Review Article

Clinical Pearls: Etiologies of Superscan Appearance on Fluorine-18-Fludeoxyglucose Positron Emission Tomography-Computed Tomography

Abstract

The term "superscan" usually refers to a characteristic pattern on skeletal scintigraphy consisting of symmetrically intense and diffuse radiotracer uptake in the skeleton with absent or diminished visualization of the genitourinary system and soft tissues. Superscans and superscan-like appearances have also been described on fluorine-18-fludeoxyglucose positron emission tomography-computed tomography (18-F-FDG PET/CT). We review reported cases of 18-F-FDG PET/CT superscans and propose criteria for differentiating pathologic superscans from physiologic causes. Knowledge of the 18-F-FDG PET/CT superscan, its reported pathologic causes, its benign imitators, and its clinical implications is important to the nuclear medicine physician or radiologist specializing in the interpretation of nuclear studies to avoid several diagnostic pitfalls.

Keywords: *Diffuse uptake, fluorine-18-fludeoxyglucose positron emission tomography-computed tomography, positron emission tomography, superscan*

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Introduction

When interpreting skeletal scintigraphy, the term "superscan" refers to a characteristic pattern consisting of symmetrically intense and diffuse radiotracer uptake in the skeleton with absent or diminished visualization of the genitourinary system and soft tissues;[1] the term has also been employed to describe a characteristic appearance on fluorine-18-fludeoxyglucose positron emission tomography-computed tomography (18-F-FDG PET/CT) imaging.[2-18]

Classically, the "super bone scan" or "superscan" was described as the appearance of the skeleton standing out "in bold relief" with diminished or absent radioactivity in the kidneys on technetium-99m methyl diphosphonate imaging[1] [Figure 1]. This appearance can lead to false-negative scans for metastasis because the diffuse and symmetric skeletal uptake may obscure individual metastatic foci.[19] Sy *et al*. hypothesized that increased uptake of radiopharmaceutical by involved bone led to reduced phosphate excretion and diminution of renal activity.[20] The differential diagnosis pertaining to a superscan pattern on skeletal scintigraphy includes diffuse osteoblastic metastasis, renal osteodystrophy, osteomalacia, and severe primary hyperparathyroidism. A superscan appearance may be caused by a prolonged delay before imaging.[21] Skeletal superscans have also been reported in hyperthyroidism[22] and osteopetrosis.[23] It may be difficult to distinguish between superscans caused by metabolic abnormalities and those attributable to diffuse osteoblastic disease; however, the uptake in metabolic disease is more uniform in appearance and extends into the distal appendicular skeleton. Intense uptake in the calvarium greater than that in the remainder of the skeleton is also an indicator of probable metabolic bone disease.[24]

By analogy, superscan patterns have also been described in 18-F-FDG PET/CT scanning.[2-18] This usage of the term "superscan" implies sequestration of radiopharmaceutical in an organ or organ system with loss or reduction in the amount of activity detected in those organs normally associated with significant physiologic uptake of

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18-F-FDG such as the kidneys, brain, or myocardium. As in skeletal scintigraphy, "superscan" has been used to refer to those 18-F-FDG PET/CT scans in which diffuse and intense activity throughout the skeleton is accompanied by reduced visualization of the usual physiologic activity, especially that of the kidneys [Figure 2]. The term has also been employed, however, to describe 18-F-FDG PET/CT scans in which liver uptake is extremely high with reduction of the normal physiologic background activity (the "hepatic superscan") [Figure 3].[2,4,5,9,11-13]

Most reported superscans on 18-F-FDG PET imaging involve malignancy.[2-8,10-18] The hypercellularity and hypermetabolism required to prevent the visualization of physiologic background activities usually implies a heavy tumor burden. As such, it is important that a distinction be made between 18-F-FDG PET/CT superscans (for which few benign etiologies have been reported) and 18-F-FDG PET/CT scans which merely display very prominent avidity of a single organ or organ system without a superscan appearance (for which there are many benign causes). For that reason, before proceeding with a review of reported superscans, we will briefly survey the benign conditions that can cause diffuse FDG-avidity in the skeleton, liver, or soft tissues.

