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Research Article

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Rare Case Study of Triple Primary Tumors (Lung, Thyroid & Breast) in a patient and Role of PET-CT in its Follow-Up

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Introduction

Positron emission tomography (PET) and computed tomography (CT) complement each other's strengths in integrated PET/CT. PET is a highly sensitive modality to depict the whole-body distribution of positron-emitting biomarkers indicating tumour metabolic activity. However, conventional PET imaging is lacking detailed anatomical information to precisely localize pathologic findings. CT imaging can readily provide the required morphological data. Thus, integrated PET/CT represents an efficient tool for wholebody staging and functional assessment within one examination. Due to developments in system technology PET/CT devices are continually gaining spatial resolution and imaging speed. Wholebody imaging from the head to the upper thighs is accomplished in less than 20 min. Spatial resolution approaches 2-4 mm. Most PET/CT studies in oncology are performed with 18F-labelled fluoro-deoxy-D-glucose (FDG). FDG is a glucose analogue that is taken up and trapped within viable cells. An increased glycolytic activity is a characteristic in many types of cancers resulting in avid accumulation of FDG. These tumours excel as "hot spots" in FDG-PET/CT imaging. FDG- PET/CT proved to be of high diagnostic value in staging and restaging of different malignant diseases, such as colorectal cancer, lung cancer, breast cancer, head and neck cancer, malignant lymphomas, and many more. The standard whole-body coverage simplifies staging and speeds up decision processes to determine appropriate therapeutic strategies.

History

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Known case of adenocarcinoma right lung, carcinoma left

breast and carcinoma thyroid (Histopathologiclly proved). Status post right-upper lobectomy 2016, left MRM 1998, thyroidectomy 1998, chemotherapy in 2016 and IMRT porta hepatis region in Jul 2017. Follow-up scan.

Technique

7.05 mCi (18F) - fluorodeoxyglucose was administered intravenously via left hand. To allow for distribution and uptake of radiotracer, the patient was allowed to rest quietly for 59 minutes in a shielded room. Imaging was performed on an integrated 64-slice PET/CT scanner, with scanning whole body. Blood glucose at the time of the injection was measured at 147 mg/dL. Serum creatinine is 0.88 mg/dL. CT scanning was performed with intravenous contrast material. All reported uptake values are maximum SUVs unless stated otherwise.

Materials and Methods

A patient reported to armed forces institute of Radiology with Histopathologiclly proved triple primary tumor of lung, breast and thyroid for PET-CT follow up scan. PET-CT was performed, and Comparison was done between the previous PET-CT dated 21-10-2021, 22-01-2021, 30-01-2020, 05-01-2018 & MRI dated 06-02-2019 to see the current status of primaries and their metastasis.

Reference hepatic SUV = 3.4

Sections through the brain show physiological parenchymal uptake. Stable appearing metabolically insignificant calcified dural lesion along left parietal bone 1.6 cm size. Activity within the glottic region, tonsils and salivary glands is physiological. Interval metabolic and morphologic regression in avid discrete subcentimetre size left level II & III cervical nodes, likely metastatic. No avid or size significant right cervical or supraclavicular nodes. For reference, left level II cervical node 0.5 cm 4 SUV (previously 0.6 cm 8.1 SUV), left level III cervical 0.4 cm 3 SUV (previously 0.5 cm 6.9 SUV) nodes. Interval metabolic regression in stable appearing avid soft tissue in post-operative left thyroid lobe bed 1.3 cm 3.2 SUV (previously same size 5.8 SUV), remains concerning for recurrence. Tissue sampling.

Interval metabolic regression in size stable avid discrete subcentimetre size bilateral hilar reactive nodes. No avid or size significant mediastinal or axillary nodes noted. For reference, left hilar-10L node 0.5 cm 3.1 SUV (previously same size 5.4 SUV). Stable appearing non-avid pulmonary fibro-atelectatic changes in left upper and right lower lobes. No concerning lung nodules noted. No pleural or pericardial effusion. Stable left mastectomy and right pulmonary upper lobectomy related changes, with no evidence of local recurrence. Stable hepatic segment VI focal calcification 0.5 cm size, likely old inflammatory. Cholelithiasis renoted. Size stable Bosniak type I non-avid bilateral renal cysts. For reference, right upper pole renal cyst 3.1 cm size. Spleen and adrenals are unremarkable. Interval metabolic and morphologic progression in precaval lymphadenopathy 4 cm size 28 SUV collectively (previously 1.3 cm 22.2 SUV collectively), having interface loss with pancreatic uncinate process and asymmetrically thick-walled duodenal second and third parts, along with interval development of avid enlarged hepatic node, likely malignant, represent progressive disease. For reference, precaval lymphadenopathy 4 cm size 28 SUV collectively (previously 1.3 cm 22.2 SUV collectively), hepatic node 1.6 cm 15 SUV. Interval development of avid filling defect in superior mesenteric vein 17 SUV, suggestive of tumour thrombosis. No avid or size significant pelvic sidewall or inguinal nodal disease. Pelvic viscera outline normally. No ascites. Stable appearing T10, L1 and L3 vertebral compression fractures, stable appearing avid inflammatory osteoarthritic changes along bilateral knee joints and non-avid spinal degenerative changes. No abnormal marrow uptake.

