095 EVOLVING BRAIN LESION: CAAN'T MAKE A DIAGNOSIS

Ruth Wood, Ruth Dobson, Louise Crowe, Panos Koumellis, Sriram Vundavalli, Dennis Chan. *Hurstwood Park Neurosciences Centre, Haywards Heath, UK*

10.1136/jnnp-2014-309236.95

A 42-year-old man with longstanding type 1 diabetes presented with subjective memory difficulties. He was awaiting combined pancreas-kidney transplant for diabetic nephropathy. Assessment demonstrated no objective cognitive impairment and no focal neurological deficit. Initial MRI brain scanning revealed a focus of subtle abnormal high signal within the right fronto-parietal region, which on serial imaging became more prominent with extension into contralateral parietal and bilateral subcortical regions at one year. DWI and MRA were normal. Microhaemorrhages were seen on T2*. Nine months post-presentation he developed focal motor seizures.

Given the concern regarding immunosuppression secondary to diabetes and chronic renal failure, together with the potential for further immunosuppression post-transplant, extensive screening for opportunistic infection and malignancy was undertaken but without diagnostic clarification. Brain biopsy was undertaken, with histological examination revealing striking deposition of amyloid- β consistent with cerebral amyloid angiopathy (CAA-A β).

CAA-A β has a spectrum of clinical phenotypes and the usual mode of presentation is with spontaneous intracerebral haemorrhage. Typical MRI findings include cerebral microbleeds and haemorrhages, siderosis and leucoencephalopathy. Presentation as a focal, evolving, non-haemorrhagic lesion is rare. A review of similar cases will be presented and the implications of a diagnosis of CAA-A β for potential transplant recipients discussed.