PICTORIAL SPECTRUM OF BRAIN ABNORMALITIES DETECTED ON WHOLE BODY F-18 FDG PET/CT SCAN

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Positron emission tomography (PET) with integrated computed tomography (CT) is a unique modality to noninvasively scan the whole body for diagnosing, staging and assessing response to therapy in various benign and malignant diseases. ¹⁸F fluorodeoxyglucose (FDG) is the most commonly used radiotracer for PET/CT imaging in cancer patients. FDG is a glucose analogue which is the predominant substrate for brain metabolism. As the brain cells are obligate glucose consumers, the knowledge of physiologic radiotracer uptake within the brain is imperative for correct interpretation of abnormal sites of metabolism. Over 10,000 PET/CT scans have been performed at our centre in a 5 years' period. A spectrum of brain abnormalities, both benign and malignant, detected in cancer patients undergoing whole body ¹⁸F FDG PET/CT imaging has been compiled. **Keywords:** Fluorodeoxyglucose; Positron Emission Tomography; Computed Tomography; Magnetic Resonance Imaging; Brain Tumours, Brain Metastasis; Benign Cerebral Lesions

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INTRODUCTION

PET/CT rapidly provides anatomic and functional three-dimensional views of the body and can be used for longitudinal assessment of disease over time. An ¹⁸F FDG PET/CT scan allows the in vivo study of cerebral glucose metabolism and reflects neuronal and synaptic activity.^{1 18}F-FDG uptake is usually higher in the frontal, parietal and occipital areas than in temporal cortex. In the subcortical structures, basal ganglia have slightly higher activity than the cortex. Metabolic activity is lower in medial temporal cortex including hippocampal areas, compared to neocortical regions.² Comprehensive clinical history and correlation with CT and MRI scans adds specificity and aids in arriving at the correct imaging diagnosis.

Primary CNS tumours account for about 1–2% of all malignancies.³ FDG uptake is able to assess the degree of malignancy at the time of diagnosis since low-grade tumours are typically less metabolic than high-grade tumours.⁴ FDG metabolism is thought to be proportional to but not quantitatively equal to glucose metabolism in brain tumours.⁵ In our series tumours like pituitary macroadenoma were hypermetabolic. Lymphomatous involvement of brain parenchyma however had a variable pattern of uptake with hypermetabolic lesions in some patients and photopenic defects in others

CASE 1

30 years old male with poorly differentiated carcinoma of the right lung.



Figure-1 (A) ¹⁸F FDG PET/CT scan showing a large space occupying photopenic (no uptake) lesion in the left parietal lobe causing mass effect on the lateral ventricle



Figure-1 (B) T2-Weighted MR shows high signal, mass like abnormality involving left temporal and frontal lobes. There was heterogeneous signal drop on diffusion weighted (DW) images demonstrating some fluid component, although most of the tumour was solid.



Figure-1: (C) Diffusion weighted MR images showed significantly restricted diffusion in the more solid components



Figure-1: (D) T1-Weighted images showed no significant post contrast enhancement and tumour was of low signal intensity throughout



Figure-1: (E) Follow up CT showed intra-axial nodules involving the brain parenchyma, compatible with hyper dense metastases indicating disease progression

A 30-year-old male with a pituitary macroadenoma.



Figure-2: (A) The T1-Weighted pre and post contrast coronal image demonstrates a large sellar/supra-sellar mass with superiorly placed haemorrhagic foci. The solid non-haemorrhagic component of this tumour demonstrates restricted diffusion



Figure-2: (B) ¹⁸F FDG PET/CT scan showing a large primary sellar tumour with para-sellar extension and heterogeneous FDG uptake [Standardized uptake value (SUV) 9.6]

CASE 3

A 20-year-old male with headache, vomiting, shortness of sight and seizures.



Figure-3: (A) T2-Weighted and FLAIR MR images show periventricular low intensity nodules, abnormal high signal involving the corpus callosum and periventricular white matter with oedema in relation to the lateral ventricular system.



