Review

The emerging role of cardiovascular MRI for suspected cardioembolic stroke

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Introduction

Stroke is a leading cause of morbidity and long-term disability worldwide and is often the result of embolic material from the heart or proximal aorta. These are referred to as cardioembolic sources of stroke. The investigation of patients with suspected cardioembolic stroke has traditionally been the mainstay of echocardiography. Cardiac magnetic resonance imaging (MRI) is a powerful imaging technique that has rapidly evolved over the last decade and is playing an ever increasing role in clinical cardiovascular imaging. This review of the literature aims to furnish the reader with an understanding of the role of cardiac MRI across the spectrum of causes of cardioembolic sources of stroke by providing the reader with an overview of the indications, technical considerations, a proposed imaging algorithm, and capabilities of this technology with selected illustrated examples of disease entities.

Stroke is a leading cause of morbidity and long-term disability worldwide and is often the result of embolic material from the heart or proximal aorta. These are referred to as cardioembolic sources of stroke. The investigation of patients with suspected cardioembolic stroke has traditionally been the mainstay of echocardiography. Cardiac magnetic resonance imaging (MRI) is a powerful imaging technique that has rapidly evolved over the last decade and is playing an ever increasing role in clinical cardiovascular imaging. This review of the literature aims to furnish the reader with an understanding of the role of cardiac MRI across the spectrum of causes of cardioembolic sources of stroke by providing the reader with an overview of the indications, technical considerations, a proposed imaging algorithm, and capabilities of this technology with selected illustrated examples of disease entities.

The aetiology of stroke and CESS has been subdivided. Adams et al.4 developed a classification system based on the Trial of Org 10172 in Acute Stroke Treatment (TOAST). Within this CESS were divided into high- and medium-risk categories based on the evidence available at the time. Subsequent papers on CESS have further categorized these into an anatomical framework and changed the division to high and medium/uncertain risk5 (Table 1 classifies the causes described in this review and is adapted from the classification by Doufekias et al.5).

Pathophysiology of cardioembolic stroke

The brain receives 15% of the cardiac output and is sensitive to ischaemia. Hence material dislodged proximal to the cervicocephalic arteries has a tendency to travel into the brain where it may occlude cerebral arteries leading to infarction.5 Recent work by Harloff et al.6 has highlighted that atherothromboembolism may also originate in the
Table 1
Potential causes of cardioembolic stroke.

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<th>High risk</th>
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<td>Left atrial thrombus</td>
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<td>Left ventricular thrombus</td>
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<td>• Hypokinetic left ventricular segment</td>
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<td>Patent foramen ovale</td>
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<td>Bioprosthetic cardiac valve</td>
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<td>Aortic dissection</td>
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Non-invasive imaging techniques

At present the first-line investigation for cardiac sources of atherothromboembolism is transthoracic echocardiography (TTE), which is deemed sufficient if it is positive. Transoesophageal echocardiography (TOE) for indications such as left atrial thrombus, aortic atheroma, patent foramen ovale (PFO), and valvular strands allows for better evaluation as well as improved detection of valvular vegetations.3 The major limiting factor for echocardiography is its somewhat restricted field of view and heavy reliance on a skilled operator. Both TTE and TOE cannot completely interrogate the entire thoracic aorta and have limited sensitivity for thrombus due to its highly variable echogenicity.

Recently there has been some interest in using electrocardiogram (ECG)-gated multidetector computed tomography (MDCT) to study the left heart and great vessels in suspected CESS.9 MDCT is potentially an attractive technique for this application given its extremely fast examination time combined with high spatial resolution (0.4–0.6 mm). Currently the main drawback is its relative lack of inherent soft-tissue contrast, which rather limits its assessment of the myocardium and identification of small thrombi. Other disadvantages are high radiation burden (when a retrospective protocol is used as is required to study valve function) and exposure to potentially nephrotoxic iodinated contrast agents.

