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PICTORIAL ESSAY

Extracardiac complications of the Fontan circuit

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Abstract The Fontan operation is the primary surgical procedure used in the palliation of patients with univentricular cardiac physiology. With improved survival of children with congenital heart disease, long-term complications of the Fontan circuit are being encountered more frequently. Radiologists are more likely to see both the cardiac and extracardiac complications of the Fontan circuit. Awareness of the common extracardiac complications in children with failing Fontan circuits will aid the radiologist in making the appropriate diagnosis and guide the cardiologist caring for these patients.

Keywords Fontan procedure · Cardiac cirrhosis · Protein-losing enteropathy · Plastic bronchitis

Introduction

The Fontan circuit is a palliative surgical procedure that results in routing the systemic venous return to the pulmonary arteries without passing through a ventricular chamber. Several cardiac malformations such as tricuspid atresia, pulmonary atresia with intact ventricular septum, double inlet ventricle and hypoplastic left heart syndrome are characterized by the existence of only one functional ventricle. Left untreated, this single ventricle has to maintain both the systemic and pulmonary blood circulations. This results in chronic volume overload to the single ventricle and arterial desaturation. In 1971, Francis Fontan [1] reported a new approach for the surgical management of tricuspid atresia. Fontan performed a classic Glenn shunt, in which the superior cavoatrial junction was ligated and the superior vena cava (SVC) connected to the right pulmonary artery (PA), which was divided from the main PA. In addition, a conduit was created between the right atrium and the left PA. This routed the SVC blood into the right pulmonary circulation and the inferior vena cava (IVC) blood into the left pulmonary circulation, bypassing the intervening ventricle [1]. Since the original description, the Fontan procedure has undergone multiple modifications. Most adults have a modified classic Fontan, which consists of direct anastomosis between the right atrial appendage and pulmonary artery (Fig. 1). In recent years, the Fontan circulation is accomplished by using a bidirectional Glenn shunt (end-to-side anastomosis between the SVC and right PA) that routes SVC blood to both lungs, and a lateral tunnel or extracardiac conduit Fontan to direct blood from the IVC to the pulmonary circulation [2, 3]. In the lateral tunnel Fontan, a prosthetic tunnel is created within the right atrium. The inferior aspect of the tunnel is anastomosed to the IVC, and the superior aspect is anastomosed to the PA. With an extracardiac Fontan, a polytetrafluoroethylene conduit is created between the IVC, which is separated from the right atrium, to the right PA outside the

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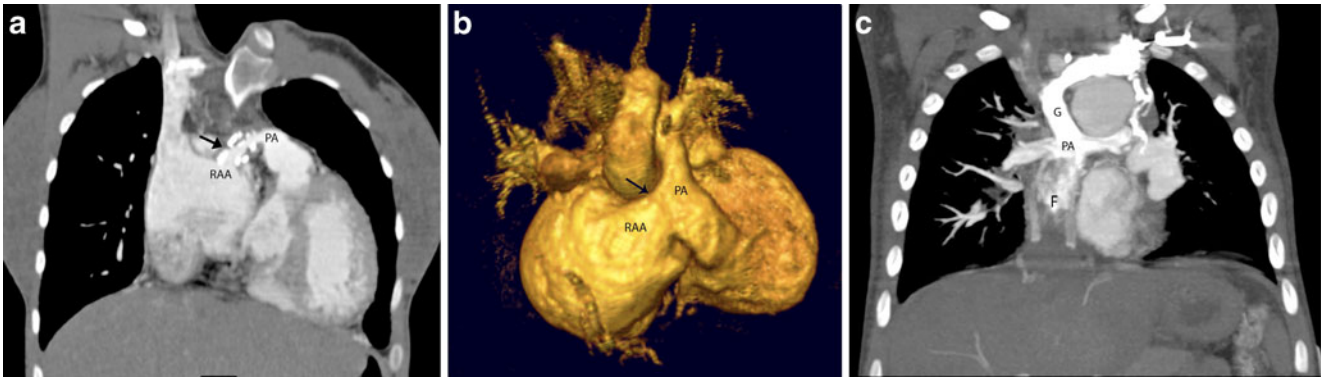

Fig. 1 Fontan anatomy. Coronal multiplanar (**a**) and 3-D reconstructed (**b**) images show the classic Fontan circuit with a direct anastomosis (*arrow*) between the right atrial appendage (*RAA*) and the pulmonary artery (*PA*). **c** The most recent modification of the Fontan technique is the extracardiac conduit (*F*) between the inferior vena cava and right pulmonary artery (*PA*). All systemic venous return is routed into the pulmonary circulation through the extracardiac conduit and the superior cavopulmonary anastomosis (Glenn shunt = *G*)

right atrium (Fig. 1). Both techniques result in smooth laminar blood flow from the IVC to the PAs.

