Metabolically Active Subacute Infarct Masquerading as Metastasis: A Finding not to be overlooked in Asymptomatic Oncology Patients

Abstract
A 50-year-old man with carcinoma of the right buccal mucosa underwent staging whole-body 18F-fluorodeoxyglucose positron emission tomography–computed tomography, which revealed a hypermetabolic heterogeneously enhancing lobulated primary lesion in the right buccal region and an incidental finding of subacute stroke. The case highlights the importance of discriminating brain neoplasms mimicking stroke from true ischemic stroke, which is crucial for appropriate management of patients in an oncology setting.

Keywords: Hypermetabolic peri-ischemia, hypometabolic ischemic core, ischemic stroke, stroke masquerading brain tumors

A 50-year-old man with recent diagnosis of moderately differentiated squamous cell carcinoma of the right buccal mucosa underwent staging whole-body 18F-fluorodeoxyglucose (18F-FDG) positron emission tomography–computed tomography (PET-CT). The whole-body and dedicated brain images were acquired following IV administration of 5 mCi 18F-FDG on Siemens Biograph Vision 600 Digital PET CT scanner with contrast-enhanced CT. Images revealed an intensely hypermetabolic (SUVmax: 15.86) heterogeneously enhancing lobulated soft-tissue density lesion in the right buccal region with metabolically active ipsilateral nodal deposits [Figure 1a-c].

An incidental finding of metabolically inactive hypodense area (red arrow) was seen in the right parieto-occipital region [Figure 2a-d and f], with irregular FDG-avid cortical gyral enhancement in the right parietal and right frontoparietal regions (white arrows [Figure 2a-f]).

The patient had no neurological symptoms, and history did not suggest a previous or recent stroke. Subsequently, magnetic resonance imaging (MRI) showed T2 fluid-attenuated inversion recovery hyperintensity [Figure 3: II] involving the right middle cerebral artery–posterior cerebral artery cortical watershed zone and the right frontal parasagittal internal watershed zone, with gyral enhancement and corresponding diffusion hyperintensity [Figure 3: Ia], with normalized apparent diffusion coefficient [Figure 3: Ib] and susceptibility-weighted imaging blooming areas (hemorrhagic transformation). Arterial spin labeling showed hypoperfusion [Figure 3: IV] in the right cerebral watershed zones.

Magnetic resonance (MR) angiography [Figure 3: V] confirmed focal severe narrowing (>90% narrowing) of the right cavernous internal carotid artery (ICA) anterior segment and severe narrowing of bilateral V4 vertebral artery, with eccentric wall thickening and enhancement in three-dimensional T1 (vessel wall imaging, Figure 3: VI a and b) sequence. Mean transit time [Figure 3: III] and time to peak were increased in the right cerebral watershed zones in MR perfusion. MRI findings confirmed right supraclinoid ICA narrowing with right cerebral hemisphere subacute watershed zone infarcts.

Brain metastasis resulting from primary head-and-neck squamous cell carcinomas (HNSCC) is rare, occurring in <1% of all reported cases.[11] However, some studies have also shown brain metastasis of 6%–9% in head-and-neck cancers.[2-3] Central nervous system metastases.

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involvement by HNSCC is commonly seen via direct invasion of the skull base or through perineural invasion.\(^1\) In addition, human papillomavirus status may predispose patients to develop brain metastasis.\(^4\) This case highlights the importance of distinguishing true ischemic stroke that may exhibit metabolic activity in subacute phase from malignant brain tumors, such as gliomas, meningiomas, metastases, and uncommon lesions such as intravascular lymphoma, that can masquerade as acute stroke.\(^5\) The above metabolic pattern of hypometabolic ischemic core and the surrounding hypermetabolic active inflammatory phase of subacute ischemic stroke representing viable tissues in the ischemic penumbra is attributable to the upregulation of parenchymal and microvascular glucose transporters (GLUTs), lateralization of GLUT1 mRNA expression to the ischemic hemisphere,\(^8\) and “trapping” of \(^18\)F-FDG-6-phosphate due to increased hexokinase activity in the peri-ischemic regions in response to hypoxia. The discrimination between brain tumors mimicking stroke and a true ischemic stroke is, therefore, crucial for proper management.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for the images and other clinical information to be reported in the journal. The patients understand that their name and initials will not be published and due efforts will be made to conceal their identity.

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**Figure 1:** 18F-Fluorodeoxyglucose PET–CT showing intensely hypermetabolic (SUV\(_{\text{max}}\): 15.86) heterogeneously enhancing lobulated soft-tissue density lesion in the right buccal region (a and b) and metabolically active ipsilateral cervical nodal deposits (c)

**Figure 2:** (a-f) 18F-fluorodeoxyglucose positron emission tomography–computed tomography showing an incidental finding of metabolically inactive hypodense area noted in the right parieto-occipital region with irregular fluorodeoxyglucose-avid cortical gyral enhancement in the right parietal and right frontoparietal regions
Conflicts of interest

There are no conflicts of interest.

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