Advances in and the increasing availability of magnetic resonance imaging (MRI) have led to its increasing use in the diagnosis of stroke and imaging of the carotid arteries. Routine cardiovascular MRI (CMR) imaging in the context of stroke does not currently form part of consensus guidelines but there is an increasing body of literature to support its role, as an adjunct to echocardiography, in selected cases. CMR may be of value in patients unwilling or unable to undergo TOE or in specific cases where the echocardiographic findings have been inconclusive. Current indications for CMR in stroke are patients with a TTE study that is indeterminate for the presence of LV thrombus, and where the potential for a false negative TOE result exists, e.g., LV or left atrial appendage (LAA) thrombus or suspected cardiac mass.2 In addition, CMR can provide assessment of the great vessels as well as detection of small right-to-left shunts that may not be reliably evaluated with competing methods. This review aims to furnish the reader with an understanding of the indications, protocols, and key CMR features in the setting of CESS.

Cardiovascular MRI technique

Unlike MRI examinations in many other body parts, cardiac MRI (CMR) frequently requires hands-on physician input at the time of scan acquisition to assess and modify the study depending upon preliminary findings and patient anatomy.

We suggest using a core structural assessment protocol to assess anatomy, flow, valves, and biventricular systolic function, combined with a late-phase post-gadolinium sequence to look for intracardiac thrombus (Table 2). Such a protocol can be performed in around 45 min, but requires a co-operative patient who is compliant with breath-hold instructions. Additional sequences such as those to look specifically for a PFO can be added at the discretion of the supervising physician but have not yet been extensively validated in routine clinical practice.

Static “black blood” prepared double-inversion recovery (DIR) fast spin-echo sequences provide assessment of great vessel connections including pulmonary venous anatomy. They also enable visualization of vessel wall thickening as may be present with large vessel vasculitis. Dynamic “bright blood” prepared cine sequences are acquired using a steady-state free-precession (SSFP) technique, which produces intrinsically high contrast between the blood pool and adjacent valves and myocardium without the need for exogenous contrast material. SSFP sequences are acquired in standard cardiac imaging planes and enable detailed assessment of myocardial contractility and valve function. SSFP sequences are conventionally acquired with an 8–10 s...
breath-hold (depending on heart rate) but acceleration techniques, such as parallel imaging, may be helpful to reduce examination times in this cohort of patients who may struggle with a prolonged time in the scanner. For those unable to breath-hold, “real-time” cine SSFP can be used. It should be noted that sequence acceleration often comes at the expense of reduced spatial resolution and an acceptable trade-off must be sought.

For assessment of a possible right-to-left shunt [whereby blood is shunted from the right side of the heart to the left side of the heart, e.g., atrial septal defect (ASD)] the ratio of pulmonary blood flow (Qp) to systemic blood flow (Qs), which is normally approximately equal to 1.0, is measured using velocity-encoded phase-contrast sequences acquired perpendicular to the proximal ascending aorta (Qs) and main pulmonary artery (Qp). This technique permits detection of even small shunt fractions, but it should be noted that it will not be reliable in the setting of significant valvular regurgitations.¹⁰

Gadolinium-enhanced angiography of the thoracic aorta and great vessels provides depiction of patho-anatomical alterations, including protruding arch atheroma and carotid dissection. Delayed gadolinium enhancement imaging using an inversion recovery sequence to null signal from normal myocardium facilitates detection of intracardiac thrombus in the left atrium or left ventricle. This is performed at the end of the examination, approximately 10–15 min after gadolinium administration and is based on the principle that gadolinium accumulates within an expanded interstitial space, such as myocardial fibrosis from prior infarction as well as some benign and malignant cardiac tumours. Typically thrombus appears as a low signal-intensity mass surrounded by high signal-intensity cavity blood and/or hyperenhanced myocardial scar.¹¹ Selecting a long inversion time (600 ms) has been shown to further improve the sensitivity of this technique for thrombus detection by making it appear homogeneously black and even more conspicuous from adjacent structures.¹²

**CMR specific imaging features**

In this section the individual causes of CESS are discussed and the relative value of CMR reviewed.