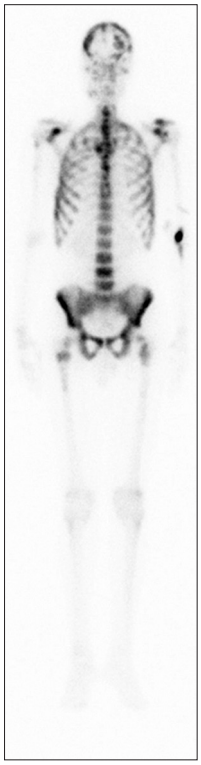

Figure 1: Technetium-99m methyl diphosphonate bone superscan. A technetium-99m methyl diphosphonate bone superscan demonstrating intense, diffuse uptake in the axial and proximal appendicular skeleton as well as more focal uptake in the calvarium in this patient with metastatic prostate adenocarcinoma. Since the radiotracer dose has been entirely sequestered by the extensive bony lesions, the normal renal excretion is not visualized

Review

Benign fluorine-18-fludeoxyglucose positron emission tomography-computed tomography superscans

Conditions causing diffuse avidity of the skeleton on fluorine-18-fludeoxyglucose positron emission tomography-computed tomography

18-F-FDG skeletal avidity is usually mild and relatively uniform with uptake predominantly in the bone marrow. The most common causes of generalized intense bone marrow avidity are rebound of the bone marrow following recent chemotherapy or drug-induced bone marrow activation by erythropoiesis- and colony-stimulating factors (CSFs) [Figure 4]. The appearance of activated marrow on 18-F-FDG PET/CT can be very similar to that of diffuse metastatic disease of the bone, with many reported cases of diffuse marrow activation appearing as metastatic disease and vice versa.[25-28] Pyrexia, from infection or other causes, has also been found to occasionally cause a diffuse and pronounced avidity in the bone marrow. This is believed to be due to interleukin-mediated upregulation of glucose transporters.[29] Hollinger *et al*. found that CSF-mediated diffuse marrow activation was indistinguishable from metastasis on 18-F-FDG PET/CT imaging for up to 4 days after the last administration of growth factors;[26] however, we were unable to find any report of diffuse marrow activation sequestering enough radiotracer to cause a superscan appearance on PET/CT. Primary[30] and secondary[14] hyperparathyroidism have also been reported as mimicking diffuse skeletal metastasis on ^{18}F-FDG PET/CT, and due to the accompanying renal failure, the diffuse skeletal activity with diminished renal excretion caused by secondary hyperparathyroidism is particularly reminiscent of the classically described skeletal superscan, and correlation with renal function laboratories may aid in avoiding diagnostic pitfalls.

Conditions causing diffuse avidity of the liver on fluorine-18-fludeoxyglucose positron emission tomography-computed tomography

Diffusely increased homogeneous activity of the liver is seldom seen on 18-F-FDG PET/CT. The physiologic background activity of the liver is usually greater than the activity of the blood pool. Due to this prominent activity at baseline, a lesion-to-liver ratio is sometimes used to assess the relative hypermetabolism of suspicious lesions seen on 18-F-FDG PET/CT imaging. Hepatic activity is typically heterogeneous which can make detection of primary liver lesions somewhat difficult on 18-F-FDG PET/CT.[20] Infectious etiologies for the nonsuperscan (that is, without loss of physiologic uptake) diffuse liver uptake reported in the literature include hepatosplenic tuberculosis[31] and Q-fever hepatitis.[32] Insulin administration to diabetic patients or release of endogenous insulin in the postprandial state can increase hepatic activity in a relatively diffuse, homogeneous