Survival rates of Fontan patients have significantly improved since its first description with a reported 20-year survival of 85% [4]. As the number of survivors increases, health care providers are more likely to encounter the long-term complications of the failing Fontan physiology. Often times, these complications present outside of a congenital heart disease clinic. In a follow-up of survivors, 11% were found to have clinically significant morbidity, including protein-losing enteropathy, liver dysfunction or stroke, at a median age of 8 years old [5]. In this article, we review the extracardiac complications that a radiologist may encounter when imaging a child with a Fontan circuit.

Hepatic complications

The absence of a right ventricular pumping chamber in the Fontan circuit results in chronically elevated pressures in the systemic venous system. The net effect on the liver is passive congestion, dilation of hepatic sinusoids and arterialization of hepatic flow. Eventually, the chronic hepatic congestion results in centrilobular necrosis, fibrosis and, ultimately, cardiac cirrhosis. Early clinical manifestations of passive hepatic congestion include coagulopathy and cholestasis, though children are often asymptomatic with consequences of passive hepatic congestion first detected on imaging [6].

CT findings of passive hepatic congestion in Fontan patients include a reticular enhancement pattern in the portal phase, which is typically more marked in the periphery of the liver (zonal distribution) (Fig. 2) [7, 8]. This appearance is similar to the hepatic congestion seen in chronic obstructive venous outflow conditions such as Budd-Chiari syndrome and constrictive pericarditis. The chronic passive venous congestion results in arterialization of the hepatic blood flow, which can lead to the development of regenerative nodules. The regenerative nodules seen in congestive hepatopathy have an imaging appearance similar to that of focal nodular hyperplasia of the liver. The lesions are typically iso-intense to liver on precontrast images, intensely enhancing on arterial phase, and isointense to liver parenchyma on portal and equilibrium phase images (Fig. 3). Chronic passive hepatic venous congestion can also result in the formation of intra- and extrahepatic venovenous collaterals (Fig. 4).

There are several reported cases of hepatocellular carcinoma (HCC) developing in children with cardiac cirrhosis secondary to a failed Fontan [8–10]. So, the regenerative nodules need to be differentiated from HCC on imaging. Regenerative nodules become isointense to liver parenchyma on portal/equilibrium phase images and

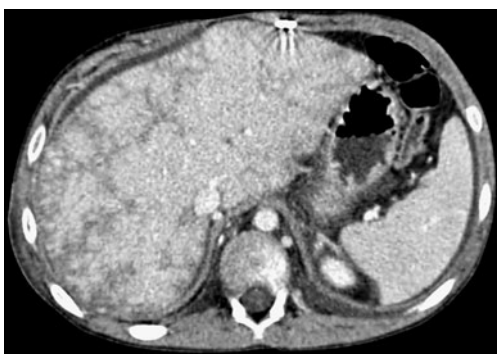

Fig. 2 Passive hepatic congestion secondary to Fontan circuit in a 9-year-old boy with hypoplastic left heart syndrome, status post Fontan procedure. Contrast-enhanced CT of the abdomen shows a reticular enhancement pattern of the liver, most prominent in the liver periphery (zonal distribution). This is generally most prominent in the portal venous phase

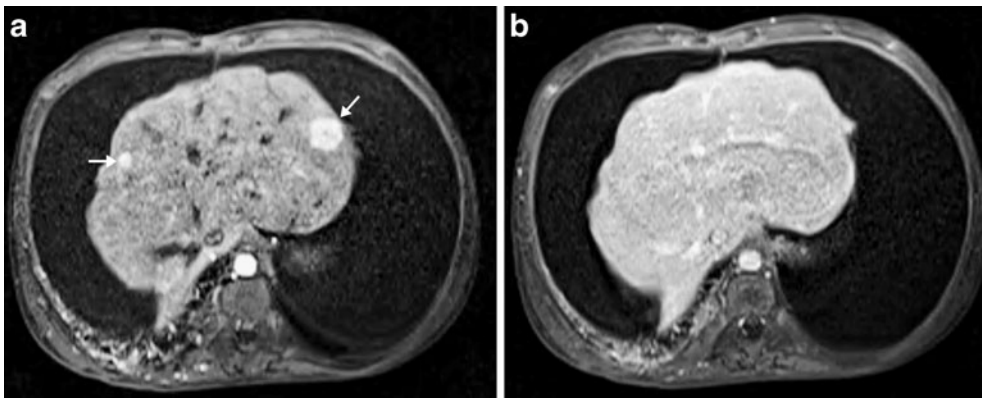

Fig. 3 Cardiac cirrhosis with regenerative nodules in a 15-year-old girl status post Fontan procedure for unbalanced AV canal, D-transposition of great vessels and pulmonary atresia. **a, b** Dynamic breath-hold gradient-echo images with Multihance® through the liver show a cirrhotic liver. Two arterial enhancing lesions are present (*arrows*), which become isointense with liver parenchyma on portal venous phase (**b**), consistent with regenerative nodules

retain contrast on delayed images obtained 1–3 h after injection of gadolinium-BOPTA (Multihance®) or 30 min after injection of gadoxetate disodium (Eovist®) [11]. In contrast, HCC becomes hypointense on delayed images, in keeping with washout, and does not show retention of contrast on delayed images obtained with Multihance® or Eovist®.

