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PICTORIAL REVIEW

Native aorto-ostial coronary lesions on CT coronary angiogram

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ABSTRACT:

Aorto-ostial coronary lesions (AOLs) are important to detect due to the high risk of catastrophic consequences. Unfortunately, due to the complexities of these lesions, they may be missed on invasive coronary angiography. Computed tomography coronary angiogram (CTCA) is highly sensitive and specific in detecting AOLs, and has the additional advantage of demonstrating the surrounding anatomy. CTCA is particularly useful when assessing for AOL aetiologies in addition to atherosclerotic disease, e.g. Congenital anomalies, extrinsic Compression, Iatrogenic, Arteritis and Other, such as Thrombus, Embolism, Dissection and Spasm. This gives rise to “CIAO (TEDS)” as a proposed aide-mémoire and will form the structure of this pictorial review.

INTRODUCTION

Aorto-ostial coronary lesions (AOLs) are important to detect due to the likely higher risk of extensive myocardial infarction and sudden cardiac death compared with non-ostial coronary lesions. AOLs encompass pathology that results in >50% luminal compromise within the first 3 mm of the coronary artery orifice¹. AOLs can be missed on invasive coronary angiography due to variable coronary ostial take-off angle, deep ostial catheter intubation and C-arm angulation/fluoroscopic projection. Adjunct techniques of intravascular ultrasound (IVUS), fractional flow reserve (FFR) and coronary vasodilatory reserve (CVR) assist in AOL detection², but require specialised operator training and specific equipment that may not be widely available. Multislice computed tomography coronary angiogram (CTCA) offers an alternative diagnostic modality with a 95% sensitivity and 98% specificity in detecting AOLs².

The majority of AOLs are secondary to atherosclerosis and therefore usually accompanied by coronary atheroma elsewhere. Consequently, non-atherosclerotic aetiologies should be considered when an isolated AOL is identified. The clear advantage of CTCA over conventional

angiography is not only in volumetric delineation of AOLs but also the ability for surrounding anatomical assessment. This pictorial review will highlight the utility of CTCA in diagnosing AOLs of native coronary arteries and describe various non-atherosclerotic aetiologies. To this end, we propose an aide-mémoire “CIAO (TEDS)” (Table 1).

AOL AETIOLOGICAL REVIEW

Atherosclerotic disease

Atherosclerotic disease is the most common cause for AOL. Diffuse atherosclerotic changes are typically present, and ostial lesions are usually calcified and eccentric¹. Isolated atherosclerotic AOLs are far less common, occurring predominantly in females¹ (Figure 1). Atherosclerotic disease of the aortic root with mural calcification can also uncommonly lead to aortoostial stenosis, with the global extent of disease clearly delineated with CTCA (Figure 2).

Congenital

Coronary artery anomalies are varied and may comprise of aberration in origin, course and termination, as well as abnormalities of intrinsic coronary anatomy. Congenital AOLs predominantly relate to anomalous origins with inter

Table 1.

Non-atherosclerotic native coronary AOL aetiologies: "CIAO (TEDS)"		
C	Congenital compression (extrinsic)	Anomalous origin with inter arterial course Coronary artery hypoplasia/atresia Circumferential ostial sphincter-like muscle Coronary ostial ridge Aortic cusp hypoplasia Supravalvular aortic membrane Main pulmonary artery dilatation Aortic root pseudoaneurysm Sinuses of Valsalva aneurysm Aortic root abscess Tumour
I	Iatrogenic	Previous invasive coronary angiogram Aortic valve replacement Mediastinal radiation therapy Percutaneous pulmonary valve implantation
A	Arteritis	Inflammatory arteritis (Giant Cell, Takayasu's) Infective aortitis (bacterial, spirochetes, mycobacteria)
O	Other: Thrombus Embolus Dissection Spasm	Prothrombotic state Thromboembolism, septic embolus, tumour embolus Spontaneous, iatrogenic Vasospasm