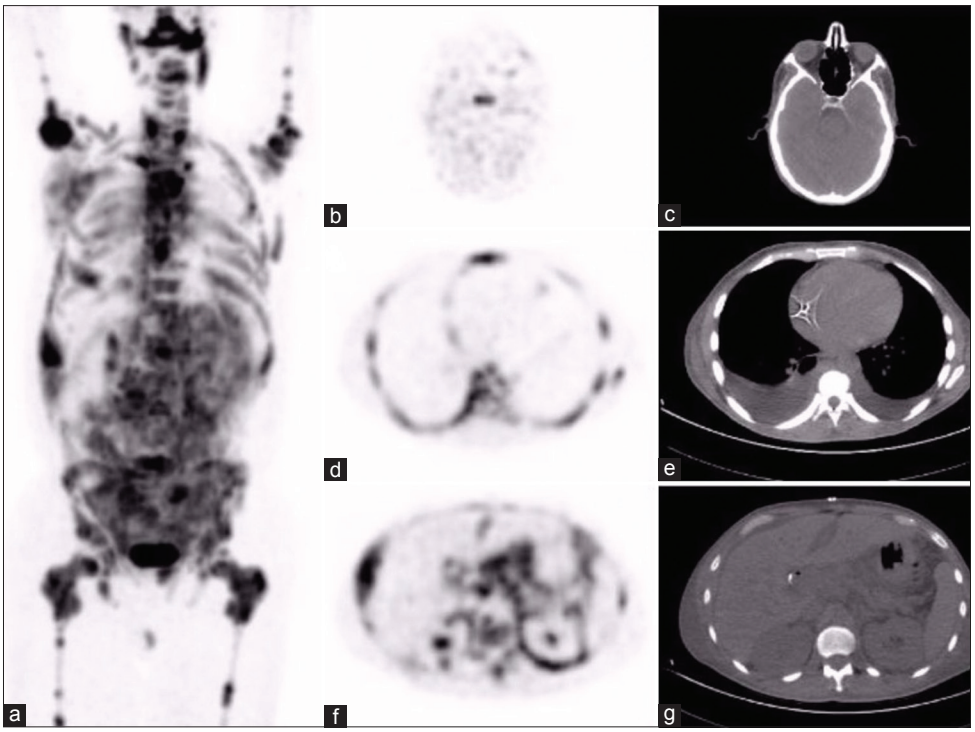

Figure 2: Fluorine-18-fludeoxyglucose positron emission tomography-computed tomography superscan composite. (a-g) From left to right and top to bottom: (a) Fluorine-18-fludeoxyglucose positron emission tomography whole-body anterior projection maximum intensity projection in a patient with high-grade Burkitt lymphoma demonstrates a pathologic superscan with unusual lack of avidity of the brain, heart, renal parenchyma, and liver. There is lymphomatous involvement of the axial and appendicular skeleton, bilateral kidneys, retroperitoneum, abdominal mesentery, thyroid, axillae, mediastinum, and cervical regions. (b and c) Axial fluorine-18-fludeoxyglucose positron emission tomography (b) and noncontrast low-dose computed tomography (b) through the skull demonstrates a hypermetabolic lesion in the region of the dorsum sellae with near blood-pool fludeoxyglucose avidity throughout the brain parenchyma. (d and e) Axial fluorine-18-fludeoxyglucose positron emission tomography (d) and noncontrast low-dose computed tomography (e) at the level of the heart demonstrates intense hypermetabolic activity throughout the visualized bony structures with background-level avidity throughout the myocardium. There are bilateral photopenic pleural effusions. (f and g) Axial fluorine-18-fludeoxyglucose positron emission tomography (f) and noncontrast low-dose computed tomography (g) through the upper abdomen demonstrate near blood-pool level fludeoxyglucose avidity of the renal cortices, liver, and spleen. There are intense hypermetabolic foci throughout the mesentery and retroperitoneum, body wall, bony structures, prominent lymph nodes, and in a solid renal lesion