Protein-losing enteropathy

Protein-losing enteropathy (PLE) is a rare but life-threatening complication after the Fontan operation. PLE is characterized by hypoalbuminemia secondary to the excessive loss of proteins from the intestinal lumen. Its incidence in children with the Fontan circulation has been estimated to be as high as 13.4% [12]. In a multicenter study of 114 children with PLE after the Fontan procedure, the median age at diagnosis was 11.7 years, with onset of PLE as early as 1 month to as late as nearly two decades after the Fontan procedure [13]. The etiology of PLE remains unclear, and proposed theories are as follows [14, 15]. The lack of a forward pumping chamber between the systemic venous return and the pulmonary arteries results in chronically elevated systemic venous pressures including the splanchnic circulation. This results in increased pressures in the enteric lymphatic system, intestinal congestion, and leakage of proteins and lymphocytes from the dilated lymphatics. In addition, the low cardiac output state predisposes the child to mesenteric ischemia and subsequent intestinal mucosal injury and protein leakage (Fig. 5) [15].

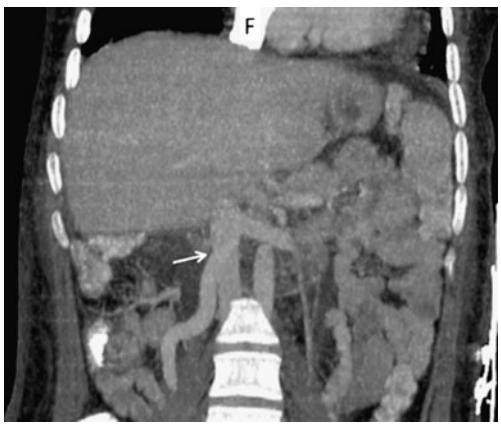

Fig. 4 Portosystemic collateral secondary to passive hepatic congestion in a 21-year-old woman with hypoplastic left heart, post Fontan procedure. Coronal thick slab (2 cm) maximum-intensity projection (MIP) image from a CT of the chest, abdomen, pelvis performed as part of a precardiac transplant evaluation reveals a portosystemic shunt draining into the inferior vena cava at the level of the renal veins (*arrow*). This collateral was arising from the superior mesenteric vein (not shown)

Clinical manifestations of PLE include peripheral edema, ascites, pleural/pericardial effusions secondary to hypoalbuminemia, diarrhea secondary to malabsorption and hypocalcemia (Fig. 6). In more severe forms of PLE, children can develop immunodeficiency from hypogammaglobulinemia and loss of lymphocytes in the intestine, hypercoagulopathy due to loss of clotting factors, and chronic malnutrition. Laboratory tests reveal low total blood serum protein <5.5 g/dl and low albumin <3.5 g/dl [15]. The development of PLE can result in significant morbidity and mortality with a reported 5-year survival after diagnosis of PLE at 50% of children [13].

Plastic bronchitis

Plastic bronchitis is a rare but serious complication of the Fontan circuit, occurring in less than 1–2% of children. Plastic bronchitis is characterized by the formation of large gelatinous branching airway casts. These casts are large and

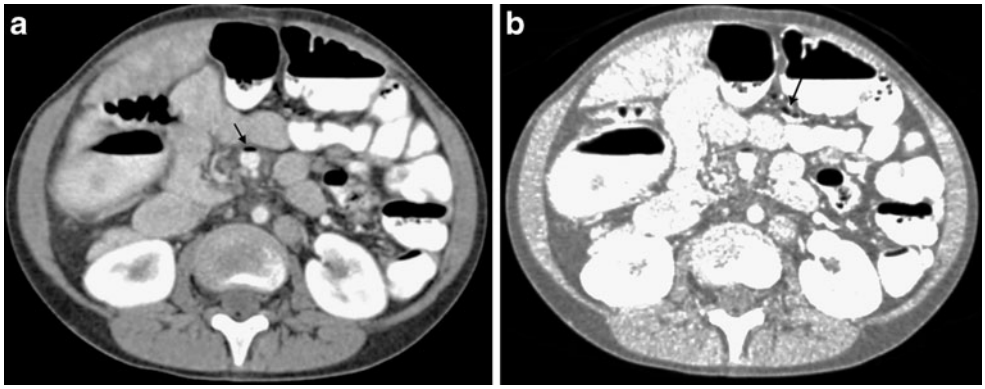

Fig. 5 Protein-losing enteropathy in an 8-year-old girl with a Fontan shunt for single ventricle physiology with known debilitating protein-losing enteropathy. Contrast-enhanced CT performed as part of routine precardiac transplant work-up shows air (*arrow*) in the superior mesenteric vein (**a**) and its tributaries (**b**). There was no pneumatosis and the girl had no acute abdominal symptoms at the time of the exam. The mesenteric air resolved spontaneously on a follow-up scan

