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Imaging in the Adult Patient with Fontan Circulation

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Short title: Imaging in the Adult Fontan Patient

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Clinical vignette

A 32-year old woman was seen for acute chest pain radiating to the neck and arms. The paramedical team witnessed her cardiac arrest on arrival and successfully defibrillated ventricular fibrillation by a single shock. After a stable transfer to the local hospital she had a second witnessed arrest necessitating further defibrillation. History revealed good functional capacity and no other cardiac symptoms. Examination showed clear lungs, normal heart sounds, a normal abdomen, a blood pressure of 110/79 mmHg and an oxygen saturation of 100%. The electrocardiogram showed signs of inferolateral infarction. Cardiac troponin I was 1.06 ng/mL. Telemetry confirmed intermittent non-sustained ventricular tachycardia and sinus bradycardia. Her medical therapy included bisoprolol 2.5 mg and warfarin (INR: 1.7). Due to tricuspid atresia, transposition of the great arteries, pulmonary stenosis and ventricular septum defect (VSD) she had undergone superior cavo-pulmonary shunt (Glenn) aged 4, and atrio-pulmonary (AP; Fontan) connection aged 7 years. Three years before, recurrent supraventricular tachycardia necessitated multiple electrophysiological studies with ablation of focal right atrial (RA) tachycardias and cavo-tricuspid isthmus-dependent flutter.

Echocardiography showed preserved ventricular function, mild flow acceleration across the sub-aortic VSD, mild mitral regurgitation as well as dilated systemic and hepatic veins. Computed tomography (CT) of the pulmonary arteries (PA; CTPA) was subsequently performed, reporting a large opacification defect in the proximal left pulmonary artery (LPA), suggestive of pulmonary embolism (Fig. 1). Cardiac magnetic resonance imaging (CMR) thereafter showed a patent Fontan pathway, no thrombi and normal pulmonary arborization. The coronary sinus, the RA, the cardiac,

hepatic and the systemic veins were severely distended (IVC; Fig. 2 and 3) with diastolic flow reversal in the inferior vena cava. No systemic-to-pulmonary collaterals (SPC) were identified. Pulmonary venous return was normal. Left ventricular (LV) ejection fraction (EF) was 50%. Focal hypokinesia of the lateral and inferolateral segments with late sub-endocardial Gadolinium enhancement were present (Fig. 4 and 5). A large circumflex artery was seen, arising from the non-coronary sinus, whereas the right coronary artery was reported as non-developed. A catheter study subsequently showed a pressure of 10 mmHg in the Fontan circuit, confirmed this coronary anatomy and ruled out any obstructions (Fig. 6 and 7). Serial electrocardiograms showed the typical progression of acute myocardial infarction.

The Fontan circulation: Principle and pitfalls

The Fontan procedure is a well-established surgical approach to improve survival in univentricular heart disease, re-routing systemic venous blood flow directly to the lungs in a multi-staged approach. Passive pulmonary blood flow is driven by a trans-pulmonary pressure gradient, thus bypassing the heart in order to offload the ventricle. The original technique proposed anastomosing the RA appendage to the PA. Several modifications have led to the contemporary total cavo-pulmonary connection (TCPC) since.[1] Long-term complications are frequent (Table 1). While some represent potentially treatable complications, a significant proportion of patients undergo hemodynamic deterioration even in their absence. This may result in ‘Fontan Failure’, a state characterized by multi-organ dysfunction, peripheral edema, ascites and abnormal protein turnover. Its pathogenesis is still incompletely understood. Once established, the outcome is poor and heart transplant is the only recognized cure.

Early diagnosis and transplant listing are crucial to survival. Routine imaging is, therefore, paramount in the follow-up of Fontan patients.

