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Review

Penetrating atherosclerotic ulcer of the aorta: A continuing debate

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Aortic penetrating atherosclerotic ulcer (PAU) is a relatively common incidental finding on thoracic computed tomography (CT) examinations. This is likely to relate to the steady increase in the number of CT examinations performed and also due, in part, to the increasing age of the general population. There is as yet no consensus on the management of incidental PAUs in asymptomatic patients. This article aims to review the literature and discuss the natural history, prognosis, and management of incidental PAU.

Introduction

Penetrating aortic atherosclerotic ulcer (PAU) is a poorly understood entity that may clinically mimic other causes of acute aortic syndrome but which has imaging features that are distinctly different.[1] With the advent of multidetector computed tomography (MDCT), the radiological diagnosis of PAU has increased, and it is frequently identified as an incidental finding at CT performed in elderly patients for unrelated conditions.[2] Relatively little is known about the natural history of PAU and so the significance of detecting such a lesion, especially in asymptomatic patients, is debatable and no consensus exists on the appropriate management of this group of patients.[3,4]

Incidence

The true incidence of PAU in the population is unknown. PAU usually affects individuals in their seventh decade of life or older, with concomitant severe atherosclerosis involving the aorta and multiple co-morbidities, which make them poor candidates for treatment.[5] Common comorbidities include hypertension, diabetes, chronic obstructive pulmonary disease (COPD), cardiac and renal failure. There is also usually a long history of smoking and hyperlipidaemia.[3] In the absence of atherosclerosis, a PAU can occur in a younger patient with connective tissue disorder or after mycotic plaque rupture. The incidence of PAU in patients presenting with acute aortic syndrome is estimated between 2.3–7.6%.[6–8]

Definition and pathogenesis

PAU is part of the spectrum of acute aortic syndrome: symptomatic aortic aneurysm, aortic dissection, intramural haematoma (IMH), and PAU.[9] Clinically, it may simulate an aortic dissection causing severe acute chest pain radiating to the inter-scapular area. Although there is considerable similarity in the initial clinical presentation of these disorders and all can rapidly progress to life-threatening complications, their pathogenesis differs. PAU was first described by Shennan in 1934[10] but was subsequently recognized by Stanson et al.[7] as a distinct pathologic entity. According to the definition of Stanson et al., in PAU an atherosclerotic plaque ulcerates and disrupts the internal elastic lamina, burrowing through the intima into the aortic

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media. The media is exposed to pulsatile arterial blood flow, which can lead to haematoma formation within the media of the aortic wall. The plaque may precipitate a localized dissection of the media associated with a variable amount of haematoma in the aortic wall, may break through into the adventitia to cause a pseudoaneurysm, or may rupture. Instead, aortic dissection starts with a tear in the intima but eventually contains two tears: one entrance tear from the lumen through the media and one re-entry tear from the media back into the lumen. In contrast, IMH does not involve an intimal tear but is caused by a rupture of the vasa vasorum of the media, which results in mural haematoma contained within the aortic wall without direct communication to the aortic lumen.[8] Complications of PAU include development of localized IMH due to erosion of vasa vasorum by the ulcer, pseudoaneurysm, progression to overt aortic dissection, or rupture.[2]

It should be acknowledged that most of the literature on PAU is based on imaging features rather than on histological confirmation. In previous reports, authors have identified PAUs as a cause of IMHs, whereas others have considered PAUs and IMHs to be distinct, unrelated entities.[8,11,12] Indeed, a lack of agreement in the terminology used in the literature to describe IMH and PAU is evident. In this article, the authors have used the term "penetrating atherosclerotic ulcer" to describe the classic description of penetrating atherosclerotic ulcer by Stanson et al.,[7] with or without IMH.

Radiological diagnosis

A clue to the diagnosis of PAU can often be provided from the chest radiograph (Fig 1). In a series of 16 patients with a PAU[1] the chest radiograph was abnormal in all cases, with diffuse ($n = 14$) or focal ($n = 2$) enlargement of the descending thoracic aorta. Thirteen out of 16 patients had experienced chest or back pain. Other plain radiographic findings include a widened mediastinum, pleural effusion, and deviated trachea. However, in the majority of asymptomatic patients the chest radiograph is likely to be normal. Further imaging is required in order to obtain a definitive diagnosis.

CT has largely replaced angiography in the assessment of the thoracic aorta due to its ready availability, rapid examination time, and the added advantage of comprehensive anatomical evaluation of the thoraco-abdominal aorta and surrounding structures with two and three-dimensional image reconstruction.[4] PAU is diagnosed at CT as a focal contrast-filled, outpouching of the aorta in the absence of a dissection flap or false lumen (Figs 2 and 3), which may be surrounded by concomitant subintimal haematoma located beneath the frequently calcified and inwardly displaced intima.[13] The ulcer is often associated with localized thickening of the aortic wall. There is usually associated extensive aortic calcification and diffuse atherosclerosis. PAUs have been described along the whole length of the aorta but most commonly involve the mid and distal descending thoracic aorta.[3] The infra-renal aorta accounts for approximately one-third of cases.[4] Magnetic resonance imaging (MRI) can also be used to diagnose PAU[14,15]; however, this may be impractical in the acute setting due to availability and relatively lengthy examination time. Transoesophageal echocardiography shows a crater-like ulcer with rough edges, commonly with associated extensive aortic plaque. It has been shown to have high sensitivity and specificity and offers the added advantage of portability; however, it does not allow examination of the entire aorta and is operator dependent. Also, it is an invasive examination with a small but significant complication rate.[16] Table 1 summarizes the characteristics of PAU.

Natural history: the evidence

There is a paucity of studies on PAUs. Even fewer studies have investigated the natural history of asymptomatic PAU, which remains poorly defined.[2,12,17]

Location of the ulcer in the ascending aorta and acute pain are believed to be the most important factors influencing the decision to offer treatment.[18] PAU involving the

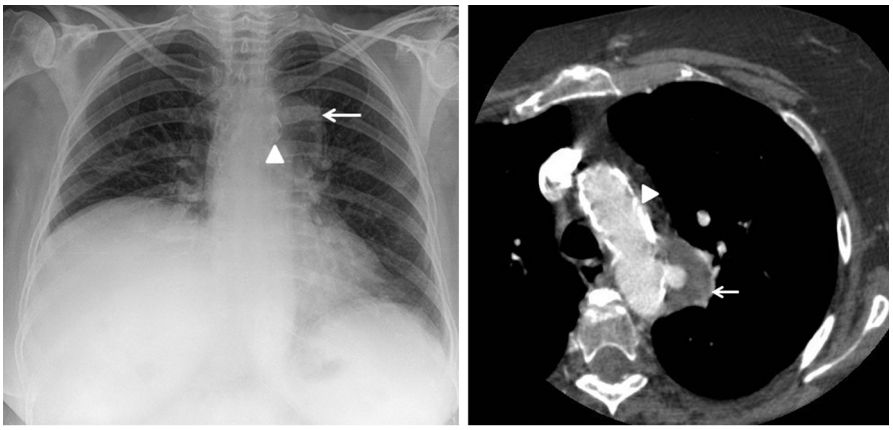

Figure 1 (a) Chest radiograph showing focal dilatation of the aortic arch (arrow), the aortic calcification is demonstrated medially. (b) Transaxial CT imaging showing the penetrating ulcer with associated IMH.