Cushingoid facies on ^{18}F -FDG PET/CT

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Received: 19 December 2013 / Accepted: 17 March 2014 / Published online: 16 April 2014
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A variety of physiological artefacts can mimic pathology or even result in unreliable diagnostic images. An uncommon artefact can be seen in patients with glucocorticoid-induced Cushing syndrome. This 10-year-old girl, diagnosed with a mediastinal T-cell lymphoma, showed solitary mediastinal activity on baseline ^{18}F -FDG PET (a). She was treated with chemotherapy and high doses of oral glucocorticoids, which eventually led to clinical signs of Cushing syndrome, including moon face and buffalo hump. Treatment of her disease was complicated by biopsy-proven cerebral fungal infection (aspergillosis) suggested on MRI. A second ^{18}F -FDG PET/CT scan, performed to assess the extent of aspergillosis, showed increased heterogeneous regional uptake in the cutaneous tissues of the head, neck and shoulders (b) with normal findings on low-dose CT, and relatively reduced hepatic ^{18}F -FDG uptake. This abnormal ^{18}F -FDG distribution hampered correct scan reading and the scan was therefore decided not to be of diagnostic value for assessment of aspergillosis.

This abnormal uptake pattern can be explained by glucocorticoid-induced promotion of the differentiation preadipocyte into mature adipocytes in central fatty tissue [1], which in turn have relatively high GLUT1 expression [2]. Reduced liver uptake can be due to prolonged use of

glucocorticoids, inducing increased hepatic glucose output [3], resulting in increased “washout” of ^{18}F -FDG.

If changes in ^{18}F -FDG uptake pattern due to high doses of glucocorticoids are seen, caution should be exercised in interpreting ^{18}F -FDG PET scans because of possible artificial uptake reduction within regions of disease activity.

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