How to use imaging in the pathological Fontan circulation

A full echocardiographic study should be part of every outpatient visit in intervals of no more than 12 months (Table 2). Functional assessment remains challenging, especially in the systemic RV, but serial imaging can reveal deterioration over time using M-mode, tissue Doppler or fractional area change. Mitral inflow velocities can reveal abnormal diastolic function (Fig. 8). Other techniques for functional assessment have been described.[2, 3]

The advantages of CMR are widely recognized (Table 3).[4] Vascular anatomy can be imaged with high spatial detail, even in the presence metal, poor breath-holding or slow blood flow.[4-7] Gadolinium-contrast application is recommended for increased tissue contrast, better delineation of vascular anatomy and shunts as well as tissue viability assessment. We have recently demonstrated the usefulness of time-resolved contrast enhanced imaging in children and adults with congenital heart disease (CHD), where contrast passage can be visualized throughout the entire cardiac cycle, even in free breathing.[8] This offers additional diagnostic information that could point towards collateral flow or flow obstacles and obviates the need for timing in the acquisition of data. Volumetric and functional analysis of the ventricle is typically achieved by stacked cine imaging in the short axis (e.g. 2D b-SSFP).[5, 9] Different techniques of image planning and post-processing have been described and should be consistent within an institution in order to keep inter-observer variability low and thus accurately detect changes in volumes and function over time.[5] This is crucial as end-

diastolic volume is a strong predictor of death and transplant-free survival.[10] For flow measurement, real-time phase-encoding velocity mapping sequences are more accurate than breath-held phase-contrast imaging techniques due to the effects of breath holding on passive pulmonary filling.[11] However, due to the limited availability of these sequences, retrospectively gated phase-contrast CMR in free breathing is commonly regarded standard by most centers. Calculating the difference between pulmonary venous return and PA flow is the most accurate way to determine SPC flow.[12] Phase-contrast imaging of the systemic outlet also allows for quantification of semilunar valve regurgitation and can be useful for the quality control of ventricular measurements by comparing stroke volumes from both methods. Total venous flow return is a known CMR biomarker of Fontan decompensation. However, its role in the early diagnosis of hemodynamic failure has yet to be evaluated.[13]

CT is an excellent alternative to CMR as it allows for a comprehensive assessment of anatomy at sub-millimeter isotropic resolution, cardiac and valvar function as well as of stent patency (Table 4).[14] Short scanning times make this imaging modality ideal for acute situations where information needs to be acquired quickly with minimal preparatory effort and little patient cooperativeness. Cardiac CT is also the best available technique for imaging metallic intravascular stents and devices within the Fontan circulation. However, temporal resolution is poor compared to that of CMR and echocardiography and its association with ionizing radiation, albeit low on modern systems, limit its routine use. Therefore, CT should be restricted to cases where CMR is either unavailable or unsafe to perform and where additional information such as stent or shunt patency must be obtained.

The importance of invasive imaging by cardiac catheterization has declined substantially with the increased availability of CMR and CT. Due to its association with ionizing radiation and procedural risk the indication for invasive, catheter-based imaging is of limited use for routine imaging (Table 5). Moreover, in patients with previous cardiac surgery, vascular access can be difficult to obtain. Therefore, invasive catheter studies should be reserved to cases where non-invasive options have been exhausted. 'Hybrid' CMR-augmented cardiac catheterization can assess pulmonary vascular resistance prior to cardiac transplantation.[15]

Challenges and advances in Fontan imaging

Diastolic dysfunction is a key issue in univentricular CHD and non-invasive diagnosis remains a challenge (Table 1). Echocardiographic tissue deformation indices do not typically comply with normal values from biventricular hearts but may reveal progressive dysfunction over time. Recently, hemodynamic stress protocols using dobutamine or fluid boluses have been demonstrated to detect latent diastolic dysfunction by CMR and/or cardiac catheter.[16, 17]

The dual cavo-pulmonary blood supply often results in preferential streaming from the superior vena cava (SVC) to the right PA and from the IVC to the LPA. This may lead to several problems. Incomplete opacification of one branch PA after contrast application can mimic pulmonary embolism – as was the case in this patient (Fig. 2). This can be overcome by injecting contrast into the right arm, the lower extremity or both, depending on the clinical question. Moreover, preferential blood supply from the lower body to one lung is known to promote arteriovenous malformations in the

contralateral lung due to the lack of hepatic venous return. Phase-velocity CMR in a perpendicular plane relative to the branch pulmonary arteries has been shown to be more accurate than perfusion scintigraphy for the measurement of differential lung perfusion.[18] More recently, time-resolved '4D-flow' CMR-sequences have emerged as a novel approach for surgical planning and risk stratification.[19] While typically longer to acquire than conventional '2D' flow-mapping sequences, their advantage lies in the possibility to obtain all flow-encoded information within a defined volume along all spatial directions as a single dataset and to arbitrarily define the plane of flow measurement in retrospect. Flow patterns can thus be visualized multi-dimensionally and interpreted in conjunction with anatomy.[20] Similarly, though technically different, computational fluid dynamics are increasingly used to assess the interactions between blood flow and anatomical structures.[21]

