18-F Fluorodeoxyglucose Positron Emission Tomography/Computed Tomography of a Large Inflammatory-Hepatocellular Adenoma

Abstract

We report a case of an 81-year-old male evaluated for a liver space-occupying lesion. US-guided biopsy and immunohistochemistry were suggestive of hepatocellular adenoma (HCA)-inflammatory (with telangiectasia). Serial 18-F fluorodeoxyglucose (18F-FDG) positron emission tomography/computed tomography scans revealed a heterogeneously enhancing hypermetabolic mass in the right lobe of the liver, remaining stable for a span of 3 years. This case highlights that benign rare tumors of the liver such as HCA can be intensely FDG avid and that uptake cannot conclude its malignant transformation.

Keywords: *18-F fluorodeoxyglucose positron emission tomography/computed tomography, hepatocellular adenoma, inflammatory*

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An 81-year-old male was evaluated for a liver space-occupying lesion. Baseline contrast-enhanced magnetic resonance imaging (MRI) revealed a large solitary well-encapsulated lobulated lesion, heterogeneously hypointense on T1, and heterogeneously hyperintense on T2 weighted imaging in segment VII of the liver. On the hepatobiliary phase, the lesion appeared predominantly hypointense with a subtle area of contrast retention within the lesion and subtle restriction on diffusion-weighted imaging-? Benign/?inflammatory in nature. Biopsy was advised for confirmation. Baseline serum alpha fetoprotein (AFP) level was 2.65 ng/ml (0–8.5). The US-guided biopsy of the liver mass showed 1–3 thick cord of cells, few binucleated hepatocytes, interspersed several areas of sinusoidal dilatation, peliosis, and telangiectasia, with evidence of few unpaired arteries and no portal tracts, no evidence of fibrosis was seen. Immunohistochemistry revealed Serum amyloid A (SAA) positive but focal, Glypican-3 negative, CD34 diffuse staining in the sinusoidal lining, glutamine synthase – diffuse strong positive, HSP-70 staining suboptimal to comment, β-catenin – only occasional cells show nuclear aberration expression. Pathological features were suggestive of hepatocellular adenoma (HCA) – Inflammatory (with telangiectasia). CE computed tomography (CECT) scan showed large capsulated arterial enhancing mass in the right lobe of the liver. The patient underwent one cycle of Transcatheter arterial chemoembolization (TACE) as it was a bulky HCA with a risk of hemorrhage. Post TACE, CECT scan demonstrated 50% necrosis in the arterially enhancing solid component - suggestive of partial response.. Figure 1 shows serial 18-F fluorodeoxyglucose positron emission tomography/CT (18F-FDG PET/CT) maximum intensity projection (MIP) and corresponding transaxial images acquired in 2018, 2019, and 2020. Figure 1a-c are MIP images showing increased uptake in the right abdominal region and bilateral inflammatory mediastinal lymph nodes. Figure d, e (noncontrast) and f are the transaxial fused images showing a large FDG avid mass in the right lobe of the liver involving segments VII and VI splaying the RPV branches and abutting the RHV (in recent scan measuring 14.2 cm × 12.0 cm × 14.0 cm, AP × TR × CC, SUVmax 6.4; previously in 2019 measuring 13.8 cm × 11.7 cm × 13.1 cm SUVmax 6.7). Figure g-i are the triple-phase CT images acquired with recent PET/CT showing heterogenous arterial enhancement with a persistent peripheral rim of enhancement in porto-venous phase. There was no evidence of any other metabolically active lesion in

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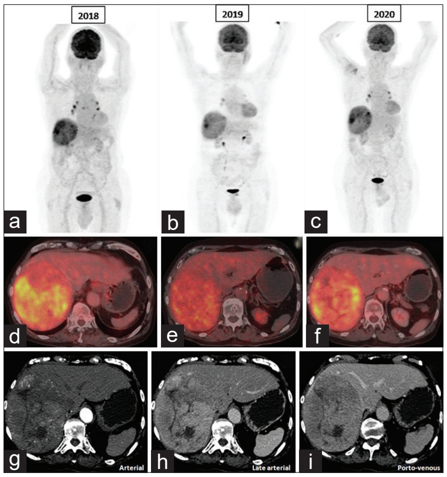

Figure 1: Serial fluorodeoxyglucose positron emission tomography/computed tomography images of 2018, 2019, and 2020. Maximum intensity projection images (a-c) show uptake in the right abdomen and bilateral inflammatory mediastinal lymph nodes. Transaxial fused images (d, e [noncontrast], and f) show large fluorodeoxyglucose avid mass in the right lobe of the liver. Triple-phase computed tomography images (g-i) shows heterogeneous arterial enhancement with a persistent peripheral rim of enhancement. There was no change in size or metabolic activity in serial fluorodeoxyglucose positron emission tomography/computed tomography images, suggestive of stable disease

the body. There is no significant interval change in size or metabolic activity of the liver mass noted in serial PET/CT scans – suggestive of stable disease. Serial serum AFP levels were 2.75 ng/ml (2018); 6.49 ng/ml (2019), and 24.13 ng/ml (2020) (0–8.5 ng/ml). In view of rising the trend of serum AFP level, repeat biopsy was advised to rule out the malignant transformation of HCA.

HCA is the second-most common benign liver neoplasm after focal nodular hyperplasia being more common in reproductive age females (1: 8–10).[1] HCA is characterized by varied genetic and molecular abnormalities, pathology, tumor biology, and radiological features, with sizes varying from <1 cm up to 30 cm. Bioulac-Sage *et al*.[2] classified HCA into four types most common group is the (i) inflammatory/telangiectatic HCA (35%–50%), which present a high risk of hemorrhage and a slight risk of the malignant transformation (ii) second common is the HNF1-α activated type (35%-40%), has the least risk of the malignant transformation (iii) third, activated b-catenin mutation (10%–15%) (iv) unclassified type (5%–10%). Triple-phase CT and MRI (with the advent of newer liver-specific MRI contrast agents such as gadoxetate disodium (Gd-EOB-DTPA) are the investigations that are the choice.[3] HNF1-a subtype of HCA is reported to have false-positive PET findings, with a mean SUVmax of 7.8 (range: 3.9–12.5). Increased FDG uptake may be attributed to enhanced glucose metabolism due to higher tumor cell metabolism or due to inflammatory cells or increased cell density.[4] Other benign causes of focal increased metabolic activity in the liver include abscesses, Cryptococcosis, and hemangioendothelioma.[5] Very few cases of FDG-avid inflammatory HCA are reported in the literature.[6] 11C-acetate and 18F-Fluorocholine are the other optional tracers. Complications with HCA occur when the tumors outgrow their blood supply and include rupture, hemorrhage, thrombosis, infarction, minimal risk of malignant transformation, and rarely even cystic degeneration. Risk factors for malignant transformation of HCA include-large size (>5 cm), multiplicity, β-catenin-mutated subtype of HA, and the male gender.[7] Therefore, identification and accurate treatment decisions of HCA are important to avoid an unnecessary biopsy, surgery, or chemotherapy.[8] Highlights of this case include – High metabolic activity can be seen in inflammatory HCA and a conclusion on malignant transformation cannot be made based on the uptake. Larger studies are needed to confirm the role of PET-CT in HCA evaluation and prognosis.

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 his images and other clinical information to be reported in the journal. The patient understand that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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