Crossed cerebellar diaschisis demonstrated by [18]F- FDG-PET/CT

To the Editor: It is important to recognize significant metabolic patterns like crossed cerebellar diaschisis (CCD) in oncology patients undergoing fluorine-18-fluorodeoxyglucose positron emission tomography/computed tomography (^{18}F-FDG-PET/CT) scans. CCD is a known complication of stroke both in acute and chronic phases. It occurs due to interruption of the cortico-ponto cerebellar tracts, leading to depression of regional neuronal metabolism and blood flow [1] resulting in hypoperfusion and hypometabolism on cerebral perfusion and metabolism studies, respectively.

As an example, we present a 55 years old case of papillary serous cystadenocarcinoma of the right ovary, referred to us for a ^{18}F-FDG-PET/CT study following completion of treatment to rule out the possibility of residual and/or metastatic disease. She had been treated with total abdominal hysterectomy, bilateral salpingo-ophrectomy and infra-colic omentectomy followed by 6 cycles of carboplatin and paclitaxel based chemotherapy. She was a known hypertensive, taking regular antihypertensive medication. The ^{18}F-FDG-PET/CT scan was done approximately 60min following the intravenous injection of 370MBq of ^{18}F-FDG. The patient was positioned supine with hands down on a whole body Full Ring PET/CT camera (Discovery STE16-GE, USA camera). Initial scout was obtained to localize acquisition from the vertex to mid thigh. A low dose CT of this area was done for attenuation correction and co-registration, followed by the usual 3D PET emission scan at 2min/bed position for 7 bed positions. Images were reconstructed using a 3D VUE algorithm, which is similar to reconstructive algorithms, used by GE healthcare and viewed on the Xeleris workstation (GE) using the volumetric protocol. Visual evaluation of the maximum intensity projection (MIP) image revealed physiological tracer distribution in the body but on changing the window for brain evaluation, relatively decreased ^{18}F-FDG uptake was noted in the right cerebellar hemisphere (Fig. 1). Evaluation of transaxial slices of the brain revealed reduced ^{18}F-FDG uptake in the left basal ganglia region (Fig. 2A and C) and diffusely decreased ^{18}F-FDG uptake in the right cerebellar hemisphere (Fig. 3A and C). Correlative CT revealed a hypo-dense lesion in the left basal ganglia, left internal capsule and left peri-ventricular region (Fig. 2B) suggestive of an ischemic aetiology, while the cerebellar hemispheres did not reveal any structural abnormality (Fig. 3B). A detailed history revealed that she had suffered a left middle cerebral artery infarct 8 years ago with paresis of right side of the body and deviation of the face toward left. The left basal ganglia hypometabolism was thus the result of the ischemic episode while the right cerebellar hypometabolism represented crossed cerebellar diaschisis.

Figure 1. Posterior maximum intensity projection image showing right cerebellar hypometabolism (arrow).
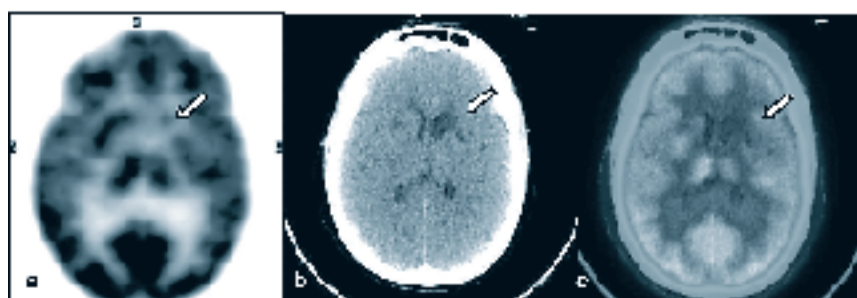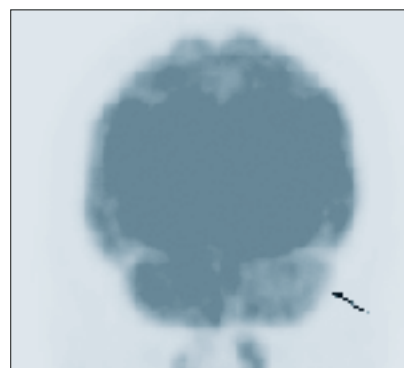