Finally, liver cirrhosis and hepatic neoplasia are increasingly recognized sequelae of the Fontan circulation and can worsen prognosis.[22] Screening for cirrhosis by MR-elastography offers several advantages over conventional, ultrasound-based techniques.[23] Regular screening for structural abnormalities of the liver using ultrasound, MR or CT is considered standard.[22]

Approach to the presented patient

Our patient presented with life threatening arrhythmia and therefore, a number of potentially deleterious and reversible differential diagnoses had to be rapidly eliminated. Pulmonary thromboembolism was initially suspected due to sub-therapeutic anticoagulation and poor blood flow in the Fontan pathway, supported by an opacification defect in the LPA. However, this was subsequently demonstrated to

be caused by preferential pulmonary blood streaming and CMR revealed evidence of poor function of the Fontan circuit and myocardial infarction. No coronary artery anomalies were detected which raises the possibility of spasm or thromboembolism. Abnormal hemodynamics, including chronically reduced coronary perfusion pressures with possible atrial arrhythmia, may have contributed to the infarction. Moreover, it is conceivable that sluggish drainage from the cardiac veins may have precipitated thrombus formation and caused venous myocardial infarction. Such a thrombus may have spontaneously migrated into the pulmonary vascular bed and dissolved under anticoagulation therapy. An ICD was implanted to prevent sudden death as per current guidelines.[24] Transvenous leads are difficult to position in the Fontan circuit and therefore, a subcutaneous system was used.

Early recognition of the Failing Fontan physiology remains challenging. In the presented case, no lesion amenable to treatment was found to potentially improve hemodynamics. Though Fontan pressure was acceptable, this finding must be interpreted in the context of procedural anesthesia. The presence of AP-type Fontan is a known risk-factor for deteriorating hemodynamics as it causes energy loss, dilatation and, thus, poor flow in the circuit.[25] This, in combination with her history of intractable arrhythmia and myocardial infarction, puts the patient at high risk of death, irrespective of her preserved EF and good functional status.[25-27] Because timely listing is crucial, our patient was, therefore, subsequently referred for heart transplant evaluation.

In summary, complex and univentricular CHD is a growing disease entity in the adult population and necessitates rigorous longitudinal monitoring to identify patients at

risk. Appropriate imaging protocols for the chronic and acute management of this patient group are essential and different modalities should be used in a complementary fashion to assess hemodynamics, anatomy and prognosis. Early recognition of failing hemodynamics in Fontan patients is essential to secure long-term survival.

Conflict of Interest Disclosures: none.

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Legends

Figure 1. Computer tomography pulmonary angiography showing an opacification defects in the left pulmonary artery (arrow).

Figure 2. 3D-reconstruction from MR-angiography showing severe dilatation of the cardiac veins and coronary sinus (asterisks).

Figure 3. Sagittal image from b-SSFP cine (CMR) showing a grossly dilated RA and sluggish flow in the atrio-pulmonary segment (asterisks).

Figure 4. Short axis image (CMR) showing late Gadolinium enhancement in the LV inferolateral wall (arrow).

Figure 5. Long axis image (CMR) showing late Gadolinium enhancement of the LV inferolateral wall (arrows).

Figure 6: Hemodynamic measures of the presented patient.

Figure 7. Right anterior oblique projection from coronary angiography showing a large circumflex artery (arrow) with obtuse marginal branches, arising from the non-coronary sinus.

Figure 8. Pulsed wave Doppler signal across the mitral valve, indicating abnormal diastolic function.

Tables

Table 1. Complications of the Fontan circulation and proposed imaging approach.