Figure-3: (B) Diffusion weighted images showed restricted diffusion in most of the subperiventricular nodules



Figure-3: (C) ¹⁸F FDG PET/CT scan shows irregular nodular enhancement along the lateral ventricle. [SUV 6.6]

A 21-year-old male with hodgkin disease.



Figure-4: (A) [Baseline ¹⁸F FDG PET/CT scan] Photopenia seen in right frontal lobe



Figure-4: (B) MRI shows lobulated extra-axial dural based mass with post contrast enhancement. This mass is low signal intensity on T2 and isointense on T1 sequences



Figure-4: (C) Post chemotherapy ¹⁸F FDG PET/CT scan] No obvious intracranial lesion identified on CT. Mild asymmetry seen in FDG uptake secondary to healing process.

CASE 5

A 30 years' female with left temporal swelling for 2 months, diagnosed non-Hodgkin lymphoma (NHL) on biopsy.



Figure-5: (A) [Baseline ¹⁸F FDG PET/CT] Disease in the left skull vault with extra cranial extension causing erosion of the temporal bone.



Figure-5: (B) T2 W and DWI MR images shows a lobulated intensely enhancing soft tissue mass around left temporal bone having both intra and extra cranial component.



Figure-5: (C) [Post chemotherapy ¹⁸F FDG PET/CT]

No residual soft tissue in left temporal region. Residual photopenia represents response to treatment and ongoing healing process.

BRAIN METASTASES

Accurate identification of cerebral metastases is important for staging, prognostication and determination of appropriate therapy^{6, 7}. Brain metastasis show varying degrees of metabolic activity on PET scan ranging from photpenia to intense uptake in lesions and appearing isoor hyperdense on CT scan. In our compilation, patients with primary ovarian, malignant histiocytosis and neuroendocrine tumours had hypermetabolic brain metastasis while a patient with primary lung carcinoma had ametabolic (cold) metastasis.

CASE 6

59 years' female with ovarian carcinoma stage IIIC. Post chemotherapy and surgery developed right hemiparesis.



Figure-6: MRI shows left frontal lobe lesion which is hyper metabolic on ¹⁸F FDG PET/CT scan.

CASE 7

60 years' female with malignant histiocytosis of right thigh, post amputation 1 year ago, presented with seizures for 1 month.



Figure-7: (A) ¹⁸F FDG PET/CT shows hypermetabolic [SUV17] enhancing nodule in the right frontal lobe with mass effect on lateral ventricle and surrounding oedema. Additionally, metastatic lesions also identified



Figure-7: Additionally, metastatic lesions also identified at (B) Large thyroid mass [52 mm] with mediastinal extension. (C) Necrotic appearing left lower lobe lung mass. (D) Enlarged para aortic lymph nodes. (E) Sclerosis in left proximal femur with intense ¹⁸F

FDG uptake indicating bony metastasis.

CASE 8

A 57 Years male with right lung adenocarcinoma developed sudden onset of right arm weakness.



Figure-8: Axial ¹⁸F FDG PET/CT show a peripherally enhancing right occipital lesion with no FDG uptake indicating metastasis.

CASE 9

A 26 years' male with headache associated with vomiting and gradual loss of vision in left eye was diagnosed with a metastatic small cell neuroendocrine tumour post craniotomy.



Figure-9: (A) [¹⁸F FDG PET/CT scan for evaluation of residual disease] Axial images showed postsurgical changes in left frontoparietal region with no evidence of residual disease.

A 58 years' female with high grade right frontal glioma had suspicion of residual disease/post-operative changes on post craniotomy MRI.



Figure-10: (A) ¹⁸F FDG PET/CT showed heterogeneous slightly enhancing lesion involving the right frontal lobe with central necrosis and intensely active peripheral component [SUV of 17.2] signifying residual disease.

BENIGN CEREBRAL LESIONS

A variety of benign brain lesions may mimic disease or behave as a confounder to PET interpretation. Brain infarcts due to cerebrovascular accidents and stroke present as photopenic areas of absent metabolic activity indicating loss of functioning neuronal tissue.

