning of CPB. However, complete removal of air from cardiac chambers is not always possible by standard methods and delayed release of air trapped within the chambers or pulmonary veins may still occur.[1] Although the interval between the aortic cross clamp release and termination of CPB is more susceptible for air embolism, significant intracardiac air may still be detected later either due to the release of trapped air or fresh entrainment, as was seen in this patient. Some of the reported causes of air embolism include inadvertent reversal of left ventricular vent suction tubing and air being sucked into the left atrium when cardiotomy suction tube was wedged deep inside the pulmonary artery.[2] Generally symptoms of cerebral air embolism are nonspecific and includes confusion, altered mental status, seizures, and focal neurological deficits. CT scan usually reveals hypodense lesions with sulcal effacement and loss of gray-white matter differentiation.[3] Immediate interventions in this scenario are directed towards preventing further air entry in the circulation and reducing the volume of air entrained. Treatment strategies include 100% oxygen administration, steep Trendelenburg position of the patient, maintaining supranormal blood pressure and cardiac output, retrograde cerebral perfusion, hyperbaric oxygen therapy, and prophylactic lidocaine.[4]

CONCLUSION

Continued vigilant TEE monitoring is essential during weaning from CPB and in this case it helped in retracing the anatomical location responsible for air entrainment.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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Submitted: 07-Apr-2020 **Revised:** 29-May-2020
Accepted: 21-Jun-2020 **Published:** 18-Oct-2021

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Videos Available on: www.annals.in

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Quick Response Code:

Website: www.annals.in

DOI: 10.4103/aca.ACA_68_20

How to cite this article: Desai PM, Shabadi RV, Chengode S. Air entrainment after De-airing. Ann Card Anaesth 2021;24:507-9.