Complication	Possible causes	Possible imaging modalities
Obstruction of the venous-to-pulmonary pathway	Thromboembolism, external compression (e.g. dilated atria), traction/torsion (e.g. after shunt)	Echocardiography, CMR, CT, catheter study: direct visualization of obstruction or signs of upstream dilatation; pressure gradient (catheter)
AV-valve regurgitation	Ventricular dilatation, congenital dysplasia	Echocardiography, CMR, catheter, (CT): direct visualization of regurgitant jet; estimation of regurgitant fraction (CMR)
Outlet obstruction	(Sub-) Valvar obstruction, VSD restriction, (neo-) aortic obstruction	Echocardiography, CMR, CT, catheter study: direct visualization; estimation of pressure gradient (catheter and echocardiography)
Systolic dysfunction	Systemic RV, decreased preload, increased afterload, poor coronary perfusion pressure	CMR, echocardiography (CT, catheter): volume measurement and calculation of ejection fraction

Diastolic dysfunction	Volume overload (e.g. shunt in early stages; high collateral flow), ventricular hypertrophy	Catheter study: direct pressure measurement (consider fluid challenge) Echocardiography: tissue deformation indices (longitudinal follow-up) 'Hybrid' CMR under dobutamine stress
Increased pulmonary vascular resistance	Chronic high Q_P in early stages; disproportionate growth of pulmonary vasculature	Catheter study and 'hybrid'-CMR: direct pressure measurement and calculation of pulmonary vascular resistance
Collateral flow	Venous decompression, lack of hepatic venous flow to lungs, chronic cyanosis	CMR, catheter study, (CT): direct visualization of collateral vessels and collateral flow estimation Echocardiography: indirect visualization of veno-venous collaterals by contrast injection
Liver disease (cirrhosis, neoplasia)	Chronic hepatic venous congestion	Transient/MR-Elastography Liver ultrasound, CT, MR

Table 2. Echocardiography in Fontan imaging.
Strengths
• Availability, low cost, relative ease of use, bedside compatibility • Lack of invasiveness and ionizing radiation, no contraindications • High temporal and spatial resolution • Relatively robust to arrhythmia • Good characterization of valve function
Weaknesses
• Inter- and intra-observer variability • Oftentimes poor acoustic window secondary to surgery • No myocardial tissue characterization • Limited use for assessment of ventricular volume and function, especially in the systemic RV • Unsuitable for shunt estimation and visualization of collaterals • Certain structures may be difficult to visualize (pulmonary veins, conduit)

Table 3. Magnetic resonance in Fontan imaging.
Strengths
• Comprehensive imaging of virtually any structure, independently of user and anatomical problems • 3-D imaging • Lack of invasiveness and ionizing radiation • High spatial resolution • Tissue characterization • Gold standard for volumetry and functional assessment • Shunt estimation
Weaknesses
• Expensive, lengthy examination technique, requires patient cooperation; unsuitable for acute setting • Limited availability, reserved to expert centers • Post-processing • Limited temporal resolution • Some sequences vulnerable to arrhythmia • No pressure measurement • Contraindications apply (metal, pregnancy, claustrophobia)
Pitfalls
• Upper limb contrast injection can mimic pulmonary embolus • Sluggish, swirling blood flow may cause image artifacts mimicking thrombus

Table 4. Computed tomography in Fontan imaging.
Strengths
• Good availability, short scan duration, suitable for acute work-up • Comprehensive, user-independent imaging of virtually any structure • 3-D imaging • Non-invasive • Gold-standard for stent assessment • Very high spatial resolution • Good coronary imaging • Suitable for volumetry and functional assessment
Weaknesses
• Exposition to ionizing radiation (albeit low on modern systems) • Poor tissue contrast • Low temporal resolution • Limited hemodynamic data • Limited use in valve assessment
Pitfalls
• Upper limb contrast injection can mimic pulmonary embolus

Table 5. Cardiac catheter studies in Fontan imaging.
Strengths
• Possibility to intervene • Very high spatial and temporal resolution • Suitable for stent imaging • Pressure and flow measurement • 3-D reconstruction on modern systems (albeit limited) • Good coronary imaging
Weaknesses
• Exposition to ionizing radiation • Invasive • May require anesthesia or sedation • Contrast-associated nephrotoxicity • Vascular access may be difficult after repeat cardiac surgery