Tuberculosis (TB) of the central nervous system is still prevalent in many developing countries.⁸ Most patients with malignancies are immunocompromised due to chemo radiotherapy for the treatment of their primary disease. In such cases TB can produce signs and symptoms which can imitate neoplastic disease.⁹ Patients with sputum negative pulmonary TB and extra pulmonary TB are difficult to diagnose and may be missed at all points of care.¹⁰ Active TB avidly takes up ¹⁸F FDG, both in pulmonary and extra pulmonary lesions¹¹.

CASE 11

19 years old male with hodgkin disease.



Figure-11: (A) [Baseline ¹⁸F FDG PET/CT] Axial images show linear FDG uptake along the base of brain predominantly on the left side.



Figure-11: (B) On T2-Weighted MRI images, small intense focus was seen in the right frontal white matter and left cerebral hemisphere, close to the 4th ventricle with post contrast enhancement on T1-Weighted images.



Figure-11: (C) Coronal on T1-Weighted post contrast images show basal meningeal enhancement. Analysis of cerebrospinal fluid showed growth of Mycobacterium tuberculosis confirming TB meningitis.

CASE 12

A 64-year male with colon carcinoma.



Figure-12: [Baseline ¹⁸F FDG PET/CT scan] Axial images showed encephlomalacia and corresponding photopenia on PET/CT images indicating an infarct in the territory of the left middle cerebral artery.

A 71 years old female with ovarian carcinoma stage III.



Figure-13: Axial PET/CT images showed focally dilated occipital horn of left ventricle. Further neurological workup showed no concerning aetiology.

CONCLUSION

Brain lesions display varied patterns of metabolic activity based on a heterogeneous spectrum of actiologies and cerebral glucose metabolism. Understanding PET/CT appearance pattern of various intracranial metabolic abnormalities in conjunction with other radiological imaging enables correct interpretation and improves specificity of the PET study.

REFERENCES

- Berti V, Mosconi L, Pupi A. Brain: Normal Variations and Benign Findings in FDG PET/CT imaging. PET Clin 2014;9(2):129–40.
- Kochunov P, Ramage AE, Lancaster JL, Robin DA, Narayana S, Coyle T, *et al.* Loss of cerebral white matter structural integrity tracks the gray matter metabolic decline in normal aging. Neuroimage 2009;45(1):17–28.
- Stewart BW, Wild C, International Agency for Research on Cancer, World Health Organization, editors. World cancer report 2014. Lyon, France: International Agency for Research on Cancer; 2014.
- Di Chiro G. Positron emission tomography using [18F] fluorodeoxyglucose in brain tumours: A powerful diagnostic and prognostic tool. Invest Radiol 1987;22(5):360–71.
- Spence AM, Muzi M, Graham MM, O'Sullivan F, Krohn KA, Link JM, et al. Glucose metabolism in human malignant gliomas measured quantitatively with PET, 1-[C-11]glucose and FDG: Analysis of the FDG lumped constant. J Nucl Med 1998;39(3):440–8.
- Kim YS, Kondzielka D, Flicklligar JC, Lunsford LD. Stereotactic radiosurgery for patients with non-small cell lung cancer metastatic to the brain. Cancer 1997;80(11):2075–83.
- Young RF. Radiosurgery for the treatment of brain metastases. Semin Surg Oncol 1998;14(1):70–8.
- Rock RB, Olin M, Baker CA, Molitor TW, Peterson PK. Central nervous system tuberculosis: pathogenesis and clinical aspects. Clin Microbiol Rev 2008;21(2):243–61.
- Kamaleshwaran KK, Shinto AS, Natarajan S, Mohanan V. F-18 FDG PET/CT in Tuberculosis: Non-Invasive Marker of Therapeutic Response to Antitubercular Therapy. Int J 2015;2(1):23.
- Skoura E, Zumla A, Bomanji J. Imaging in tuberculosis. Int J Infect Dis 2015;32:87–93.
- Ankrah AO, van der Werf TS, de Vries EF, Dierckx RA, Sathekge MM, Glaudemans AW. PET/CT imaging of Mycobacterium tuberculosis infection. Clin Transl Imaging 2016;4:131–44.

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