**Left heart thrombus**

**Left atrial thrombus (high risk)**

Atrial fibrillation (AF) causes uncoordinated atrial activation with decreased flow velocities and the potential for spontaneous thrombus formation, which most often occurs within the left atrial appendage. The incidence of
stroke in patients with AF is around 5% per year which is seven times that in people with sinus rhythm. TOE is the reference standard for detection of atrial thrombi but is very reliant on operator experience, especially within the atrial appendage because of its complex shape. CMR can be used as an alternative to TOE to evaluate the left atrium and left atrial appendage for thrombus in patients where TOE examination is incomplete or inconclusive, but there is currently a paucity of published research as to its accuracy in this setting. Resting perfusion and late gadolinium enhancement are the most accurate means of assessment as thrombus usually stands out on these sequences due to clear delineation from the surrounding blood pool (Fig 1).

**LV thrombus (high risk)**

LV thrombus may result from a number of causes that are commonly related to myocardial infarction (MI). Those that have been correlated with stroke are recent myocardial infarction (<4 weeks; high risk); akinetic LV segment (high risk); myocardial infarction (>4 weeks, <6 months; medium risk); and hypokinetic LV segment (medium risk).

With acute MI the absolute risk of stroke is 2% in the first 30 days, which is thought to be due to multiple factors including dysrhythmias, hypotension, coronary and carotid plaque inflammation, and impaired LV systolic function. Most mural thrombi occur within the first 2 weeks of MI and usually in the context of a large infarct causing reduced LV function or apical akinesia. The risk of embolism is at its highest when the thrombus is mobile or protrudes into the ventricle. Most thrombi are thought to slowly resolve over several months following an MI and the risk of embolism is believed to be low after the first 3 months. CMR is a well validated and highly accurate technique for detection of LV thrombus using delayed gadolinium enhancement sequences (Fig 2).

**Dilated cardiomyopathy (high risk)**

Dilated cardiomyopathy (DCM) carries a high risk of LV thrombus formation, which occurs secondary to reduced flow velocities, abnormal endocardial surfaces, and a hypercoagulable state. Indeed the prevalence of LV thrombus in patients with DCM is around 40%. Stroke risk is positively correlated with worsening systolic function with the incidence of thromboembolism ranging from 1–3.5% per year. LV thrombi in DCM can be challenging to detect.

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**Figure 1** Left atrial thrombus in a 58-year-old woman. (a) Axial SSFP image showing two well-circumscribed, low-signal lesions attached to the left atrial wall by a short stalk (arrows). Also note the presence of a left pleural effusion (asterisk). The differential diagnosis was between left atrial myxomas and thrombus. (b) Axial image from dynamic gadolinium rest perfusion study showing complete absence of first-pass enhancement in keeping with poorly vascularized lesions (arrows). (c) Two-chamber, T1-weighted, delayed-phase image obtained 10 min following gadolinium administration showing complete absence of late enhancement within the most superior lesion (arrows). (d) Sagittal, T1-weighted, delayed-phase image showing complete absence of enhancement, which is typical for thrombus. Findings are suggestive of thrombus, which was confirmed on follow-up imaging as the lesions completely resolved after a period of anticoagulation (not shown).
with TTE/TOE as they are not always located within the most dyskinetic segments and it can be difficult to fully interrogate a very dilated ventricle if acoustic windows are limited. CMR is the technique of choice to investigate the cause of DCM as myocardial tissue characterization using late gadolinium enhancement, may identify signs of prior infarction (subendocardial or transmural gadolinium enhancement within recognized coronary perfusion territories). An idiopathic aetiology of DCM is suggested by an absence of enhancement or thin line of enhancement within the interventricular septum.

Any associated LV thrombus is usually readily identified.

**Arrhythmias related to left heart thrombus (high risk)**

Atrial dysrhythmias are not considered in this review as CMR does not form part of the routine diagnostic work-up; however, it is a very useful means of detecting associated left atrial thrombus. Cardiogenic causes of stroke pertaining to dysrhythmias are AF (other than lone AF; high risk); sick sinus syndrome (high risk); atrial flutter (medium risk); and lone AF (medium risk).

