

IMAGES IN RHEUMATOLOGY

Giant cell arteritis after radiation therapy: cause or coincidence?

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BACKGROUND

Giant cell arteritis (GCA) is the most common form of primary systemic vasculitis in the elderly. It predominantly involves the temporal arteries but can also affect the aorta and its main branches¹. Radiationinduced arteritis (RIA) may occur as a result of radiation therapy (RT), consisting of a quick-onset localized vasculitic phenomenon with arterial inflammation and thickening, mimicking GCA^{2, 3}. Moreover, a potential association between malignancy and GCA has also been reported, with a prevalence of around 7% and a slight predominance of haematological disorders (including myelodysplastic syndromes), although a solid relation could not be proved yet⁴⁻⁶.

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CASE REPORT

An 86-year-old woman was referred to our rheumatology clinic with a six-month history of weight loss (10kg), asthenia, and scapular and pelvic girdle pain with morning stiffness. She denied headache, visual symptoms or jaw claudication. She had a background of childhoodonset asthma, dyslipidaemia, coronary artery disease and a slow-growing lung mass in the right superior lobe, identified four years prior, which on fluorodeoxyglucose (FDG)-positron emission tomography (PET) assessment showed a standardized uptake value (SUV) increase from 1.5 to 3.5 units over three years. Due to the suspicion of indolent lung carcinoma and difficulty to obtain confirmatory histology by interventional procedures, treatment with stereotactic body RT was performed (Figure 1). She received a total of 55 Gray/units in five fractions, every other day over nine days, with subsequent regression in the lung mass diameter (from 4.3 to 1.6cm). One month after finishing RT, the patient began feeling the abovementioned symptoms. On physical examination, she struggled to walk and had bilaterally impaired shoulder abduction, both because of severe stiffness and mild muscle weakness (probably because of disuse). Systolic blood pressure was asymmetrical



Figure 1. Image of the stereotactic body radiation therapy dosing plan applied to the suspected carcinoma mass located in the superior lobe of the right lung. Light blue area corresponds to 20% isodose. Although there is proximity with the vascular structures of interest, the radiation dosing at the left carotid, axillary and subclavian arteries is supposed to be residual.



Figure 2. Fluorodeoxyglucose positron emission tomography images, depicting abnormal metabolic activity in the topography of both axillary arteries (A), with a maximum standardized uptake value (SUV) of 3.4 units, suggestive of vasculitis. No metabolic activity was depicted in the follow-up exam, six-month after glucocorticoid initiation, neither on arteries nor on the pulmonary mass (B).



Figure 3. Ultrasound images of the right (A) and left (B) axillary arteries, in longitudinal view, showing a bilateral halo sign (arrows) with a maximum intima-media complex thickness (IMT) of 1.35 mm measured on the left. Longitudinal view of the right (C) and left (D) common carotid arteries depicting a bilateral halo sign (arrows) with a maximum IMT of 1.8 (bilaterally), but also with concomitant atherosclerotic plaques (asterisks). Longitudinal view of the left subclavian artery (E), depicting a halo sign (arrows) with a significant IMT increase, maximum of 2.12 mm. Longitudinal view of right common superficial temporal artery (F) not depicting a halo sign nor increase in the IMT. The halo sign is usually considered for IMT measurements \geq 1 mm in the carotid, subclavian and

on the upper limbs (131mmHg right; 141mmHg left). Blood tests showed normocytic/normochromic anaemia (Hb 8.3g/dL) and increased acute phase reactants (APR; ESR 107mm/hr and CRP 1.7mg/dL). A follow-up FDG-PET showed no activity in the pulmonary lesion but identified an increased uptake in both axillary arteries (Figure 2), with a maximum SUV of 3.4 units. There was no uptake in the shoulder or hip joints. Ultrasound of the temporal, axillary, subclavian and common carotid arteries was performed, showing a halo sign in both axillary and common carotid, as well as left subclavian (Figure 3), compatible with the diagnosis of GCA with exclusive extracranial involvement. Treatment with prednisolone 40mg/day was started with significant improvement and sustained clinical remission over the following four months, during which prednisolone was tapered to 12.5mg/day. No glucocorticoid-sparing therapy was used and there was no clinical indication to start chemotherapy APR normalized and no metabolic activity was depicted by FDG-PET on the lungs or large vessels at six months (Figure 2).

DISCUSSION AND CONCLUSION

We present the case of a large vessel vasculitis resembling a typical primary systemic vasculitis, which we believe to be the main aetiology of our patient's condition. However, in our case, other potential causes cannot be neglected. RIA is a rare complication of RT that tends to be limited to the radiated site but may also affect the surrounding healthy tissues⁷. To the best of our knowledge, no cases of RIA with GCA/ polymyalgia rheumatica symptoms or concomitant increase in APR have been previously described. Although our patient presented with elevated APR and vasculitic changes in the left subclavian, carotid and axillary arteries and the radiated lung lesion was on the right, the temporal

coincidence between events and the relative proximity between structures does not allow exclusion of RIA as the underlying contributor for this patient's condition. Moreover, paraneoplastic GCA in this case is a less likely possibility given the improvement in the lung lesion documented by PET when the symptoms started, as well as the good response to glucocorticoid therapy observed during follow-up. This case draws attention to RIA and paraneoplastic syndrome in the differential diagnoses of GCA.

Key message: Radiation-induced arteritis and paraneoplastic phenomenon should be taken into account when diagnosing a large vessel vasculitis.

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