A rare case of aorta-right atrial tunnel demonstrated on coronary computed tomography angiography

TO THE EDITOR: Anomalies of coronary arteries (CAs) are an uncommon cause of chest pain and can lead to sudden death.^[11] In aorta-right atrial tunnel (ARAT), an abnormal extracardiac tubular communication exists between the ascending aorta and the right atrium (RA).^[2] The embryological pathogenesis of ARAT is not certain, but congenital deficiency of aortic medial elastic lamina is a possible cause. Patients can be asymptomatic, have precordial murmurs, or even develop congestive heart failure.^[3] For decades, premorbid diagnosis of CA anomalies has been made with conventional angiography, but retrospectively gated coronary computed tomography angiography (CTA) is an accurate, quick and non-invasive alternative for diagnosis and treatment planning.^[4]

A 4-year-old boy presented with retrosternal chest pain and paroxysmal breathlessness for 2 years, which had become worse over the past week. There was no history of cyanotic spells, failure to thrive, fever, cough, haemoptysis, syncopal attacks, pedal oedema or easy fatigability. On two-dimensional echocardiography (2D echo), an abnormal channel was noted behind the aorta arising from the non-coronary sinus, showing an arterial waveform on colour Doppler ultrasound associated with continuous restrictive flow in the RA (Fig. 1 and Fig. 2 (Supplementary file: https://www.samedical.org/file/2017)). The interatrial and interventricular septa were intact. These findings prompted three differential diagnoses: aneurysmal dilatation of the left coronary artery (LCA) arising from the non-coronary cusp with a retro-aortic course, an LCA-RA fistula, and ARAT.

For conclusive diagnosis, the child underwent CTA using a 128-section multidetector CT scanner (Philips Medical Systems, India). A dilated vessel was seen arising just above the left coronary sinus (Fig. 3 - Supplementary file: https://www.samedical.org/file/2016), coursing posteriorly in the aorta-left atrial groove (Fig. 4 - Supplementary file: https://www.samedical.org/file/2018) and interatrial groove, measuring 12 mm at the ostium and 7 mm in the mid-portion. It bifurcated 1.3 cm proximal to termination, with the larger channel opening into the RA, 6 mm below the superior cavoatrial junction (Fig. 5 - Supplementary file: https://www.samedical.org/file/2019), and the smaller channel terminating at the superior cavoatrial junction. The left anterior descending artery and LCA appeared to originate ~1.1 cm distal to its origin. A diagnosis of ARAT was considered more likely than coronary-cameral fistula (CCF).

Mild cardiomegaly with normal situs, and atrioventricular concordance with normal tricuspid and mitral valves and venous connections were noted. Features of pulmonary hypertension in the form of dilation of the main pulmonary artery and its visualised branches and mosaic attenuation in the lungs were seen. Digital subtraction angiography (DSA) confirmed ARAT (Fig. 6 - Supplementary file: https://www. samedical.org/file/2020).

The patient was prescribed oral diuretics for 5 months preoperatively and underwent intracardiac repair. Intraoperatively ARAT was confirmed, the RA was opened and the fistula opening was closed. The



Fig. 1. Two-dimensional echocardiography, showing a channel (blue arrow) arising from the left coronary sinus of the aorta (blue star).

fistula track was traced from aorta to pulmonary artery and ligated. A postoperative 2D echo showed no flow across the previous tract. The patient was stable postoperatively and was discharged on oral diuretics. Written informed consent was obtained from the patient's guardian for publication of this case report and the accompanying images, which have been anonymised.

ARAT is thought to be caused by a congenital aortic medial elastic deficiency and weakness which, combined with high aortic pressure, leads to tunnel formation, draining proximally into the low-pressure RA.^[2,5] A posterior course in the left sinus tunnel and an anterior course in the right sinus tunnel are seen. However, this theory does not explain tunnels arising from the non-coronary sinus.^[2] ARAT causes a left-to-right shunt that over time leads to volume overload, pulmonary hypertension, congestive cardiac failure, a tunnel or sinus aneurysms, and rupture. Delayed diagnosis is associated with increased age-related surgical mortality.^[2]

Clinically, patients can be asymptomatic or present with breathlessness on exertion, fatigue, chest pain, and recurrent respiratory tract infections. On examination, a continuous murmur at the right parasternal border is noted. Findings on chest radiography are nonspecific, relating to chamber enlargement, pulmonary hypertension and cardiac failure.^[2]

On CTA, ARAT is seen as a dilated vessel arising from the aorta or CA, draining into one of the heart chambers.^[2] Origin above the sinotubular ridge differentiates ARAT from aneurysm of the sinus of Valsalva, and absence of myocardial branches differentiates it from a CCF.