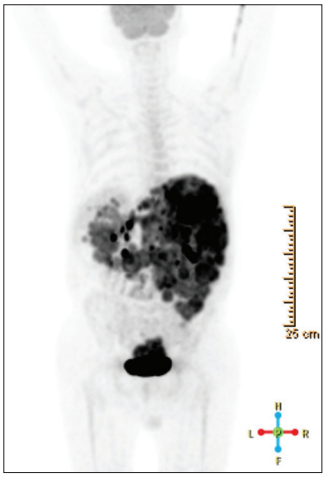

Figure 3: Fluorine-18-fludeoxyglucose positron emission tomography hepatic superscan. Maximum intensity projection of an fluorine-18-fludeoxyglucose positron emission tomography in a middle-aged man with a history of sigmoid adenocarcinoma demonstrating hepatic superscan. There are diffuse hypermetabolic lesions throughout the entire enlarged liver. Hypermetabolic mesenteric and retroperitoneal lymph nodes are also seen along with a hypermetabolic lesion in the region of the sigmoid colon. The avidity of the liver lesions is notably more intense than that seen in the brain

fashion.[21] There are also reports of malignant causes of diffuse hepatic activity without a superscan including primary hepatic lymphoma[33] and diffuse hepatic angiosarcoma.[34]

Conditions causing diffuse avidity of other tissues on fluorine-18-fludeoxyglucose positron emission tomography-computed tomography

Soft tissue activation, especially in the muscles, is another frequently encountered cause of radiotracer sequestration with the potential for reduction in both physiologic background and tumor uptake. 18-F-FDG uptake in the muscles is typically seen as symmetric, mild-to-moderate linear activity;[35] however, focal and asymmetric activation of muscle can be caused by certain exercises or, rarely, by speech and/or swallowing in patients with unilateral laryngeal nerve palsy.[21] Significant uptake can also be observed in the muscles of respiration with hyperventilation and in the cervical muscles with tension or anxiety.[36] Benzodiazepines may be helpful in cases in which muscular radiotracer uptake secondary to anxiety-associated hyperventilation or cervical muscle tension confounds 18-F-FDG PET/CT interpretation.[37]

Diffuse and symmetric activity throughout the muscles on 18-F-FDG PET/CT can result from exercise or from

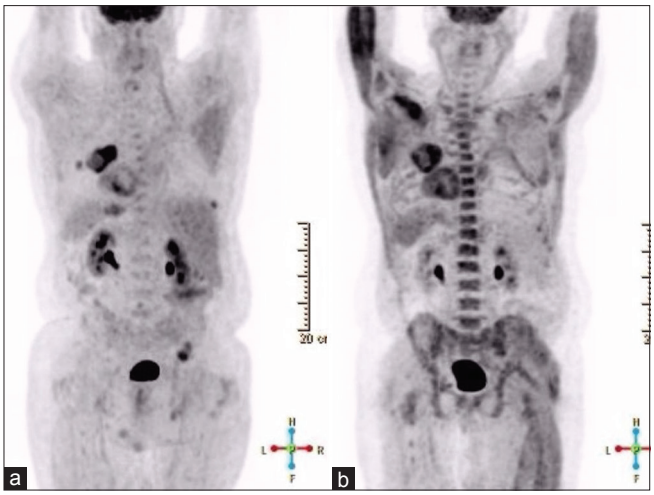

Figure 4: Fluorine-18-fludeoxyglucose positron emission tomography demonstrating diffuse marrow uptake postchemotherapy. (a) Maximum intensity projection of an fluorine-18-fludeoxyglucose positron emission tomography in an elderly male with lung cancer shows a large lesion within the left lung as well as diffuse muscle uptake. Diffuse bone marrow uptake in the setting of marrow reconstitution following chemotherapy limits detection of bony lesions. (b) Five months later, repeat fluorine-18-fludeoxyglucose positron emission tomography redemonstrated the patient's lung mass, now with a new hypermetabolic lung nodule. The previously seen diffuse bone marrow uptake has normalized after recovery from chemotherapy