more cohesive than those seen in ordinary mucus plugging. Plastic bronchitis can occur in a variety of other conditions including asthma, allergic bronchopulmonary aspergillosis, cystic fibrosis, lymphangiomatosis and sickle cell disease [16]. The casts of plastic bronchitis seen in a failing Fontan circuit are acellular mucin casts as compared to the eosinophilic inflammatory casts seen in the other conditions [16, 17]. The proposed pathogenesis of plastic bronchitis in children with the Fontan circuit includes hypersecretion of airway mucus, abnormalities of pulmonary lymphatic drainage and poor cardiac output. The clinical manifestations include chronic respiratory symptoms such as persistent cough and wheezing. Chest radiographic findings include segmental collapse, obstructive hyperinflation, areas of patchy consolidation and abrupt termination of a bronchus (Fig. 7). CT can demonstrate bronchial casts causing partial or complete obstruction of the central airways without associated bronchiectasis [18]. The diagnosis is confirmed by bronchoscopy demonstrating thick casts in the airway or by expectoration of the casts.

Right-to-left shunting

Worsening hypoxemia in Fontan patients can be secondary to right-to-left shunting caused by pulmonary arteriovenous malformations or systemic vein to pulmonary vein collaterals (Fig. 8). Right-to-left shunts can cause systemic arterial desaturation resulting in late clinical deterioration in children treated with Fontan operations. Though pulmonary AVMs have been described more commonly post-Glenn procedure, they are known to occur in Fontan cases as well especially if the hepatic outflow is asymmetrically distributed into the lungs [19]. The development of pulmonary AVMs has been attributed to the lack of pulsatile flow in the pulmonary bed secondary to the absence of a pumping

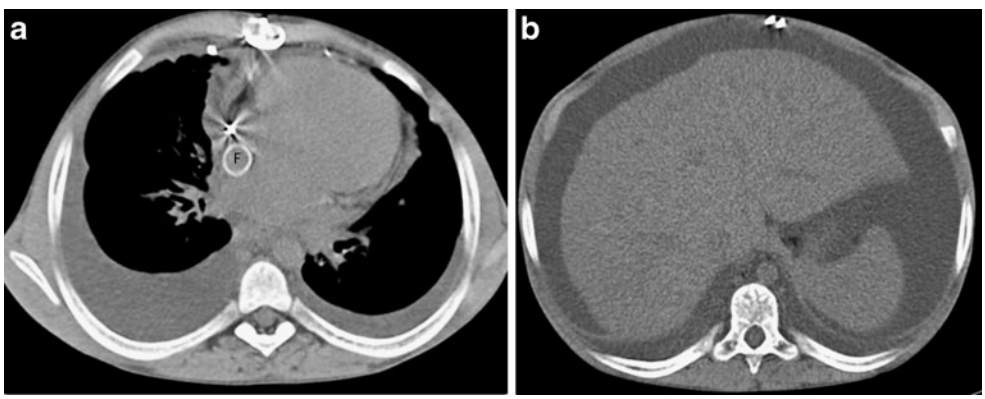

Fig. 6 Protein-losing enteropathy in a 12-year-old boy post failed Fontan for hypoplastic left heart, awaiting cardiac transplant. **a, b** Non-contrast CT of the chest and abdomen shows an extracardiac Fontan conduit (*F*). Bilateral pleural effusions, small pericardial and massive ascites are present. The liver is cirrhotic (cardiac cirrhosis). The boy had a history of frequent stooling, hypocalcemia 7.7 mg/dl (normal 8.6–10.3 mg/dl), hypoalbuminemia 2.4 g/dl (normal 3.2–5.0 g/dl) and hypoproteinemia 4.7 g/dl (normal 6.5–8.5 g/dl) consistent with a diagnosis of protein-losing enteropathy