**Sources of paradoxical embolism**

**PFO and atrial septal aneurysm (medium/unclear risk)**

PFO is a flap-like interatrial communication at the fossa ovalis, which permits intermittent bidirectional blood flow between the atria and the possibility for “right circulation” thrombus to bypass the filter-like action of the lungs and pass directly into the left heart/systemic circulation. Transient right-to-left shunting is thought to be produced by intermittent elevation of right atrial pressure, such as with inspiration, coughing, straining, and Valsalva manoeuver. In adults its prevalence ranges from 25–34%. Estimates suggest that PFOs have low pathogenicity\(^1\) but in young patients (<56 years) with an unexplained stroke, PFO is the commonest cardiac finding and several studies have documented a positive association.\(^5\) An atrial septal aneurysm (ASA) describes excessive septal bowing into the atria and is strongly associated with a PFO; it has a prevalence of 1% and is found more commonly in patients with ischaemic stroke than in the general population (7.9 versus 2.2%).\(^5,19\)

TOE/TTE are the reference standard techniques for diagnosing and assessing a PFO including its degree of patency and the presence of an ASA.\(^5\) ASA appears as septal bowing >10 mm into either atria and is usually easily seen with CMR (Fig 3). The presence of a flap or channel-like appearance of the interatrial septum is suggestive of a PFO; however, turbulence from shunting on SSFP sequences is rarely seen if atrial pressures are equal during image acquisition. In addition the relatively low spatial resolution of CMR (1–2 mm) often limits reliable assessment.\(^20,21\) First-pass gadolinium-enhanced perfusion CMR performed during Valsalva maneuver can diagnose a PFO if there is contrast flow from the right to left atrium; however, this technique detects only 63% of shunts identified on TTE.\(^22\) Recently a first-pass perfusion CMR method with real-time “mapping” of enhancement curves in the left and right atria has been described for PFO assessment. In a small pilot study of 15 patients with a known PFO, investigators used analysis of enhancement curves to successfully diagnose a PFO in all patients and reliably distinguish it from...
other causes of right-to-left shunting, such as pulmonary arteriovenous malformations. This technique is rather time intensive and is yet to be validated in routine clinical practice.

**ASDs (medium/unclear risk)**

ASD is the commonest form of congenital heart disease presenting in adulthood and clinical manifestations include stroke and pulmonary hypertension. There are four ASD subtypes reflecting defects in different portions of the interatrial septum: ostium secundum (70%), ostium primum (20%), sinus venosus (10%), and coronary sinus (<1%). TTE is generally an accurate means of diagnosing most secundum, primum, and coronary sinus defects but can identify only 12% of sinus venosus ASD (SVASD). SVASD reflects a defect in the wall that normally separates the superior vena cava (SVC) or inferior vena cava (IVC) from the left atrium and is located outside the fossa ovalis. There is a high association (85%) of partial anomalous drainage of the right superior pulmonary vein into the SVC. The multiplanar capabilities of CMR make it well suited for identification of SVASD and any anomalous veins (Fig 4).

**Left-sided cardiac tumours (high risk)**

Primary cardiac tumours are rare with a lifetime incidence of 0.001%; approximately 75% are benign with most of these being due to left atrial myxoma or valvular fibroelastoma. A left heart tumour carries a significant risk of systemic embolization and stroke, either from detachment of tumour fragments or accumulated thrombus. Indeed around 30% of myxomas present clinically with neurological symptoms. Owing to its ability to provide detailed tissue characterization, CMR is the reference standard technique for the assessment of a suspected cardiac mass with a high accuracy for distinguishing tumour from thrombus. Myxoma typically appears as a mobile polypoid mass attached to the interatrial septum by a short pedicle; although broad-based sessile lesions are also recognized. Myxomas usually have a villous surface texture and display patchy late gadolinium enhancement due to varying components of myxoid, fibrous, haemorrhagic, and ossified material (Fig 5).