Figure 2. Transaxial (a) plain PET (b) plain CT and (c) fused PET/CT at basal ganglia level showing ^{18}F-FDG hypometabolism in the left basal ganglia region corresponding to a well defined hypodense area (arrow) with mild intraventricular dilatation on CT.
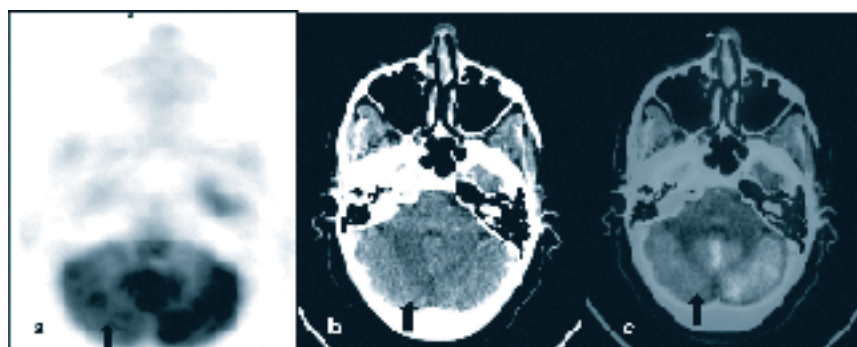

Figure 3. Transaxial (a) plain PET (b) plain CT and (c) fused PET/CT at the cerebellar level showing diffuse ^{18}F-FDG hypometabolism in the right cerebellar hemisphere while the corresponding CT does not show any abnormality (arrow).

Diaschisis (from Greek words: dia = throughout and skizo = split, meaning «shocked throughout») is a sudden loss of function in a portion of the brain connected to but at a distance of a damaged area. The site of the originally damaged area and of the diaschisis are connected to each other by neurons. The term diaschisis was coined by von Monakow in 1914 [2]. Nowadays, CCD is considered to be a result of depression of regional neuronal metabolism and cerebral blood flow caused by dysfunction in an anatomically separate but functionally related neuronal region [1]. Many connections between the cerebral hemispheres and the cerebellum exist with the cortico-ponto-cerebellar being the most numerous and accounting for 40 times all other afferent sources combined [3]. The first order neurons arrive in the ipsilateral pons to synapse with the second order neurons before they cross to the contra-lateral cerebellar hemisphere via the middle cerebellar peduncle. Injury at any point can result in decreased metabolism of the contra-lateral cerebellar hemisphere. Interruption of these tracts can occur from a range of injuries including tumour (especially malignant frontal lobe tumours that extend onto the parietal lobe [4]), stroke, gliosis, epilepsy or trauma, with supra-tentorial ischemic strokes after middle carotid artery occlusion being the most common aetiology. CCD can also be seen in patients with final infarcts and also in patients with initial hypo-perfusion recovering without morphologic sequelae [1, 5-7]. In the acute phase (i.e. within 3h of stroke), CCD indicates impaired tissue function due to penumbral flow (defined by CBF values below 20mL/100g min^{-1}) and is reversible. Later than 24h after stroke, persisting CCD becomes a surrogate marker of tissue damage, independent of cortical (re)perfusion [7].

Baron et al. in 1980, first described CCD in a positron emission tomography (PET) study and demonstrated matched reduction in cerebral blood flow and oxygen extraction fraction, in the contra-lateral cerebellum in patients with supratentorial ischemic stroke [8]. Although the number of patients is small in series already published and the incidence of CCD on PET in acute stroke is unclear, several series of mixed chronicity of stroke have reported frequency of CCD after stroke greater than 60% [9-11] and a little lower as detected by the perfusion weighted magnetic resonance imaging [12]. CCD has been demonstrated on ^{18}F-FDG-PET up to 20 years after a cerebro-vascular accident [13] indicating the irreversibility of the lesion once the acute phase is over. This ^{18}F FDG-PET/CT study showed abnormal findings reflecting glucose hypometabolism due to CCD, eight years after the stroke episode.

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