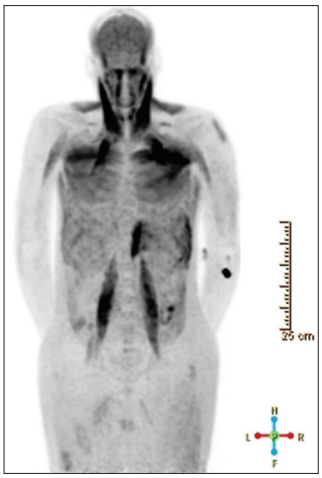

Figure 5: Fluorine-18-fludeoxyglucose positron emission tomography demonstrating "muscle scan" in a hyperglycemic patient. Maximum intensity projection of an fluorine-18-fludeoxyglucose positron emission tomography in a 48-year-old male. As the patient's blood glucose was measured at 127 mg/dL before scanning, recent exercise or noncompliance with fasting state may have led to the diffuse muscle sequestration of the radiotracer, which limited detection of hypermetabolic lesions. The patient later returned for a repeat study that was diagnostic in quality

the action of insulin on skeletal muscle; for this reason, all patients should be advised to fast for 4 h and to avoid exercise for 24 h before 18-F-FDG PET/CT.[38] While insulin administration, by the upregulation of GLUT4 transporters in myocytes, can increase muscle avidity that may reduce the sensitivity for detecting focal hypermetabolic lesions, it is necessary in diabetic patients because hyperglycemia may also cause diffuse avidity of the musculature[39] [Figure 5]. Most institutions attempt to lower serum glucose levels below 200–250 mg/dL before performing 18-F-FDG PET/CT. Turcotte *et al*. reported that an hour interval between the intravenous administration of insulin and 18-F-FDG was sufficient to prevent the so-called "muscle scan" appearance in diabetic patients requiring insulin for hyperglycemia.[40]

Pathologic fluorine-18-fludeoxyglucose positron emission tomography-computed tomography superscans

A review of the literature found 17 reports in which superscans or superscan-like images were obtained on 18-F-FDG PET/CT[2-18] [Table 1]. Nine papers reported skeletal superscans,[3,7,8,10,14,15-18] seven reported hepatic superscans,[2,4,5,9,11-13] and one made reference to a superscan pattern in both the liver and skeleton.[6] Some authors used the term "superscan" to refer to images with intense and homogeneous skeletal or hepatic activity without a notable decrease in the normal activity usually seen in kidneys, heart, or brain. Nine cases included decreased or absent activity in the kidneys[3-10,12,14,] though in one of these cases, the superscan appearance was accompanied by renal failure with secondary hyperparathyroidism,[14] a confounding factor when evaluating decreased renal activity on PET.[20] Nine cases demonstrated faint or greatly reduced physiologic activity in the brain.[2-4,6,8-10,12,18] One report deemed homogeneous intense activity of the liver greater than activity than in the brain, a "hepatic superscan."[11] The usage of the term "superscan" has clearly evolved from a more nuanced usage denoting the absence of physiological tracer activity (as seen in the original "faint kidney sign") to a more general usage which is used to denote strikingly high and diffuse activity in an organ which may or may not be accompanied by the absence of or reduction in the activity seen in the kidneys, brain, heart, or other areas of physiological uptake.

Seven authors reported hepatic superscans.[2,4,5,9,11-13] The degree of reduction in physiological activity in these scans ranged from near absence of uptake in the brain, heart, and kidney in two cases of diffuse large B-cell lymphoma[4,12] to a case of primary hepatic lymphoma[11] in which all physiological activities were present at near normal levels, but liver uptake was more intense than that of the brain. This variety demonstrates the shift in usage of "superscan" from strictly meaning those scans with diminution or loss of physiological activity to a broader meaning meant to convey a very heavy disease burden seen in a particular organ system. The reported liver superscans included cases of breast cancer, Hodgkin's disease, primary hepatic lymphoma, chronic myeloid leukemia, hepatic tuberculosis, and two cases of diffuse large B-cell lymphoma. Interestingly, the two cases of diffuse large B-cell lymphoma[4,12] had the most pronounced reduction in physiological activity with near absence of cardiac, renal, and brain uptake. The case of primary hepatic lymphoma[11] had the most well-preserved physiological activity, with