Fibroelastoma is a small benign tumour composed of collagen and elastic tissue, which can arise from any endocardial surface. They are most often located on the atrial side of the mitral valve and aortic side of the aortic valve. Most remain asymptomatic, but peritumoural thrombosis can lead to stroke and surgical resection is usually advocated for left-sided lesions. Fibroelastoma usually appears as a well-circumscribed, mobile nodule on cine SSFP with perilesional turbulence. Uniform late gadolinium enhancement of these lesions has been reported and is presumably a reflection of their fibroelastic tissue composition (Fig 6).

**Left-sided native valvular disease**

TTE is the first-line imaging method for assessment of aortic and mitral valve disease with CMR reserved for those with equivocal findings or unable to undergo TOE. CMR has inferior spatial resolution to TTE/TOE (2 versus 1 mm/0.5 mm), which precludes detailed study of leaflet thickness but can still provide a reliable assessment of valve motion and function. A variety of CMR techniques can be used to quantify valvular stenosis and regurgitation, including planimetry of orifice area and phase-contrast transvalvular flow analysis.

**Mitrinal stenosis with AF (high risk)**

Patients with mitral stenosis and AF are at greater risk of stroke compared with those with mitral stenosis alone. The precise reason that mitral stenosis increases the risk of stroke is unclear but postulated mechanisms are embolization of small amounts of calcification, the thickened valve

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**Figure 4** Sinus venous ASD and partial anomalous pulmonary venous drainage in a 40-year-old woman with pulmonary hypertension. (a) Axial SSFP image through the superior portion of the interatrial septum showing a large communication between the atria (arrowhead) in keeping with a superior sinus venosus type ASD. (b) Axial SSFP image [3 cm cranial to (a)] showing anomalous drainage of the right superior pulmonary vein into the superior vena cava (arrow), which is a common association.
leaflets, and subvalvular apparatus acting as a nidus for thrombus formation and atrial dilatation leading to AF with appendage thrombosis.28

The most common cause of mitral stenosis is rheumatic heart disease, which manifests as progressive leaflet thickening and commissural fusion. The normal mitral valve orifice measures 4–6 cm² and a narrowing of less than 2 cm² is considered clinically significant.29 Retraction and restriction of the subvalvular apparatus may also be apparent and contributes to the classical “hockey-stick deformity” appearance on two and four-chamber SSFP sequences7 (Fig 7). Less common causes of mitral stenosis include connective tissue diseases and pulmonary carcinoid tumours, which cause leaflet thickening and fixation.7

Mitral valve prolapse (medium/uncertain risk)

The association between mitral valve prolapse (MVP) and cardioembolic stroke has not been firmly established with a number of conflicting studies in the literature.30 The exact mechanism whereby MVP may cause stroke is uncertain but is presumed due to micro-thrombus formation secondary to altered flow dynamics.

MVP is defined as systolic bowing of a mitral leaflet by more than 2 mm beyond the annular plane into the left atrium. It may be caused by rupture or elongation of the chordae tendinae (sub-valvular apparatus).31 As with mitral

Figure 5 Myxoma in a 52-year-old woman with cardioembolic events. (a) Axial, T1-weighted, black-blood image showing a well-defined mass in the body of the left atrium (arrows). (b) Axial, T1-weighted, delayed-phase image obtained 15 min following administration of gadolinium showing subtle patchy areas of enhancement (arrows) (with permission from Hoey ETD et al. MRI and CT appearances of cardiac tumours in adults. Clin Radiol 2009;64:1214–1230). LA, left atrium, LV, left ventricle.

Figure 6 Mitral valve papillary fibroelastoma in a 60-year-old man with recurrent transient ischaemic attacks (TIAs). Two-chamber SSFP image showing a well-demarcated, low-signal, 1.5 cm lesion attached to the posterior leaflet of the mitral valve (arrows).

