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Case Report

Incidental retroaortic left innominate vein in adult patient

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ABSTRACT

Retro-aortic left innominate vein is a rare vascular abnormality, usually associated with congenital heart disease. Here we report a case of isolated retro-aortic left innominate vein in an adult female.

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Introduction

Retroaortic left innominate vein (RA LIV) is a rare vascular abnormality, usually associated with congenital heart disease (CHD). Here, we report a case of isolated RA LIV in an adult female.

the [Figure 1](#), the LIV courses underneath the aortic arch, posterior to the ascending aorta. It then joins the normal right brachiocephalic vein to form a normal right-sided superior vena cava (SVC). The proximal LIV is unremarkable, formed by the confluence of the left subclavian and left internal jugular veins.

Case presentation

RA LIV was incidentally identified on a contrast-enhanced computed tomography scan of the thorax performed for staging of a recently diagnosed colon cancer in a 48-year-old female patient. The patient had no known history of CHD or any vascular abnormality. The contrast-enhanced computed tomography showed normal heart and great arteries, with a left aortic arch demonstrating normal branching. As shown in

Discussion

Normally, the LIV runs anterior to the ascending aorta and joins the right brachiocephalic vein to form the SVC. Retro-aortic, sometimes also called subaortic or aberrant, LIV is a rare anatomical variant, usually seen in association with CHD and abnormalities of the aortic arch.

In one of the largest retrospective studies of CHD patients evaluated with CT chest, RA LIV was identified

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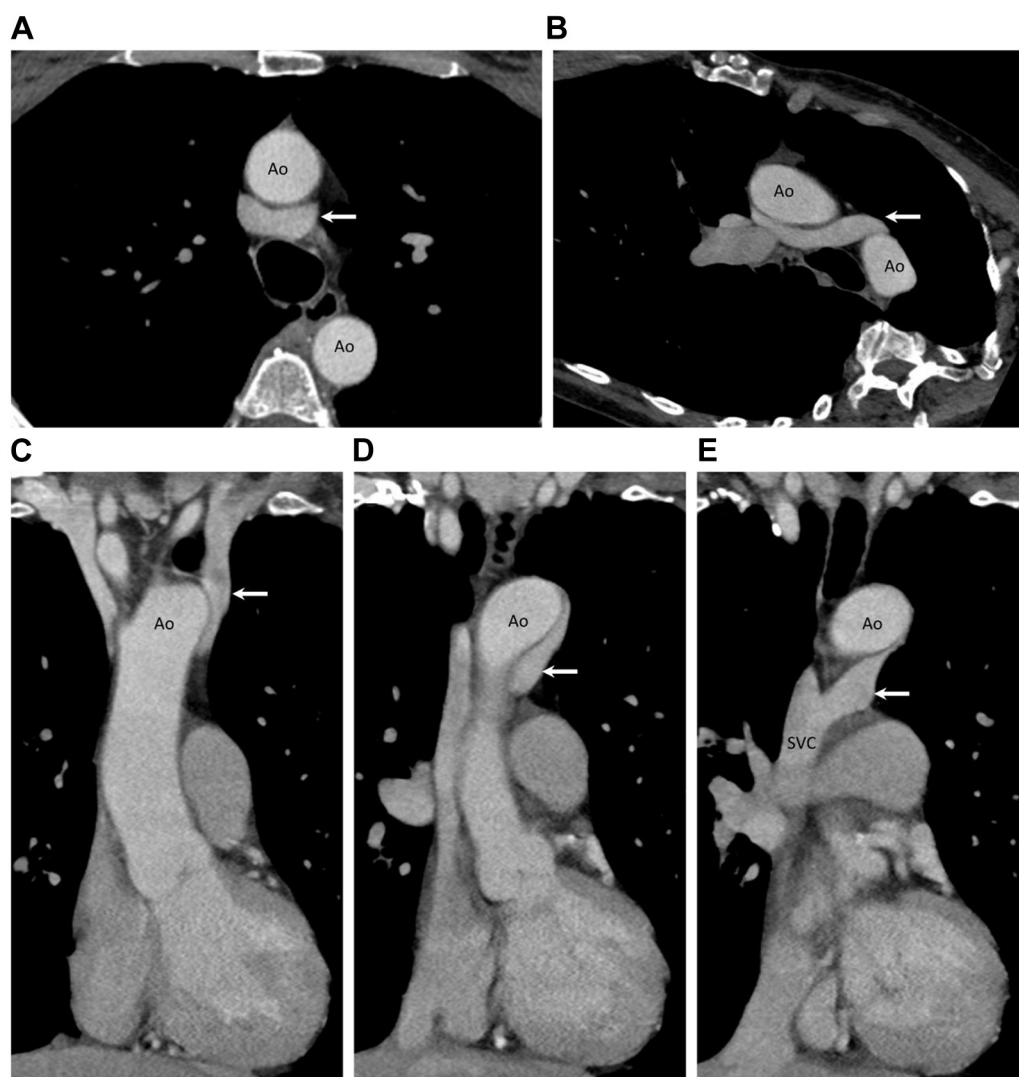