ARAT can be diagnosed using 2D echo, coronary angiography or cardiac CTA. While high spatial and temporal resolution makes DSA the gold standard in imaging CA anomalies, CTA is easily available, cost-effective and non-invasive. It can also depict cardiac morphology, vessel abnormalities and their relationships. Simultaneously, cardiac CT can pick up pulmonary abnormalities. Perfusion CT data can deliver quantitative haemodynamic information, such as blood volume, blood flow, permeability surface-area product and mean transit time.^[6] While 2D echo can depict such findings, it is user dependent with a limited acoustic window for imaging all parts of the heart. Cardiac CTA is therefore an ideal imaging modality for CA abnormalities.

Treatment should be initiated as soon as the diagnosis is confirmed, to avoid complications. Asymptomatic ARAT can be supportively managed by use of diuretics and other medications to reduce cardiac afterload, with regular monitoring. However, symptoms inevitably worsen, and fistula closure is the definitive management.^[7] Curative options include simple ligation, or ligation with reimplantation of coronary ostium or coil embolisation in selected cases. Direct closure of the atrial opening is done with a patch of aortic origin or plication of the tunnel. If the origin of the CA is deep in the tunnel, it should be reimplanted with a part of the tunnel into the respective sinus of Valsalva.^[2]

In conclusion, ARAT is a rare congenital heart disease that is accurately diagnosed on CTA. CTA helps formulate management guidelines, which are dependent on the size and location of the tunnel, haemodynamic factors, and complications. It is ideal for follow-up of conservatively managed cases. Early diagnosis of ARAT improves prognosis and reduces postoperative morbidity.

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Tracheo-oesophageal fistula in a case of organophosphate poisoning

TO THE EDITOR: Tracheo-oesophageal (TO) fistula is an abnormal connection between the trachea and the oesophagus. The fistula can be congenital or acquired, developing as a result of malignant disease, infection, trauma and ruptured diverticuli.^[1]

Prolonged mechanical ventilation with an endotracheal or tracheostomy tube can result in a TO fistula.^[2] Factors that can lead to development of a fistula include duration of intubation, cuff pressure, the type of tube used for the procedure, poor nutrition, infection and extended use of steroids, while ingestion of a corrosive substance may be a coexisting cause of necrosis of the region. Food particles and fluid from the oesophagus can enter the trachea through the fistula, leading to infection, pneumonia, congestion, bronchial obstruction and respiratory distress. Before patients develop symptoms of bronchial infection, a cough reflex immediately after ingestion of food or water is commonly noted in the initial phase. The severity of the symptoms depends on the width and length of the fistulous connection.

Investigation with bronchoscopy and contrast-enhanced computed tomography (CT) is required to exclude the possibility of a TO fistula.^[1]

A 55-year-old woman presented with complaints of coughing after ingestion of water and food for 2 months, together with difficulty in swallowing. She had ingested organophosphate 3 months previously, after which she was intubated for a prolonged period of 20 days – 12 days in the intensive care unit and 8 days in the surgical ward (further details of the intubation are not available, as it was done elsewhere). On discharge, no abnormality was noted on clinical examination. She had had no similar symptoms in the past. Upper gastrointestinal endoscopy revealed a fistulous opening of ~6 mm, ~18 cm from the incisors, with the area epithelialised. No ulcer or malignant lesion was present (Fig. 1). The rest of the oesophagus and the stomach and duodenum were normal. Results of all other routine examinations were normal.

Endoscopy was followed by a plain CT scan of the neck, which showed a TO fistula measuring $6 \times 4 \times 6$ mm (craniocaudal ×