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Review

Multimodality imaging of pulmonary infarction

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ABSTRACT

The impact of absent pulmonary arterial and venous flow on the pulmonary parenchyma depends on a host of factors. These include location of the occlusive insult, the speed at which the occlusion develops and the ability of the normal dual arterial supply to compensate through increased bronchial arterial flow. Pulmonary infarction occurs when oxygenation is cut off secondary to sudden occlusion with lack of recruitment of the dual supply arterial system. Thromboembolic disease is the commonest cause of such an insult but a whole range of disease processes intrinsic and extrinsic to the pulmonary arterial and venous lumen may also result in infarcts. Recognition of the presence of infarction can be challenging as imaging manifestations often differ from the classically described wedge shaped defect and a number of weighty causes need consideration. This review highlights aetiologies and imaging appearances of pulmonary infarction, utilising cases to illustrate the essential role of a multimodality imaging approach in order to arrive at the appropriate diagnosis.

1. Introduction

Pulmonary infarction is due to coagulative ischaemic necrosis [1]. This process occurs when parenchymal perfusion fails to meet the tissue requirements due to occlusive insults that are either intrinsic or extrinsic to the vascular lumen. Normally, dual arterial supply comprised of deoxygenated blood in the pulmonary arteries and oxygenated blood in the highly adaptable bronchial arteries protects against infarcts. The risk of ischaemic necrosis is especially increased in perturbations of blood flow that dually impede this system, be it congestive heart failure, pulmonary venous hypertension, or diminished bronchial arterial flow in systemic arterial hypotension [2]. Less commonly infarction may result from perturbations in pulmonary venous flow [3,4]. A 'complete' infarct is characterised by irreversible ischaemic injury, wherein incident capillary ischaemia increases vascular permeability with subsequent bronchial artery reperfusion leading to alveolar haemorrhage and necrosis ensuing hereafter. The end-result is a fibrotic scar. An infarct presents macroscopically as a dark necrotic area with a narrow rim of hyperaemia and inflammation, presenting microscopically as a 'ghost-like' architecture with an outer perimeter of cellular infiltrate (Fig. 1) [1,5]. The infarct can also be 'incomplete' when there is transient haemorrhagic congestion but no other acute or chronic parenchymal sequelae – the parenchymal opacity resolves in a matter of days [6,7].

By virtue of the pathophysiological mechanism, arterial infarcts are peripheral, occurring in areas supplied by medium and small sized arteries [8]. The acute infarct is a wedge-shaped (less frequently nodular) pleurally based parenchymal opacity ('Hampton hump') that forms an obtuse angle with the visceral pleura, with the apex pointing towards the pulmonary hilum and centred on a bronchovascular bundle, whereas a pulmonary venous infarction is paraseptal in distribution (Fig. 2) [6,9,10]. Less typical features of arterial and venous infarcts are increasingly reported with a move to raised suspicion of infarction with a whole array of imaging appearances [11]. Infarcts may be solitary or multiple, temporally uniform or variegated much depending on the inciting event, and characteristically the airspace opacity should diminish in size over time ('melting sign') with complete resolution or leaving residual plate-like scarring and focal pleural thickening.

Timely diagnosis of pulmonary infarction is important in order to relieve the inciting event and avert further respiratory and

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vascular compromise. This pictorial review aims to demonstrate imaging features of pulmonary infarction using a multimodality approach in order to illustrate the spectrum of aetiologies that may result in infarction. Although computed tomography (CT) currently forms the mainstay of diagnosis the use of ancillary imaging modalities is often necessary.

2. Imaging modalities

2.1. Computed tomography

The wedge-shaped, broad pleurally based 'Hampton hump' frequently has a truncated apex and a convex border with less common findings including internal air lucency, linear stranding from apex to hilum and thickened vessels leading to the apex of the opacity (Table 1) [12]. Contrast-enhanced CT pulmonary angiography (CTPA) may demonstrate decreased enhancement within consolidated lung (Fig. 3); this is present in 95% of infarcts but can also be seen in collapse, pneumonic consolidation and neoplasm [12,13]. Central lucency within peripheral consolidation is particularly indicative of pulmonary infarction ('bubbly consolidation') (Fig. 3) [14] but other causes for these appearances include cavitation, cysts and dilated airways [15]. Although cavitation within infarcts is often due to bland necrosis, the presence of superinfection of established bland infarction or primary septic emboli can give a similar appearance [16,17]. CT is particularly helpful for the detection of complications of infarcts, including potentially detrimental sterile or purulent involvement of the mediastinum, pleural space, remaining parenchyma and airways, or the thoracic cage [18–20].