Fig. 1 – Contrast-enhanced axial CT of the chest in (A) axial, (B) axial oblique, (C–E) coronal, (F) sagittal, and (G) sagittal oblique 3D reformations demonstrate aberrant retroaortic course of the left innominate vein (arrows). Ao, aorta; SVC, superior vena cava.

in 30 of 1812 patients (estimated prevalence of 1.7%) [1]. The congenital cardiac abnormalities associated with RA LIV in the above study were tetralogy of Fallot 77%, right atrial isomerism 13%, atrial septal defect 3.3%, ventricular septal defect 3.3%, and double outlet of right ventricle 3.3% [1]. Aortic anomalies (right sided and double aortic arches) were present in 73% of the patients with RA LIV [1].

Yet, in another retrospective study, RA LIV incidence in patients with CHD was found to be 0.57%, and the associated cardiovascular abnormalities were tetralogy of Fallot, ventricular septal defect with pulmonary atresia, truncus arteriosus, interrupted aortic arch, and cervical aortic arch [2].

Occurrence of an RA LIV in isolation, as in the case of our patient, is extremely rare. Its prevalence is estimated at 0.02% [2]. There is only a handful of reports in the English language medical literature of RA LIV in individuals without CHD or aortic arch abnormalities [2–5].

During embryogenesis, systemic veins develop from paired anterior and posterior cardinal veins, which form by week 4. The anterior and posterior cardinal veins join on each side of the embryo to form right and left common cardinal veins that drain into the sinus venosus (precursor of the smooth part of the right atrium) (Fig. 2A) [6]. The anterior cardinal veins extend cranially to the junctions of the internal jugular and subclavian veins on each side. Subsequently, most of the left anterior cardinal vein disappears, replaced by a newly developed transverse anastomotic channel (future LIV), which directs the venous return from the left arm, head, and neck to the right anterior cardinal vein (which together with the right common cardinal vein become the SVC). The left common cardinal vein becomes the coronary sinus, and the adjacent portion of the left anterior cardinal vein becomes the oblique vein of Marshall (Fig. 2B).

The mechanism of formation of the RA LIV remains unknown. Some authors have postulated that aberrant

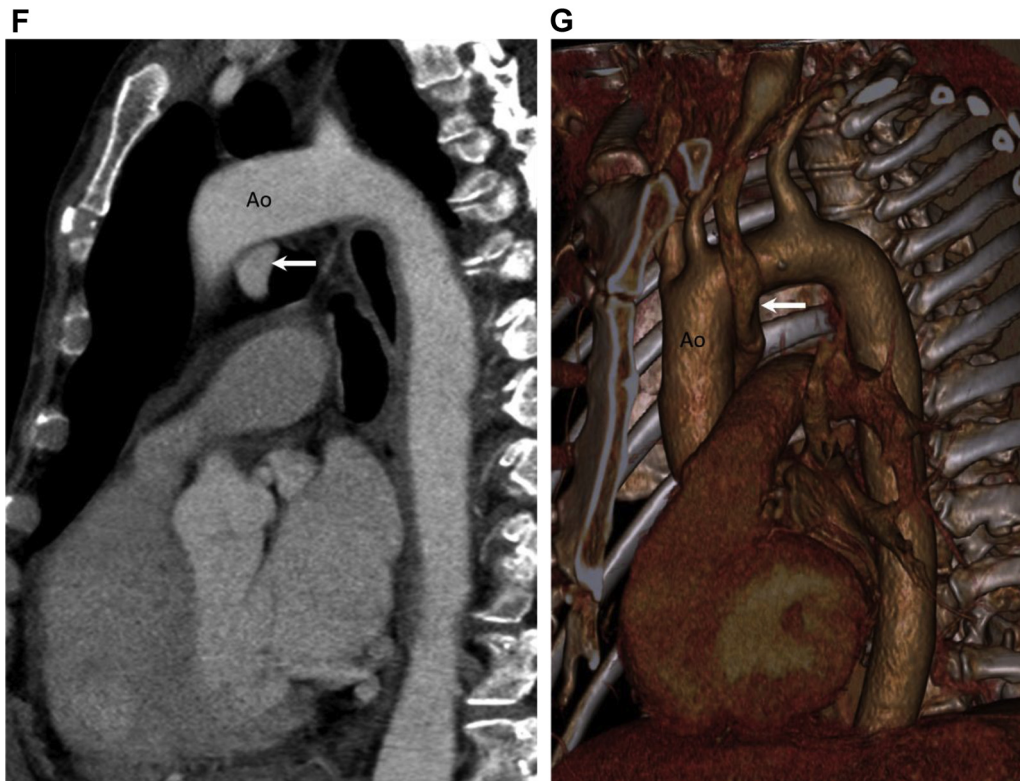


Fig. 1 – (continued).