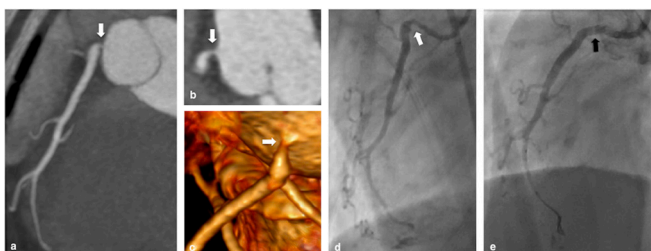
AOL, aorto-ostial coronary lesion.

arterial course resulting in a slit-like orifice (e.g. left main stem arising from right coronary cusp coursing between the aorta and pulmonary artery [Figure 3]). AOLs may also be intrinsic from ostial hypoplasia/atresia, be consequent to circumferential sphincter-like ostial muscle or be caused by an ostial ridge³. More rarely, aortic valve anomalies can compromise ostial flow, as may be seen in aortic cusp hypoplasia and with supravalvular aortic membranes⁴.

Compression

AOLs can result from compression and distortion by abnormal external structures. Severe dilatation of the main pulmonary artery is a rare but recognised cause of left main stem stenosis by direct compression or kinking of the ostium⁵ (Figure 4). Aneurysms and pseudoaneurysms of the aortic root, including isolated aneurysm of the sinus of Valsalva are another cause (Figure 5).

Figure 1. 52-year-old female presented with angina. (a) Double oblique CT MIP of the RCA demonstrates severe focal aorto-ostial stenosis (white arrow) (b) secondary to subtle concentric non-calcified plaque. (c) Volume rendering shows the change in the vessel calibre between the ostium and proximal segment. (d) Coronary angiogram before and (e) after (black arrow) treatment with RCA ostial stent insertion. MIP, maximum intensity projection; right coronary artery.



In the case of an aortic root abscess, extrinsic compression can result from the abscess itself or from a complicating pseudoaneurysm (Figure 6). Cardiac tumours, more commonly secondary metastases than primary neoplasm, can cause coronary ostial stenosis from extrinsic mass effect or tumour involvement of the aortic root (Figure 7).

Iatrogenic

Catheter cannulation during invasive angiography may rarely cause subacute or delayed onset AOL from endothelial trauma and subsequent stenosis in the absence of complicating dissection or pre-existing atheroma⁶.

Figure 2. 44-year-old male with familial hypercholesterolaemia presented with angina. (a) Axial and (b) multiplanar reformatted three chamber plane CTCA demonstrates diffuse calcification of the aortic root and ascending aorta with a large calcific ridge (white arrow). There is also severe RCA calcific ostial stenosis (black arrowhead). CTCA, CT coronary angiogram; RCA, right coronary artery.

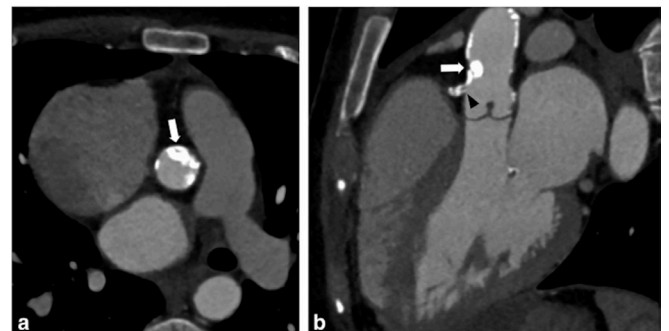
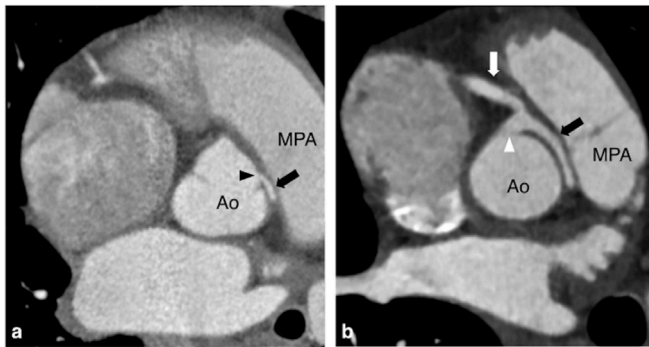


Figure 3. . Oblique axial CTs demonstrating (a) anomalous LMS (black arrow) arising from the right coronary cusp with inter arterial or "malignant" course between the aorta (Ao) and MPA, and associated slit-like orifice (black arrowhead). (b) Single coronary artery with large calibre ostium (white arrowhead) arising from the right cusp bifurcating into the RCA (white arrow) and LMS (black arrow). LMS takes an inter arterial course but is not associated with stenosis in this case. LMS, left main stem; MPA, main pulmonary artery; RCA, right coronary artery.



