Case presentation

A 42-year-old male smoker with a 15-pack year history presented to the emergency department with a 2-day history of left flank pain and progressive lower limb pain on exertion over several months. He had no urinary or gastrointestinal symptoms. Examination of the abdomen was normal as was full blood count and routine biochemistry. He was normotensive. He was on no medication. His past medical history was unremarkable other than treatment for stage 1 seminoma (pT1 Mx Nx) 9 years previously. A CT of the abdomen and pelvis was performed. Select images are shown (Fig. 1, 2) with coronal MIP images from a subsequent CT angiogram (Fig. 3).

Imaging findings

Transverse and coronal image from a CT of the abdomen and pelvis in the arterial phase demonstrate a wedge-shaped area of low attenuation in the mid left kidney. The kidneys are otherwise enhancing normally despite the absence of contrast in the abdominal aorta. Contrast is identified in the superior mesenteric artery (arrow). Coronal reformats demonstrate a cut-off with no contrast in the mid or distal abdominal aorta. Extensive calcification is present in the wall of the abdominal aorta. A CT angiogram was performed and the MIP reformats are shown. Total proximal occlusion of the abdominal aorta with extensive collateralization is demonstrated. Again noted is the presence of mural calcification of the abdominal aorta and common iliac arteries.

Diagnosis

A diagnosis of radiation induced total occlusion of the abdominal aorta with segmental infarction of the left kidney was made. The patient had undergone radiotherapy as part of his treatment for testicular carcinoma 9 years previously. The patient was referred to vascular surgery and underwent an aorto-iliac bypass.

Discussion

Radiation Induced arthrosclerosis is a well-recognized complication of radiotherapy with the majority of cases reported in the extracranial vessels following irradiation for head and neck malignancy (1). Radiation induced arterial injury has several manifestations depending on the time interval from treatment (2). In the acute setting arterial rupture or thrombosis can occur. Accelerated atherosclerotic disease is a later complication. Clinically and radiologically it can be difficult to discriminate between radiation induced disease and atherosclerosis. The diagnosis is made after considering a number of factors. The presence of isolated vessel disease in the radiation field with no other evidence of atherosclerotic disease would be suggestive, as would presentation at a young age. Vascular risk factors are often present and are associated with an increased risk of development of radiation induced vascular injury.

Butler et al (2) described 3 phases of vessel injury following irradiation. The first phase consists of initial mural thrombosis within the first 5 years following irradiation. The second phase is fibrotic vessel occlusion up to 10 years following irradiation. The final stage is a predisposition to the development of atheroma with periarterial fibrosis associated with a latent interval of 20 or more years.
Different theories for the pathogenesis of arterial occlusion have been proposed with debate over whether radiation causes direct fibrosis or causes molecular changes and subsequent fibrosis (3). Various cytokines have been implicated and suggested to cause endothelial and fibroblast proliferation. Histological analysis of the vessel wall shows endothelial proliferation and degeneration of the media with subsequent cystic medial necrosis. Damage to the vasa vasorum is often present, with resultant fibrosis of the vessel wall and stenosis of the vessel.

Other vascular risk factors are often present in these patients although it is the lack of widespread atherosclerotic disease that often permits the diagnosis of radiation induced vascular injury; with disease limited to the field of radiation. Radiation dose is implicate, a study of 14 cases of radiation induced arterial occlusion described a range of doses from 47 to 70 Gray with standard fractionation (4).

Therapy for radiation induced arterial disease includes medical, percutaneous intervention and surgical treatments. Medical treatment involves the aggressive treatment of vascular risk factors. Surgical therapy is often advocated, however the presence of fibrosis secondary to radiation can hinder dissection and careful pre-operative planning is required (5). In non-occluded vessels percutaneous treatment is therefore preferable (6). Restenosis is a frequent problem and regular follow-up imaging is recommended.

Radiation induced aortic occlusion is a relatively uncommon finding usually presenting years after therapy. The presence of vascular risk factors such as smoking, diabetes and hypertension are typical. The key radiological finding of radiation-induced arteritis is the limitation of vascular disease to the radiation field.

References