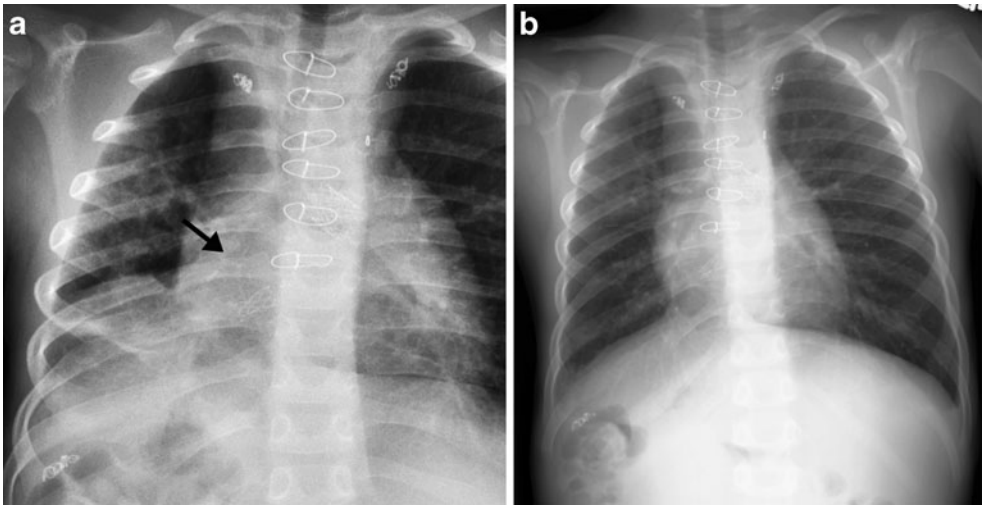

Fig. 7 Plastic bronchitis. A 9-year-old with complex congenital heart disease, 6 years after Fontan procedure, presented with recurrent cough and expectoration. **a** AP radiograph of chest shows collapse of the right middle and lower lobes with abrupt termination of the bronchus intermedius (*arrow*). A large bronchial cast was removed at bronchoscopy with follow-up radiograph (**b**) that shows re-expansion of the right middle and lower lobes

ventricle between the systemic venous return and the pulmonary bed, and the maldistribution of hepatic venous return to the lungs. The latter is supported by the following facts. Pulmonary AVMs are more likely to develop if the hepatic venous flow is directed exclusively into one lung (Fig. 8) [20]. Children with interrupted IVC

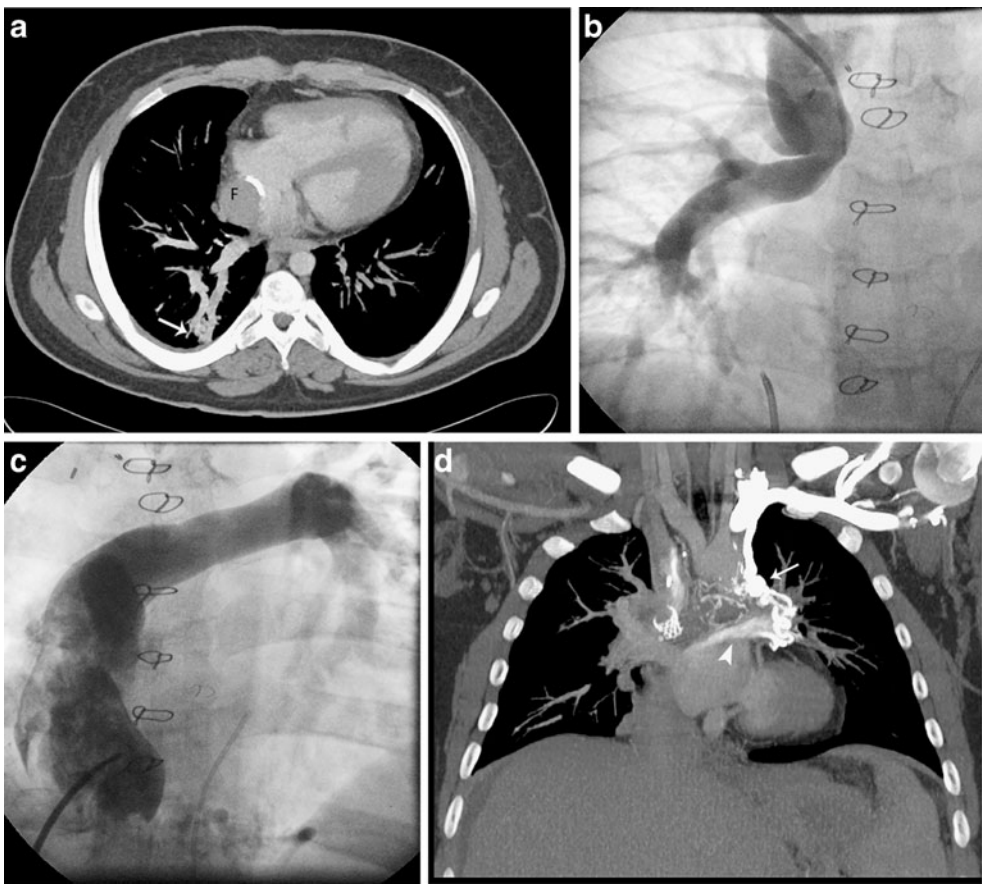

Fig. 8 Right-to-left shunting post Fontan procedure in a 16-year-old boy post Fontan for hypoplastic left heart. He presented with chest pain, shortness of breath and central cyanosis. **a** A 10-mm-thick slab projection from a contrast-enhanced CT of the chest shows an arteriovenous malformation in the right lung (*arrow*) with an extracardiac Fontan circuit (*F*). **b, c** Angiographic images show that the right lung is preferentially perfused by the Glenn shunt (superior vena cava to pulmonary artery anastomosis), while the left lung is preferentially perfused by the Fontan conduit. Diversion of hepatic effluent away from the right lung, predisposes the right lung to develop pulmonary arteriovenous malformations. **d** Coronal reconstructed 10-mm-thick slab projection image also shows a second right-to-left shunt (*arrow*) caused by a systemic to pulmonary vein shunt with dense contrast returning to the left atrium (*arrowhead*) from the left upper extremity (**b** and **c** courtesy of David Balzer, MD, St. Louis, MO)