Delayed presentation coronary ostial stenosis following aortic valve replacement is an uncommon but well-recognised entity resulting histologically from reactive aortic mural fibrosis variably involving the intima and media, although the exact pathophysiological mechanism remains unclear.⁷

When the aortic root is included within the field during mediastinal radiotherapy, AOLs may result from accelerated atherosclerosis and chronic fibrosis secondary to radiation-induced endothelial injury⁸ (Figure 8).

Iatrogenic coronary compression during right ventricular outflow tract (RVOT) pre-stenting prior to percutaneous pulmonary valve implantation (PPVI) is a feared complication due to the close proximity of the coronary arteries to the pulmonary trunk. Pre-procedure CTCA is useful for identifying patients at risk of coronary compression and unsuccessful PPVI.⁹

Figure 4. . 42-year-old female with pulmonary arterial hypertension secondary to an atrial septal defect (not shown) presented with angina. (a) Coronal CT and (b) sagittal CT demonstrate aneurysmal dilatation of the MPA with compression of LMS ostium (black arrow). LMS, left main stem; MPA, main pulmonary artery.

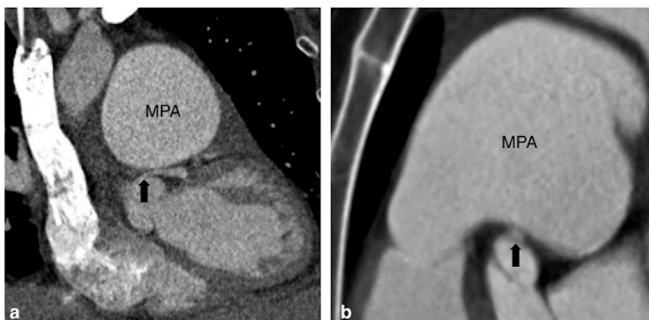
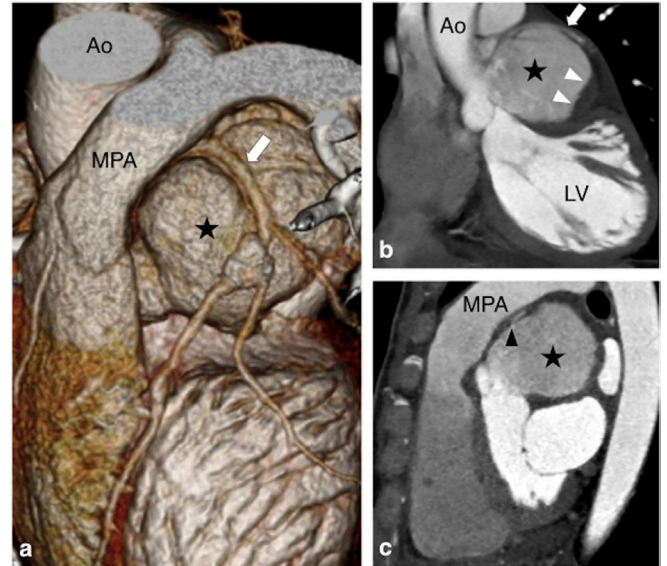


Figure 5. (a) Volume rendering, (b) coronal CT MIP and (c) sagittal CT of a 27-year-old female with a large sinus of Val-salva aneurysm (black star) involving the left coronary sinus. LMS ostium (black arrowhead) arising from the aneurysm is compressed against the MPA. The LMS (white arrow) is draped over the anterior aspect of the aneurysm. Note the laminar thrombus within the aneurysm (white arrowheads). LMS, left main stem; LV: left ventricle; MIP, maximum intensity projection; MPA, main pulmonary artery.