Figure 7 Rheumatic mitral stenosis in a 70-year-old man with rheumatic mitral stenosis. Four-chamber SSFP image showing thickening and restricted opening of the mitral valve (arrows). The left atrium is dilated and there are signs of secondary pulmonary hypertension with enlargement of the right heart chambers. LA, left atrium; RV, right ventricle; RA, right atrium.
stenosis CMR does not play a primary role in the assessment of MVP, but it may be an important finding in a CESS study. MVP is best evaluated on the two-chamber SSFP images by taking a reference line along the annulus plane from which to measure any central leaflet bowing. There will usually be an accompanying low-signal, regurgitated jet back from the valve into the left atrium caused by spin dephasing. CMR can be used to quantify the regurgitated fraction if required by dividing forward flow volume in the proximal ascending aorta (derived by proximal aortic phase-contrast flow study) by LV stroke volume (from volume analysis of the SA SSFP images).

### Aortic valve stenosis and calcification (medium/uncertain risk)

Degenerative aortic stenosis (AS) affects approximately 30% of adults over the age of 65 years. In a study of 5621 people >65 years followed up for 5 years the risk of stroke was 8% for those with aortic sclerosis (leaflet thickening with no restricted motion) and 11.6% for those with stenosis (leaflet thickening with restricted motion).[29] The pathogenesis of AS related to CESS is believed to be formation of calcific or thrombotic emboli due to increased turbulence in the sinuses of Valsalva resulting in lysis and fragmentation of red blood cells with release of pro-thrombotic factors.[3,32] Embolization of calcium to the cerebral circulation in patients with aortic valve calcification has been noted on autopsy.[33]

Aortic valve morphology, leaflet thickening, and opening can be readily assessed with SSFP images through the LV outflow tract planes (three-chamber views). Imaging features of AS are leaflet thickening, decreased valvular excursion, post-stenotic dilatation, and concentric LV hypertrophy. On SSFP flow acceleration at the level of the leaflet tips is also a typical finding. If required, AS severity can be quantified from the phase-contrast flow study to derive a peak velocity measurement, which can then be used to estimate the transvalvular pressure gradient via the modified Bernoulli equation (pressure = 4 × peak velocity²).

### Left-sided prosthetic valvular pathology

#### Mechanical prosthetic valve (high risk) and bioprosthetic cardiac valve (medium risk)

Thrombus formation around prosthetic valves represents a significant complication with annual rates of 1% for bioprosthetic valves and 4% for mechanical valves.[5] The absolute annual risk of stroke is higher for prosthetic mitral valves (2–3.5%) than aortic valves (1–2%).[34] Prosthetic valves are safe at a field strength of 1.5 T and a reported to be safe up to 4.7 T, although the MRI safety and manufacturer’s literature should always be consulted.[35,36] Large, adherent thrombi or vegetations may be occasional findings in relation to a mechanical prosthetic valve but artefact often precludes complete assessment with CMR, and TOE is the reference standard technique. There are currently no studies in the literature specifically on the use of CMR to evaluate potential CESS from prosthetic cardiac valves.

#### Infective endocarditis vegetations (high risk)

There is a high incidence of ischaemic stroke with infective endocarditis (15–20%).[3] Most strokes occur early in the course of endocarditis and causes include rupture of mycotic aneurysms as well as septic and thrombotic emboli.[37] Mitral valve involvement carries a greater risk of stroke than aortic valve involvement.[38] The risk of stroke is low in the presence of vegetations <10 mm and in the absence of severe vegetation mobility.[39]

Imaging evidence of an oscillating valvular mass, abscess, or dehiscent prosthetic valve fulfils a major criterion for diagnosing infective endocarditis.[31] CMR is not indicated as a primary method for assessing valvular vegetations as it has inferior spatial resolution compared with TOE, which is considered the reference standard technique. As such CMR should not be used to exclude vegetation, but large lesions may be occasional findings on a CESS study and are important to identify to enable further targeted imaging assessment. Vegetations appear as a low signal mobile mass, most often located on the ventricular side of the aortic valve and atrial side of the mitral valve in the direction of regurgitated flow.