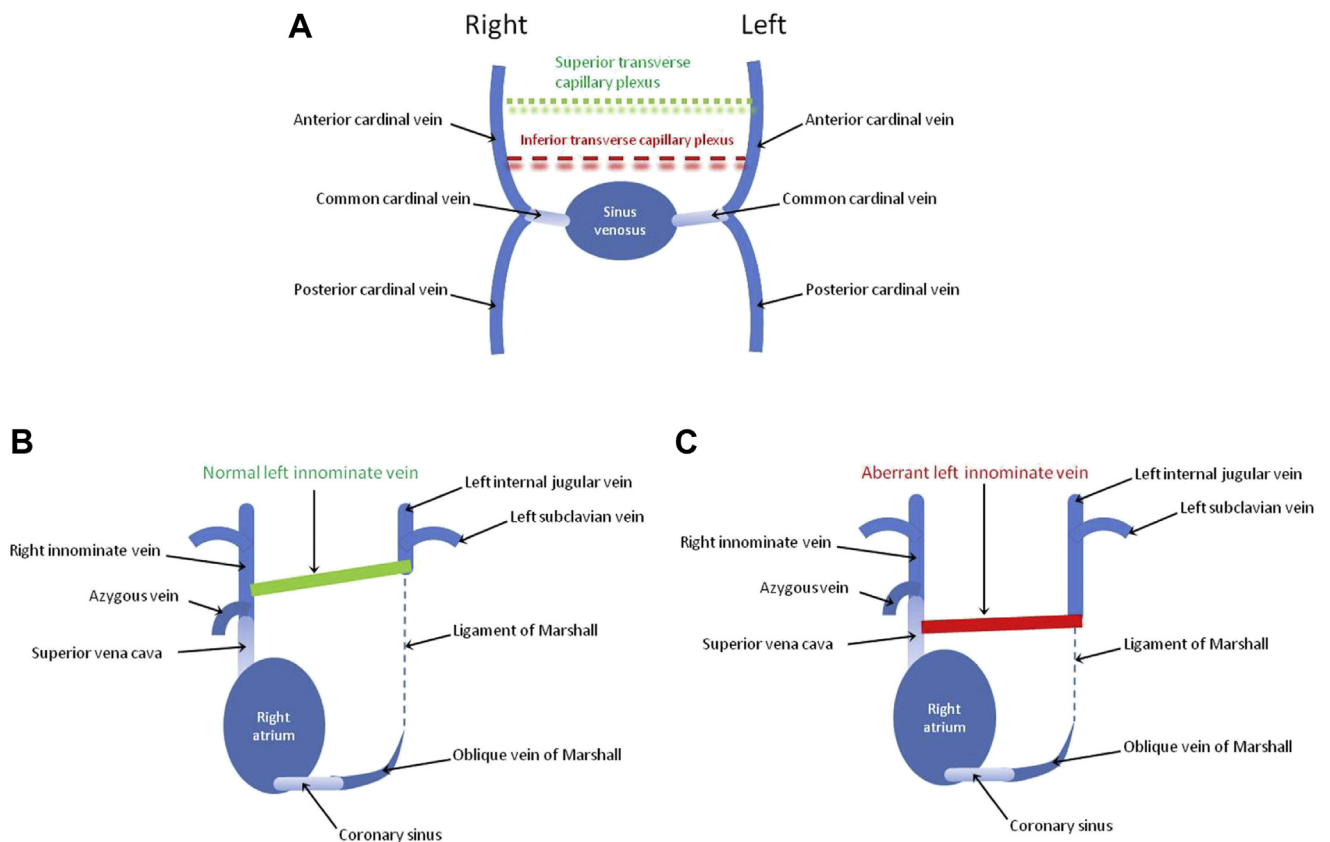


Fig. 2 – (A) Schematic representation of the precursors of the major systemic veins around fourth week of embryogenesis. (B) Pathway leading to the development of a normal left innominate vein from the superior transverse capillary plexus. (C) Pathway resulting in an aberrant left innominate vein.

innominate vein may arise as a result of failure of the usual transverse anastomosis development, and subsequent opening up of an alternative anastomotic pathway within the capillary plexus of that region [6]. In the later case, the RA LIV arises from a more caudal capillary network, below the arch and posterior to the ascending aorta (Fig. 2C). Given the relatively high incidence of RA LIV in patients with congenital conotruncal and aortic arch abnormalities, and its extreme rarity as an isolated finding, several authors have postulated that the abnormal development of the great arteries somehow interferes with the normal development of the superior transverse anastomotic channel in favor of the inferior transverse anastomosis [1].

Although in itself RA LIV is asymptomatic, its recognition during clinical investigation should raise the possibility of an association with other malformations, especially right aortic arch, cardiac septal defects, and conotruncal abnormalities. Recognition of the RA LIV, when present, is also important, as it may have implications for intrathoracic surgical planning, endovascular procedures, placement of central venous lines, and transvenous pacemakers.

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