Arteritis

Arteritis is an infrequent cause of a solitary AOL but becomes a key consideration in non-atherosclerotic bilateral AOLs, particularly in the absence of relevant radiotherapy or surgical history. AOLs can result from aortic root aortitis with mural thickening causing isolated ostial stenoses, coronary arteritis or the combination of both. Arteritis can be divided into infectious and non-infectious aetiologies.

Figure 6. 75-year-old male presented with fever, raised inflammatory markers and acute onset aortic regurgitation. (a) Axial oblique CT MIP demonstrates a large pseudoaneurysm (black star) between the aortic root and left atrium causing compression of the entire left main stem (black arrow). Note the pericardial effusion (white star). (b) CT three chamber plane shows the aortic root abscess (white arrows) as the cause of the pseudoaneurysm. Note the LAD territory myocardial hypoattenuation (white arrowheads) indicative of subendocardial infarction. MIP, maximum intensity projection.

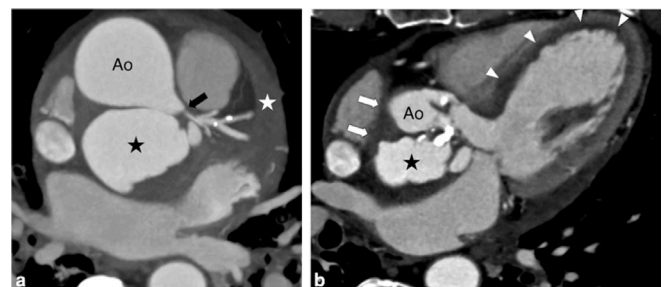


Figure 7. 70-year-old female with known bladder carcinoma presented with worsening dyspnoea and chest pain. (a) Pulmonary arterial phase axial CT and (b) delayed phase axial CT both demonstrate a large metastatic mass invading the right heart (black star) with encasement the RCA (black arrow) and (c) severe RCA ostial stenosis (black arrowhead). Note the intracardiac extension of tumour into the right ventricular outflow tract (white arrow). (d) Volume rendering with the anterior portion of the tumour (black star) carved away to highlight the RCA AOL. AOL, aorto-ostial coronary lesion; RCA, right coronary artery.

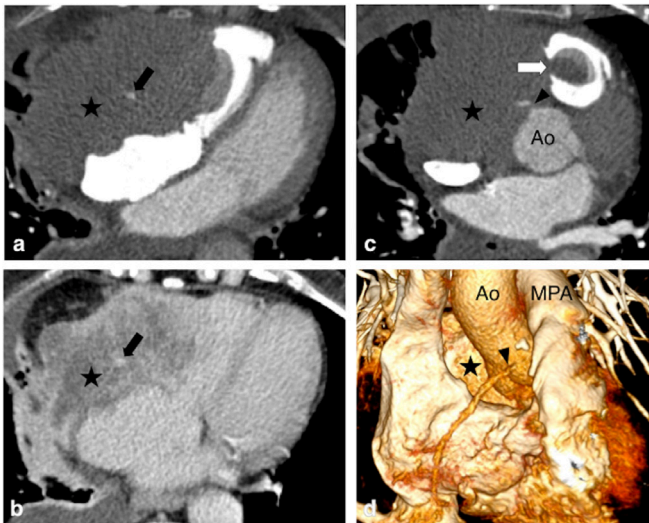


Figure 8. 42-year-old female with a distant history of lymphoma treated with mantle radiotherapy. (a) Axial non-enhanced CT demonstrates a porcelain ascending aorta with heavy calcification of the MPA, as well as (b) the great vessels (black arrows). (c) Axial CTCA shows a calcified aortic root with calcific ostial stenosis of the RCA (black arrowhead). (d) Note the sharp delineation of the irradiation field demarcated by the radiation induced pulmonary fibrosis (white arrows) on lung windows. CTCA, coronary angiogram; MPA, main pulmonary artery; RCA, right coronary artery.