Dual Energy CT lends promise for the visualisation of pulmonary infarcts and vascular occlusions [21]. Contrast-enhanced data sets are acquired at two energy spectra and reconstructed to form a conventional CTPA plus a static iodine map, the latter representing a surrogate measure of microvascular circulation and perfusion (Fig. 4) [22,23]. The functional iodine map increases diagnostic accuracy by detecting more peripheral occlusions than conventional CTPA [24]. In cases of pulmonary arterial occlusion without infarction the iodine map depicts relative oligaemia due to persistent perfusion via the bronchial arteries; infarction on the other hand is characterised by complete segmental absence of iodine due to absent perfusion and a corresponding morphological abnormality [25]. The segmental distribution aids in the differentiation of infarction from other opacities with low iodine content such as tumour, pneumonia, or even streak artefact. Dynamic Perfusion CT is another technique with potential future applications in the assessment of infarction but its use has hitherto remained limited due to radiation concerns [26]. At present CTPA remains the principal diagnostic tool with the dual energy technique potentially providing a future one-stop morphological and functional test.

Table 1
Salient imaging findings for pulmonary arterial infarcts by modality.

Radiography	Usually non-specific parenchymal opacity Wedge-shaped pleurally based opacity: Hampton hump Pleural effusion (small, unilateral), elevated hemidiaphragm (volume loss), atelectasis
Computed tomography	Hampton hump Internal lucency in peripheral consolidation: bubbly consolidation Thickened vessels leading to apex Pleural effusion (localised or diffuse on Ipsilateral side) Cavitation (<10%)
Dual energy computed tomography	Contrast enhanced: hypoenhancing centre Adjacent or ipsilateral pleural effusion Perfusion defect on iodine map with correlating parenchymal opacity
Ultrasound	Wedge-shaped, pleurally based area of reduced echogenicity Hyperechoic centre (reverberation artefact) Late contrast-enhancement
Magnetic resonance imaging	Hyperacute: hyperintense T2WI and hypointense T1WI Acute (up to 1 week): hyperintense T1WI Hyperintense signal on both T1WI and T2 WI compared with tumour Perfusion defect: visualised with static and/or dynamic contrast enhanced MRI
Ventilation/perfusion scintigraphy	Matched ventilation and perfusion defect with radiographic parenchymal opacity – 'Triple-match'
Single positron emission tomography	Consolidation in peripheral interface between severely decreased and relatively preserved perfusion areas
Positron emission tomography	FDG-avid rim along the infarct periphery Relative central photopenia Alternative appearance: diffuse uptake

2.2. Magnetic resonance imaging

Imaging appearances of infarcts on magnetic resonance imaging (MRI) vary according to signal characteristics of the aging blood that has accumulated within the alveoli [27]. Acute infarcts

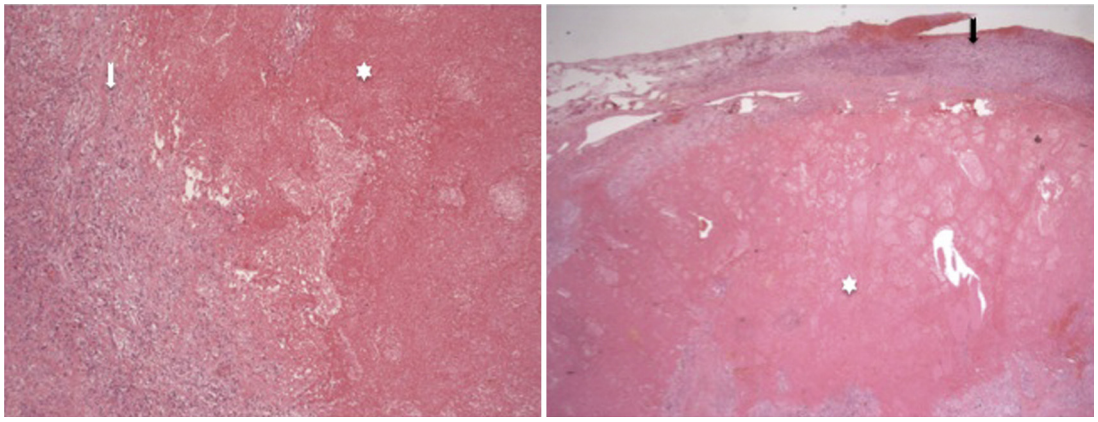

Fig. 1. Haematoxylin and eosin stain images of two different arterial infarcts. Left image (original magnification 50×) is an organised pulmonary arterial infarction (white star) with a chronic inflammatory infiltrate in the margin (white arrow). Right image (original magnification 16×) shows a peripheral infarct (white star) with pleural thickening (black arrow).