J of Nuclear Medicine Technology, first published online April 9, 2015 as doi:10.2967/jnmt.114.153627

Plaque Inflammation Imaging in Severe Carotid Stenosis and Recurrent Cerebral Ischemia

Vijay K Sharma, MD[1,2]

Prakash R Paliwal, MRCP[1]

Arvind K Sinha, MD[3]

[1]Division of Neurology, National University Hospital, Singapore

[2]YLL School of Medicine, National University of Singapore, Singapore

[3]Department of Diagnostic Imaging, National University Hospital, Singapore

Correspondence to:

Dr. Vijay Sharma, Division of Neurology, National University Health System, Level 10, Tower Block, 1E Kent Ridge Road, Singapore 119228.

Email- drvijay@singnet.com.sg

Tel. +65-67722516

Fax- +65-68723566

Financial disclosures- None of the authors declare any conflict of financial interest.

Abstract

Strong relationship exists between the severity of carotid stenosis and early stroke-risk. Inflammation is believed to be an important event for atherosclerotic plaque destabilisation and subsequent thrombo-embolism. 18-Fluoro-deoxyglucose Positron Emission Tomography (18F-FDG) can image atherosclerotic inflammation, providing information about plaque biology, which may serve as a useful biomarker for the assessment of early stroke-risk.

Key words- acute ischemic stroke; carotid stenosis; plaque inflammation; PET-CT

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Introduction

Strong relationship exists between severity of carotid stenosis and early stroke risk which is independent of other prognostic indicators and neuroimaging findings [1, 2]. Inflammation may lead to atherosclerotic plaque destabilisation and thromboembolism [3]. Increased 18-fluoro-deoxy-glucose (18F-FDG) uptake represents carotid plaque inflammation on positron emission tomography (PET) and predicts early recurrent stroke, independent of age and degree of stenosis [4].

Case Report

A 58-years old man presented with transient right hemiparesis and aphasia. His cardiovascular risk factors included hypertension, ischemic heart disease and dyslipidemia. He was diagnosed to have asymptomatic severe (>70%) stenosis of left internal carotid artery (ICA) 2-years ago.

Clinical examination and computerized tomography (CT) scan of the brain were unremarkable. CT-angiography (Fig 1A) confirmed the pre-existing severe focal stenosis of left ICA.

Clopidogrel, aspirin and atorvastatin were started with a plan for early carotid endarterectomy. However, on day-3, he developed right hemiparesis. Brain MRI revealed multiple acute ischemic infarctions in left middle (MCA) and anterior cerebral artery territories (Fig 1B). Transcranial Doppler monitoring of left MCA revealed spontaneous microemboli (Fig 1C). Carotid PET-CT, fused with CT-angiography showed increased 18F-FDG uptake in the left carotid plaque (Figure 2A, 2B), representing acute inflammation. His family members refused an urgent carotid endarterectomy. He developed another severe left MCA ischemic stroke on day-5

with clinical worsening. His further stay in the hospital was uneventful until discharge to rehabilitation unit.

Discussion

Our case demonstrates the role of imaging carotid plaque inflammation with 18F-FDG-PET as a reliable marker for risk stratification. To date, risk stratification for carotid atherosclerosis has been mainly based on arterial lumen stenosis[1] and understanding of plaque biology remains poorly understood [1].

Limited data are available on the prognostic utility of metabolic imaging with PET to predict short-term stroke risk in patients with recently-symptomatic carotid atherosclerosis. Addition of plaque 18F-FDG data to the Framingham Risk Score significantly improved long-term risk prediction [5]. In a study in Ireland, the predictive utility of 18F-FDG plaque uptake was independent of age and degree of lumen stenosis (categorised as 50-69% and 70-99%, adjusted hazard ratio [HR] 6.1, CI 1.3-28.8, p=0.02) [4]. In addition to risk stratification, molecular imaging with 18F-FDG-PET may also have potential to monitor the response of atherosclerotic plaque to therapeutic intervention.

Conclusion

Inflammation in a previously stable plaque carotid plaque is an important event that leads to local thrombosis and cerebral embolization. This phenomenon can be reliable imaged with 18F-FDG-PET imaging for better risk stratification and therapeutic strategy in carotid stenosis.

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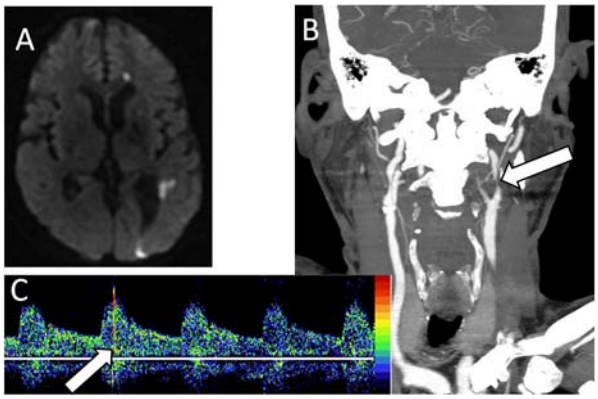

Figure 1. Diffusion-weighted MRI (A) shows ischemic infarcts in left internal carotid artery territory with high-grade stenosis on CT angiography (B). Transcranial Doppler monitoring showed spontaneous microembolic signals (C).

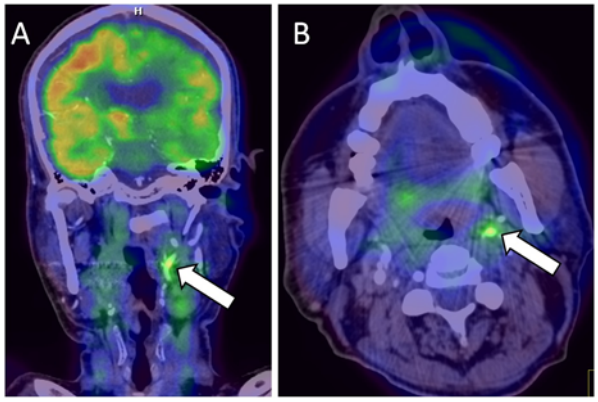

Figure 2. 18F-FDG PET-CT imaging shows increased uptake of 18F-FDG in left ICA plaque on coronal (A) and axial plane (B), suggestive of acute inflammation.