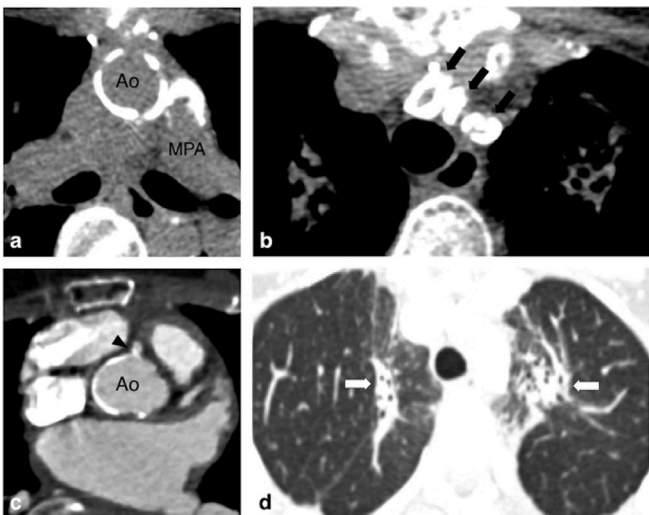
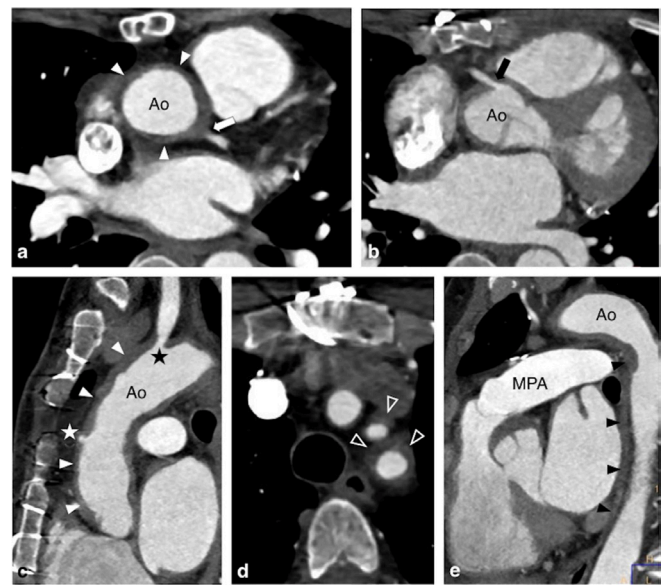


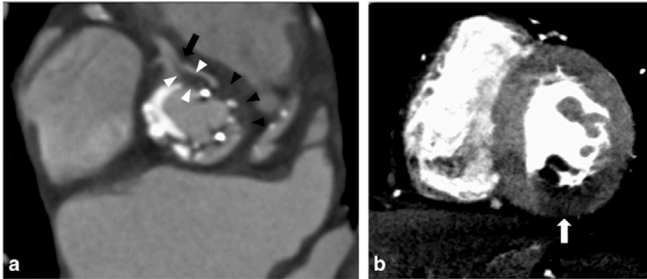
Figure 9. 47-year-old female with Takayasu's arteritis and previous CABGs presented with worsening angina. (a) Axial contrast-enhanced CT demonstrates soft tissue thickening at the level of the aortic root (white arrowheads) involving LMS ostium (white arrow) with severe stenosis, whilst (b) the ostium of the RCA (black arrow) remains preserved. (c) Note the sternotomy wires relating to previous CABG on sagittal CT. There is extensive mural thickening of the ascending aorta (white arrowheads) with stump of an occluded vein graft (white star) and narrowing of the brachiocephalic trunk ostium (black star). (d) Axial CT and (e) sagittal CT shows soft tissue involvement of the remaining great vessels (white-bordered arrowheads) and descending thoracic aorta (black arrowheads). CABG, coronary artery bypass graft; LMS, left main stem; RCA, right coronary artery.



The most common pathogens in infective arteritis include *Salmonella*, *Staphylococcal species* and *Streptococcal pneumoniae*.¹⁰ Acute bacterial aortitis results in the rapid development of typical CT findings of wall thickening, fat stranding, phlegmon or collections, which can rapidly progress to saccular aneurysm or pseudoaneurysm (Figure 6), and occasionally with gas in the aortic wall. Subacute or chronic infectious arteritis as seen in tuberculosis and tertiary syphilis, are rare in the developed world.