#### Aortic arch disease

#### Aortic atheroma (high risk if complex)

Aortic atheroma is a risk factor for cerebral ischaemia. In stroke patients over the age of 60 years, atheroma measuring >4 mm, ulcerated atheroma, and atheroma with mobile components have been classified as complex arch atheroma (CAA) and confer a fourfold greater risk of thromboembolism than small atheroma without complex features.[5,40,41] In comparison with post-mortem studies, TOE has a sensitivity of 91% and a specificity of 90% for atheroma detection.[2,42] CMR evaluation of aortic atheroma compares favourably to TOE when using DIR black-blood prepared pulse sequences with a correlation of approximately 80% between the two techniques.[5] The major advantage of CMR over TOE is an unrestricted imaging plane, which enables complete assessment of the arch with no “blind spots”. CMR displays mural thrombus as intermediate signal material on T1-weighted DIR black blood images, but it does not reliably detect calcification. The normal patent aortic lumen appears as a signal void, in this sequence, except in cases of slow flow, which can produce high intraluminal signal intensity. Contrast-enhanced MRA (CE-MRA) avoids flow-related artefact, as does SSFP, as these sequences are not reliant on inflow effects to generate signal. CE-MRA is a particularly robust technique for assessment of large, protruding arch atheroma and when used in conjunction with ECG-gating can generate an isotropic voxel dimension of 1–1.5 mm at 1.5 T.[43] Although not yet available for routine clinical use, high-resolution MRI (using ≥3 T systems) is emerging as the most promising method of non-invasively imaging and characterising aortic plaques, including identification of intraplaque haemorrhage and lipid-rich core, which are important indicators of lesion severity and propensity for rupture.[43]
Aortic dissection (medium/unclear risk)

Aortic dissection comprises a tear of the intima and inner layer of the aortic media with resultant intimomedial flap and formation of a double-channel aorta with communication between true and false lumens. Thoracic aortic dissection propagates into the carotid arteries in around 15% of cases and can cause a stroke secondary to narrowing of the true lumen or embolization of blood clots formed at the site of the tear.29 MDCT is the reference standard technique for assessment of suspected aortic dissection with sensitivity and specificity approaching 100%.29 CMR is not indicated in the emergency setting, although one study showed a sensitivity and specificity of 90 and 100%, respectively.44

Aortitis (medium/unclear risk)

Takayasu arteritis is an idiopathic, large-vessel vasculitis that is most frequently seen in young women of Asian descent. It affects the aorta, its major branches, and the pulmonary artery and is characterized by inflammation and fibrosis in the tunica media leading to aneurysm formation, segmental stenoses, and occlusions.29,43 Patients may present with neurological symptoms including stroke.

Aortitis is best detected on CMR using DIR black-blood imaging and manifests as diffuse or segmental thickening of the vessel wall, often with accompanying inflammatory changes in the peri-aortic fat. Recently, arterial wall enhancement using an inversion recovery late gadolinium enhancement technique has been described as a useful method to define active Takayasu’s arteritis and may offer improved specificity compared with serum markers.46

Figure 8 Takayasu arteritis in a 29-year-old woman with TIA-like symptoms. Sagittal, oblique, T1-weighted, black-blood image along the aortic arch showing diffuse mural thickening (arrows) indicative of a large vessel vasculitis, which can be seen to involve the great vessel origins.

Chronic or “burnt out” aortitis is characterized by aneurysmal and/or stenotic segments (Fig 8).

Conclusion

The investigation of patients with suspected CESS has traditionally been the mainstay of echocardiography. CMR is a powerful imaging technique that has rapidly evolved over the last decade. CMR has an emerging role in the assessment of many conditions associated with CESS and as such has the potential to provide a “one-stop-shop” evaluation of the entire cardio-cerebral axis during a single imaging session. This article has reviewed the role of CMR across the spectrum of causes of CESS with the aim of providing the reader with an overview of the capabilities of this technology.

References