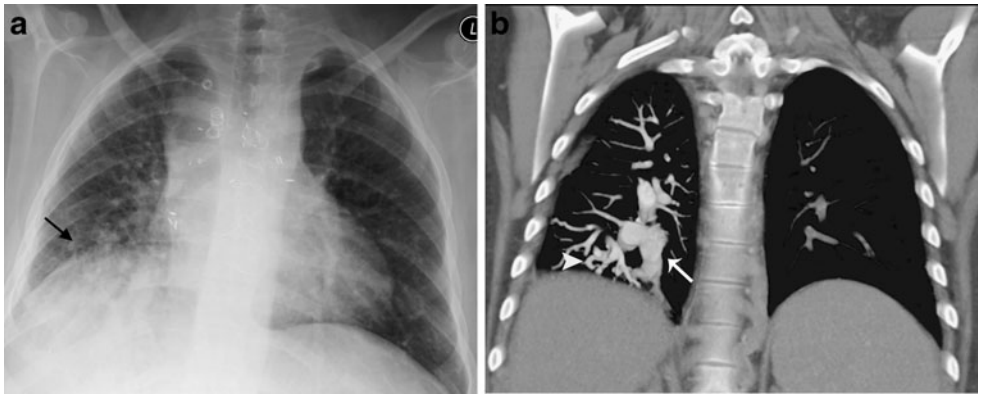

Fig. 9 Right-to-left shunt secondary to pulmonary arteriovenous malformation in a 22-year-old man with double outlet right ventricle, post Glenn shunt to right pulmonary artery and Fontan shunt to the left pulmonary artery. This results in diversion of the hepatic effluent away from the right lung. **a** Chest radiograph shows increased vascularity at the right lung base with large vessels (*arrow*). **b** Coronal reconstructed 20-mm-thick slab projection from a contrast-enhanced chest CT confirms a tangle of vessels at the right lung base (*arrowhead*) with large draining veins (*arrow*), consistent with arteriovenous malformation

with azygos or hemiazygos continuation to the SVC are known to have a high risk of developing AVMs, after undergoing a superior cavopulmonary anastomosis (Kawashima procedure) [21]. This procedure routes most of the systemic venous return to the pulmonary circulation, while the hepatic venous return remains to the right atrium. [20]. The pulmonary AVMs seen in this population are similar to the hepatopulmonary syndrome seen in children with end-stage liver disease, where an unidentified factor produced by the normal liver does not reach the lung.

Pulmonary AVMs may manifest on chest radiographs as an area of increased pulmonary vascular markings or nodular/reticulonodular opacities (Fig. 9). CT findings include the presence of abnormally enlarged pulmonary vessels extending to the periphery of the lung with the pulmonary AVM forming a small tangle of vessels (Figs. 8 and 9). The use of thick-slab (4–20 mm) maximum-intensity projections (MIPs) can aid in detecting and characterizing AVMs on CT.

Venovenous collateral vessels can develop between the venous drainage of the upper extremities and the pulmonary veins or the left atrium (Figs. 8 and 10) [22]. The development of systemic to pulmonary vein collaterals in the absence of any discrete obstruction of the innominate vein or superior vena cava suggests that they develop secondary to elevated central venous pressures [23].

Late thromboembolic complications

Thromboembolic events account for significant morbidity and mortality in the setting of the Fontan circulation (Figs. 11 and 12). The incidence of such complications has been reported at 3–20% [24]. The increased risk of thromboembolic complications in this patient population is thought to be multifactorial. Ligation of the pulmonary trunk results in a blind cul-de-sac distal to the pulmonary valve where thrombi can form. The cavopulmonary conduit