The large vessel arteritides, Giant Cell (GCA) and Takayasu Arteritis, are the most common causes of coronary ostial arteritis. Other less common non-infectious inflammatory causes include medium vessel vasculitis secondary to rheumatological disorders and HLA-B27-associated seronegative spondyloarthropathies, as well as idiopathic isolated aortitis.¹⁰ Findings on CT include diffuse wall thickening of soft tissue density demonstrating delayed enhancement, often with perivascular inflammatory stranding (Figure 9). Complications of stenosis, mural ulceration, aneurysm or dissection can occur.

Figure 10. 78 year old male with previous TAVR presented with acute chest pain and troponin rise. (a) Axial CTCA demonstrates TAVR peri-prosthetic thrombus (black arrowheads) with tongue like extension of the thrombus (white arrowheads) into the RCA ostium (black arrow). (b) Left ventricular short axis plane shows hypoattenuated mid inferior segment (white arrow), consistent with complicating myocardial infarction. CTCA, coronary angiogram; RCA, right coronary artery; TAVR, transcatheter aortic valve replacement.



Thrombus and embolus

In-situ thrombus as the cause of an AOL most commonly results from plaque rupture and initiation of the atherothrombotic cascade. In the absence of plaque rupture, prothrombotic states such as antiphospholipid syndrome, hyperhomocysteinemia, factor V Leiden and prothrombin G20210A gene mutations, may predispose to a *de novo* thrombotic AOL¹¹. Another cause is transcatheter aortic valve replacement (TAVR) peri-prosthetic thrombosis with coronary ostial extension (Figure 10).

Other sources of thromboembolism include post-infarct left ventricular apical thrombus, left atrial appendage thrombus and paradoxical systemic venous embolism across a patent foramen ovale. AOLs can also be caused by septic embolism from infective endocarditis¹². Lastly, aortic valve papillary fibroelastomas may embolise to cause coronary ostial occlusion, as a rare example of tumour embolic AOL¹³.

Figure 11. 65-year-old hypertensive male with acute onset severe chest pain and elevated troponin underwent emergency catheter angiography (a). The dissection flap was recognized during attempted selective catheterisation of LMS (black arrow). (b) CT volume rendering shows the complex dissection flap involving aortic root and ascending aorta (black arrowheads). (c) Axial CTCA demonstrates the dissection flap involving the LMS ostium with complicating *in-situ* thrombus (white arrow). CTCA, coronary angiogram; LMS, left main stem.

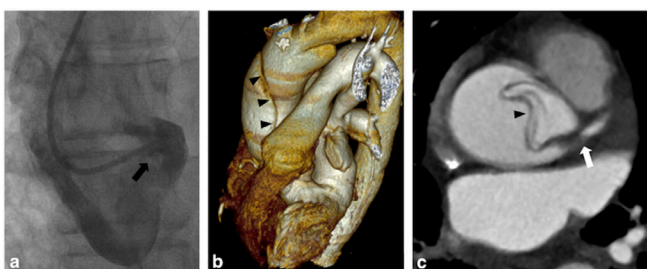
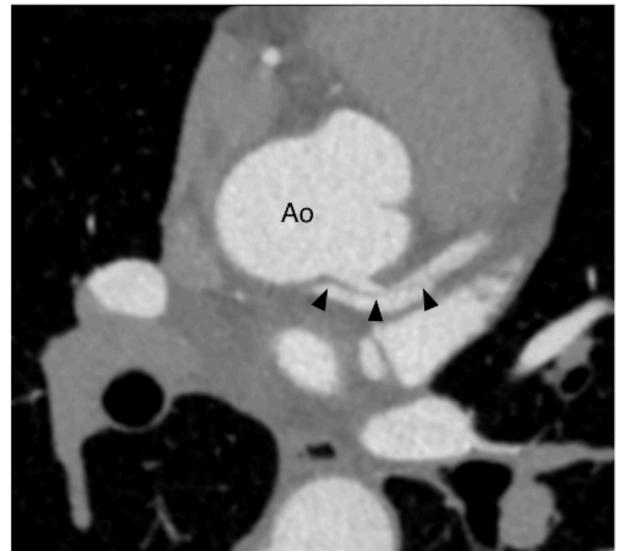