Conclusion and Discussion

Progressive disease

1. Interval metabolic and morphologic progression in precaval lymphadenopathy, having interface loss with pancreatic uncinate process and asymmetrically thickwalled duodenal second and third parts, along with interval development of prominent hepatic node, likely malignant, represent progressive disease.

2. Interval development of filling defect in superior mesenteric vein, suggestive of tumour thrombosis.

3. Interval metabolic regression in stable appearing soft tissue in post-operative left thyroid lobe bed, remains concerning for recurrence. Tissue sampling.

4. Interval metabolic and morphologic regression in left level II & III cervical nodes, likely metastatic.

5. Interval metabolic regression in size stable hilar reactive nodes.

6. Stable appearing pulmonary fibro-atelectatic changes in left upper and right lower lobes.

7. Stable appearing calcified dural lesion along left parietal bone.

8. Stable left mastectomy and right pulmonary upper lobectomy related changes, with no evidence of local recurrence.

9. Stable appearing T10, L1 and L3 vertebral compression fractures, stable appearing inflammatory osteoarthritic changes along knee joints and spinal degenerative changes.

10. Size stable Bosniak type I renal cysts.

11. Stable hepatic segment VI focal calcification, likely old inflammatory.

12. Cholelithiasis.

Role of PET/CT in follow up of malignancies

In patients with suspected malignancies both prognosis and therapeutic management particularly depend on the tumour stage. Thus, accurate tumour staging preferably encompassing the entire body is of high importance. PET is a very sensitive modality to depict the spatial whole-body distribution of positron-emitting biomarkers that indicate molecular processes underlying tumour metabolic activity [1]. The average F-18-FDG PET sensitivity and specificity across all indications in oncology are estimated at 84% (based on 18,402 patient studies) and 88% (based on 14,264 patient studies), respectively, according to Gambhir et al. [2] from a collection of 419 articles from 1993 to 2000.

However, sole PET images are lacking detailed anatomical information. Reliable localization of a lesion within a segment of an organ or even within a certain organ itself can be challenging. Thus, conventional stand-alone PET has mostly been replaced by PET/CT. PET/CT combines the complementary information of functional PET and morphological CT images in one imaging session for improved patient comfort, patient throughput, and most importantly the gain in diagnostic accuracy. FDG-PET/CT has been found to be superior to both imaging procedures acquired separately in tumour staging and restaging of different malignant diseases [3-5]. Furthermore, PET/CT potentially supports volume delineation in radiation therapy planning [6]. This may be particularly useful in the head and neck region where a multitude of sensitive structures is confined to a small area of the body. The close vicinity necessitates optimized definition of the treatment volume to minimize the risk of treatment-related toxicities. Another indication for PET/CT in radiation therapy planning is lung tumours where separation of viable tumour from atelectasis can be challenging with morphology alone [6]. While PET imaging has been available since 1980, PET/ CT has first introduced into clinical routine in 2001[7]. Thus, there are many data on PET in oncological applications available in the literature, while data on PET/CT are still limited for some tumour entities. However, depending on the indication and the radionuclide

in question, data on PET imaging may in all likelihood also apply to PET/CT [8,9].

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Conflict of Interest

No conflict of interest.

References

- Al Nakouzi N, Cotteret S, Commo F, Gaudin C, Rajpar S, et al. (2014) Targeting CDC25C, PLK1 and CHEK1 to overcome Docetaxel resistance induced by loss of LZTS1 in prostate cancer. Oncotarget 5: 667–678.
- Califano J, van der Riet P, Westra W, Nawroz H, Clayman G, et al. (1996) Genetic progression model for head and neck cancer: implications for field cancerization. Cancer Res 56: 2488–2492.
- 3. Cheung M, Kadariya Y, Talarchek J, Pei J, Ohar JA, et al. (2015) Germline BAP1 mutation in a family with high incidence of multiple primary cancers and a potential gene-environment interaction. Cancer Lett 369: 261–265.

- Consul N, Amini B, Ibarra-Rovira JJ, Blair KJ, Moseley TW, et al. (2020) Li-Fraumeni syndrome and whole-body MRI screening: screening guidelines, imaging features, and impact on patient management. Am J Roentgenol 216: 252–263.
- 6- de Wilde RF, Heaphy CM, Maitra A, Meeker AK, Edil BH, et al. (2012) Loss of ATRX or DAXX expression and concomitant acquisition of the alternative lengthening of telomeres phenotype are late events in a small subset of MEN 1 syndrome pancreatic neuroendocrine tumors. Mod Pathol 25: 1033–1039.
- Dranka-Bojarowska D, Lewinski A (2019) Multiple primary cancers in patients treated for squamous cell carcinoma of the esophagus. Pol Przegl Chir 91: 51–57.
- Gali-Muhtasib H, Kuester D, Mawrin C, Bajbouj K, Diestel A, et al. (2008) Thymoquinone triggers inactivation of the stress response pathway sensor CHEK1 and contributes to apoptosis in colorectal cancer cells. Cancer Res 68: 5609– 5618.
- 8. Hu X, Moon JW, Li S, Xu W, Wang X, et al. (2016) Amplification and overexpression of CTTN and CCND1 at chromosome 11q13 in Esophagus squamous cell carcinoma (ESCC) of Northeastern Chinese Northeastern Int J Med Sci 13: 868–874.
- Kumagai Y, Kawano T, Nakajima Y, Nagai K, Inoue H, et al. (2001) Multiple primary cancers associated with esophageal carcinoma. Surg Today 31: 872–876.