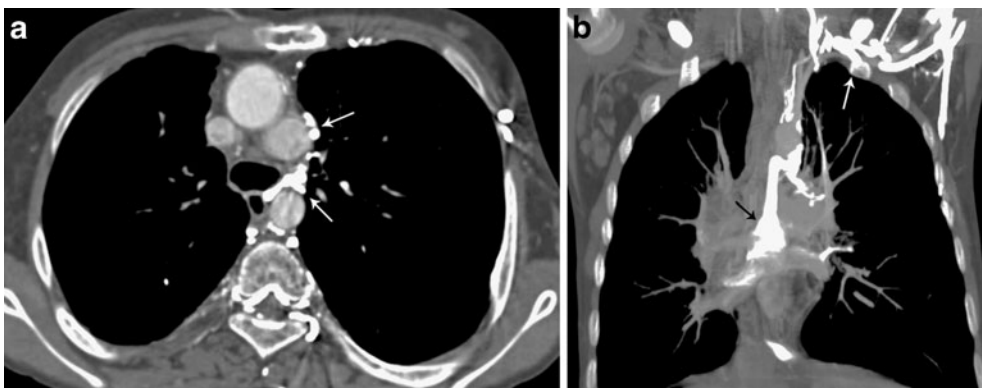

Fig. 10 Right-to-left shunting caused by systemic to pulmonary venovenous collaterals in a 15-year-old girl with Fontan and Glenn shunt for double outlet right ventricle. **a** Axial contrast-enhanced CT shows densely opacified venous collaterals in the mediastinum (*arrows*). **b** Coronal reformatted 10-mm-thick slab projection image confirms right-to-left shunting caused by mediastinal collaterals draining the veins of the left upper extremity (*white arrow*) into the left atrium (*black arrow*)

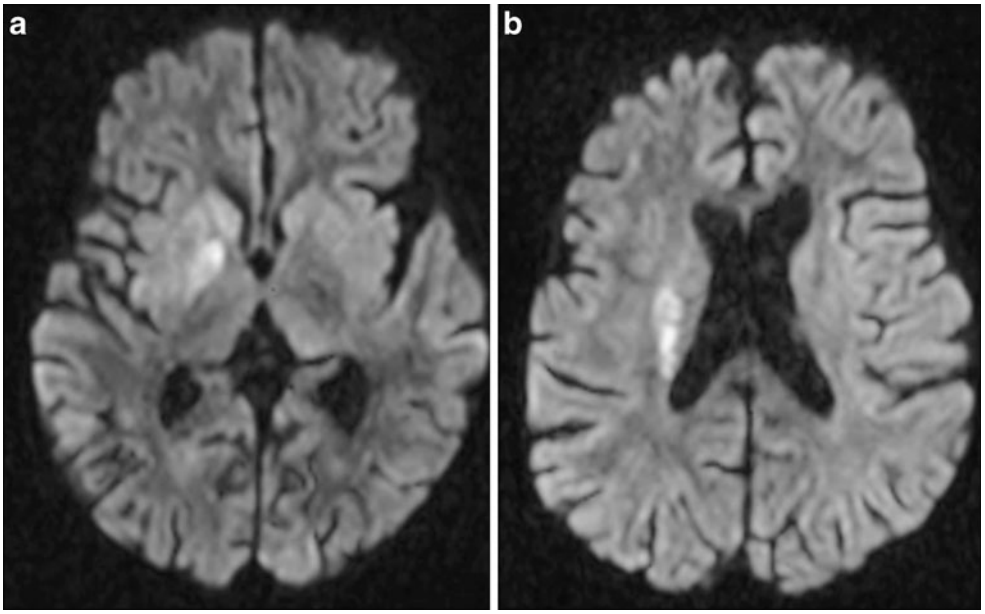

Fig. 11 Thromboembolic complications. A 4-year-old boy, post Fontan circuit for hypoplastic left heart, presented with left arm weakness. **a, b** Diffusion-weighted MR images show infarcts in the right basal ganglia and corona radiata likely embolic in etiology

is thrombogenic due to low flow, venous stasis and the presence of thrombogenic material. The classic Fontan circuit is associated with atrial dilation and stasis, increasing the risk of atrial thrombus. Though the lateral tunnel and extracardiac conduit Fontan modifications result in better streaming of blood flow between the IVC and PA, current literature suggests that the risk of thromboembolism is independent of the type of Fontan operation [25]. The development of right-to-left shunts can lead to systemic embolism from a clot in the systemic vein. Loss of clotting factors from the gut secondary to protein-losing enteropathy also puts these children at risk of thromboembolic complications. When evaluating for intravascular thrombosis, first-pass imaging with CT or MR angiography is the ideal method to evaluate for intravascular filling defects. However, a high prevalence of variant vascular anatomy in this patient population, such as bilateral superior vena cava or interrupted inferior vena cava with polysplenia, can result in mixing of contrast and unopacified blood resulting in pseudo-filling defects that can limit evaluation for intravascular thrombus. To keep mixing artifacts at a minimum, the site of intravenous contrast injection should be carefully selected based on a child's known vascular anatomy. Simultaneous injection of contrast agent from more than one site, e.g., simultaneous injection of contrast agent from both an upper and a lower extremity vein to adequately opacify the Glenn and Fontan