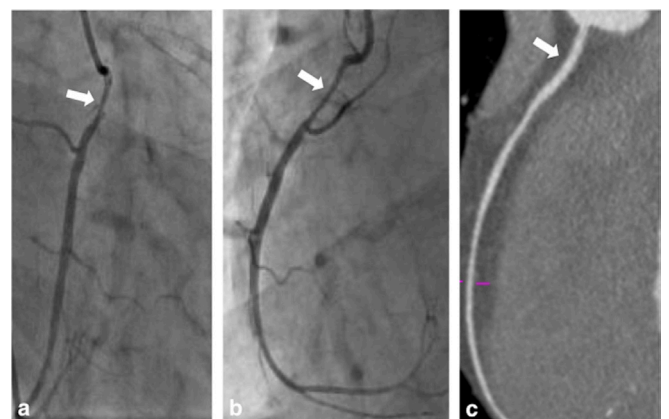
Figure 12. 47-year-old female with known fibromuscular dysplasia presented with acute chest pain. Axial CT demonstrates an acute dissection flap (black arrowheads) extending downstream from the ostium of the LMS. LMS, LMS, left main stem.



Dissection

In Stanford Type A acute aortic dissection, coronary ostial compromise may result from direct intimal flap extension or compression by the false lumen, causing extensive myocardial ischemia and acute coronary syndrome (ACS). The extent of dissection is reliably assessed on a CT thoracic aortogram with ECG-gated aortic root acquisition, although emergent invasive coronary angiography is likely the first test in the context of an ACS presentation (Figure 11).

Figure 13. 47-year-old male with HIV presented with atypical chest pain. (a) Coronary angiography shows reduced calibre of the RCA from the ostium to proximal segment (white arrow) with (b) some mild improvement post administration of intracoronary GTN. (c) CTCA performed 1 day later confirms vasospasm with curved multiplanar reconstruction of the RCA demonstrating return to normal calibre. CTCA, coronary angiogram; RCA, right coronary artery.



The coronary ostium may be primarily involved in spontaneous coronary artery dissection (SCAD), of which there are three recognised patterns as per the Saw angiographic SCAD classification¹⁴. Type 1 is an intimal tear with propagating medial dissection resulting in the classic appearance of an intimal flap dividing a false and true lumen (Figure 12). Type 2 is the more common pattern of dissecting medial intramural haematoma, presumed from a ruptured vaso vasorum, manifesting as diffuse luminal narrowing or occlusion of variable length. Type 3 is a focal intramural haematoma of less than 2 cm length. Type 2 and particularly Type 3 patterns simulate non-calcified atherosclerotic disease angiographically, and are therefore difficult to prospectively diagnose due to atherosclerosis being both a morphological mimic as well as a predisposing factor. Of the non-atherosclerotic predispositions, connective tissue disorders such as fibromuscular dysplasia are most commonly associated.¹⁴

Spasm

Coronary artery spasm is a reversible cause of an AOL. There are no specific findings on CTCA but the latter is useful in exclusion of differential aetiology and confirmation of vessel calibre

normalisation after initial catheter angiography (Figure 13). The exact pathophysiology is uncertain but is generally thought to be the result of a vasoconstrictor stimulus acting upon a hyper-reactive coronary artery. Coronary vascular hyper-reactivity may reflect a primary abnormality of the vascular smooth muscle cells, although endothelial dysfunction and other factors may contribute. Potential vasoconstrictive triggers include abnormal platelet activation, histamine, endothelin-1, hyperventilation and conflictingly, both sympathetic and vagal activity. It is likely that multifactorial mechanisms operate to manifest coronary spasm.¹⁵

CONCLUSION

Recognition of AOLs is crucial, consequent to the large area of myocardium at ischaemic risk. Although atherosclerotic disease is still the most common cause of AOLs, it is important to consider the non-atherosclerotic differential diagnoses as prompted by our aide-mémoire “CIAO (TEDS)”. As depicted by this pictorial review, CTCA is a valuable non-invasive diagnostic tool that has the capacity to interrogate associated disease processes beyond the confines of the coronary artery lumen.

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