CASE OF THE MONTH

A young man with a headache

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A 28-year-old male presented to hospital with sudden onset of headache associated with vomiting. The patient denied trauma and had no medical history of note apart from having regular headaches, which normally resolved with rest and paracetamol. He had no known drug allergies, was not on any regular medication and had no significant family history. Physical examination revealed a dense right hemiplegia and all initial laboratory investigations were normal.

An emergency CT scan of the brain confirmed a left middle cerebral artery (MCA) territory infarct associated with significant cerebral oedema and midline shift towards the right. A subsequent CT cerebral angiogram demonstrated occlusion of the left internal carotid artery. A short time after admission, the patient developed unreactive pupils and decreased GCS (Glasgow coma scale). An emergency left frontoparietal craniotomy was performed and a CT scan of the brain repeated (Figure 1). The scan demonstrated a left frontoparietal craniotomy with a large left MCA infarct with midline shift towards the right. It was noted that the left-sided sulcal effacement and ventricular size was unchanged when compared with the previous CT brain scan.

Owing to the young age of the patient, an 18-fluoro-2-deoxyglucose positron emission tomography computed tomography (¹⁸F-FDG PET-CT) scan was performed 22 days after the initial presentation to investigate a potential underlying cause (Figure 2). The images obtained

raise several questions. What are the radiological abnormalities demonstrated here on the dotted and solid arrows? What is the diagnosis?

Figure 1. CT brain scan demonstrating a left frontoparietal craniotomy with a left-sided large middle cerebral artery infarct with midline shift to the right.

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Figure 2. (a) ^{18}F -FDG PET image of the brain. (b) ^{18}F -FDG PET-CT image of the brain. Images demonstrate hypometabolism in left MCA infarct (solid arrows) and the right cerebellar hemisphere (broken arrows).

Findings

No evidence of ^{18}F -FDG uptake was observed within the aorta or main vessels to suggest a vasculitic process. However, the scan did demonstrate hypometabolism of the right cerebellar hemisphere consistent with crossed cerebellar diaschisis (CCD; dotted arrows in Figure 2). No corresponding cerebellar lesion was seen on CT and the left MCA infarct was clearly demonstrated (solid arrows in Figure 2).

Discussion

CCD is a depression of neuronal metabolism and regional blood flow in the cerebellar hemisphere. This condition is believed to be caused by interruption of the corticopontocerebellar fibres, which connect the cerebral hemispheres to the cerebellum [1]. CCD most commonly occurs following both acute and chronic phases of cerebrovascular infarction and has been demonstrated using ^{18}F -FDG PET as many as 20 years after a cerebrovascular accident [2]. CCD has also been demonstrated with single photon emission computed tomography (SPECT) [3] and magnetic resonance (MR) perfusion techniques [1]. The condition commonly manifests as reduced metabolism and blood flow in the cerebellar hemisphere contralateral to a supratentorial infarction. Other causes of CCD include trauma, gliosis, epilepsy and tumours.

CCD is closely related to the volume of supratentorial hypoperfusion in the first hours after stroke rather than severity. At later time points, CCD is strongly associated with outcome measures [4]. The most crucial determinant of the development of CCD is the location of hypoperfusion; MCA territory infarctions most commonly give rise to CCD [1]. CCD recovery can be seen in patients with small infarcts that are successfully thrombolysed leading to reperfusion of the area at all time points. Furthermore, CCD is closely related with clinical outcome measures, suggesting that it might be an

important prognostic indicator of stroke recovery as well as treatment response [1].

In our case of a young patient presenting with symptoms of a stroke, atypical underlying causes such as large vessel vasculitis (e.g. Takayasu's arteritis) were considered as a differential diagnosis. Takayasu's arteritis is a rare, idiopathic, chronic inflammatory disease with cell-mediated inflammation. This condition mainly involves the aorta and its major branches and can be detected using ^{18}F -FDG PET [5]. MR perfusion techniques are able to detect CCD, although the detection rate might be lower than for ^{18}F -FDG PET [1].

Serial measurements using functional imaging have been proposed to allow the development of individualised rehabilitation programs, where rehabilitation techniques could be synchronised with the recovery of corresponding functional areas of viable brain [6]. In conclusion, owing to the increasing utilisation of ^{18}F -FDG PET-CT and the ability of viable brain tissue to functionally recover with good quality rehabilitation therapy, it is important to recognise and report cases of CCD as they will be more frequently encountered in the future.

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