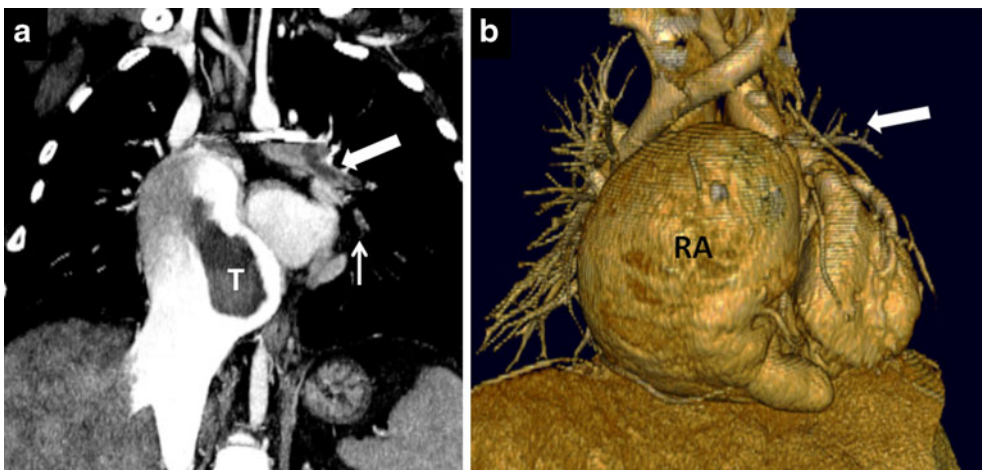

Fig. 12 Pulmonary embolism after Fontan conduit in a 25-year-old woman with history of Fontan procedure with increasing shortness of breath and cough. **a** Coronal reconstructed image from CT angiography demonstrates a large thrombus (*T*) within the dilated right atrium, as well as the left pulmonary artery (*thick arrow*) and peripheral thrombosis (*thin arrow*). **b** 3-D volume-rendered anterior projection from the CT angiogram demonstrates massive dilatation of the right atrium (*RA*) due to stasis (which led to the thrombosis), and poor visualization of the left pulmonary parenchyma vasculature due to the left pulmonary artery thrombus (*arrow*)

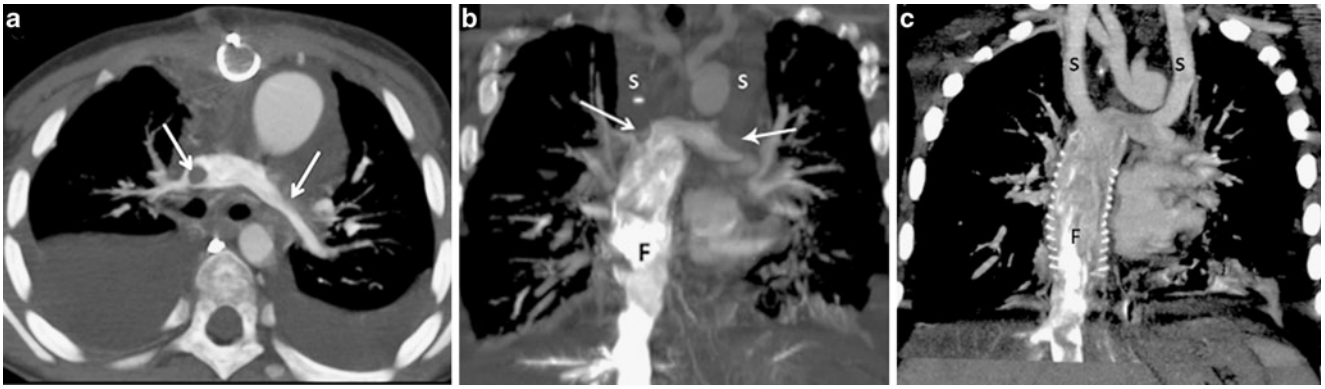

Fig. 13 Pseudo-filling defect due to mixture of contrast agent and unopacified blood. Axial (**a**) and coronal (**b**) images obtained after contrast injection in the lower extremity show a well-opacified Fontan conduit (*F*). Filling defects (*arrows*) are seen near the anastomosis of the bilateral superior vena cava (*S*) with pulmonary arteries that could mimic pulmonary embolism. **c** Delayed-phase image in the coronal plane shows resolution of the filling defect. The delayed phase also shows bilateral superior cavopulmonary anastomoses and a Fontan conduit (*F*)

circuits, can help avoid pitfalls due to mixing artifacts when performing first-pass imaging [26]. Dual-phase imaging, with a second set of images in the equilibrium phase, can also help circumvent this problem (Fig. 13). While this is done routinely with MR angiography, a case-by-case decision has to be made with CT angiography to keep the radiation exposure as low as reasonably possible.

Summary

With improvement in long-term survival, extracardiac complications are being increasingly encountered in children and young adults who have undergone Fontan surgery for congenital heart disease in infancy. Commonly encountered extracardiac complications include passive hepatic congestion, regenerative nodules, cardiac cirrhosis, protein-losing enteropathy, plastic bronchitis, extracardiac right-to-left shunts and thromboembolic complications. Often times, these complications result in the child presenting outside of a congenital heart disease clinic, requiring the general radiologist to be familiar with them. The imaging appearance of these extracardiac complications has been reviewed in this pictorial essay.

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