Table 1: Description of reported superscan and superscan-like appearances on fluorine-18-fludeoxyglucose positron emission tomography-computed tomography

References	Superscanned organ	Pathology	Comments
[2]	Liver	Hodgkin's disease	Very low brain activity
[3]	Skeleton	Renal adenocarcinoma	Brain, liver, and renal cortex only faintly seen
[4]	Liver	Diffuse large B-cell lymphoma	Minimal brain, cardiac, or renal activity seen
[5]	Liver	Breast cancer	Minimal cardiac and renal activity seen
[6]	Skeleton, liver	Small cell lung cancer	Minimal brain and renal uptake
[7]	Skeleton	Squamous cell lung cancer	Minimal kidney activity. 3-h delayed PET
[8]	Skeleton	Prostate cancer	Minimal renal, cardiac, and liver activity
[9]	Liver	Tuberculosis	Absent cardiac and renal activity. Greatly reduced brain activity
[10]	Skeleton	Acute lymphoblastic leukemia	Very low brain activity
[11]	Liver	Primary hepatic lymphoma	Liver activity more intense than brain
[12]	Liver	Diffuse large B-cell lymphoma	Absent cardiac and renal activity, very low brain activity
[13]	Liver	Chronic myeloid leukemia	Absent renal activity
[14]	Skeleton	Secondary hyperparathyroidism	Brown tumors seen in bone
[15]	Skeleton	Pancreatic neuroendocrine tumor	Reduced kidney activity
[16]	Skeleton	Small cell lung cancer	Physiological activity intact
[17]	Skeleton	Bronchogenic adenocarcinoma	Reduced kidney activity
[18]	Skeleton	Primitive neuroectodermal tumor of kidney	Reduced brain and kidney activity

PET: Positron emission tomography

the authors noting that they deemed it a hepatic superscan because liver activity was more intense than that seen in the brain. It is also interesting that the case of hepatic tuberculosis[9] demonstrated reductions in brain and renal activity, which approached those seen in the cases of diffuse large B-cell lymphoma, which one might not expect from a nonmalignant etiology.

Ten authors reported skeletal superscans,[3,6-10,14-18] including one case which the author deemed a skeletal and hepatic superscan.[6] As with the hepatic superscans, there was a wide range in the extent to which physiological activity was reduced. In general, the skeletal superscans demonstrated greater reductions in physiological activity, especially in the brain, than seen in the hepatic superscans. The etiologies reported for the skeletal superscans included renal adenocarcinoma, squamous cell lung cancer, prostate cancer, acute lymphoblastic leukemia, bronchogenic adenocarcinoma, primitive neuroectodermal tumor of the kidney, pancreatic neuroendocrine tumor, two cases of small cell lung cancer, and secondary hyperparathyroidism. It is worth noting that the absence of renal activity seen on ^{18}F-FDG-PET in the case of secondary hyperparathyroidism[13] is confounded by the loss of kidney function implicit in that condition.

In addition to those obtained utilizing ^{18}F-FDG-based PET, superscan and superscan-like appearances have also been reported with other novel PET tracers. ^{68}Ga-prostate-specific membrane antigen PET is a promising new technique for sensitive and accurate staging of primary prostate cancer and restaging of recurrent prostate cancer.[41] Agarwal reports a case of metastatic prostate adenocarcinoma which demonstrated a superscan appearance on ^{68}Ga-PSMA PET/CT, with generalized increased tracer uptake in the entire axial and appendicular skeleton and diffusely decreased physiological activity in the soft tissues. This patient had previously demonstrated a superscan appearance on bone scintigraphy.[42] ^{18}F-NaF PET/CT has recently been a subject of intense interest because of its potential to affordably provide more sensitive detection of skeletal metastases than traditional bone scintigraphy.[43] Li reports a case of metastatic recurrence of prostate cancer that revealed a superscan appearance on ^{18}F-NaF scintigraphy, with intense uptake in the axial skeleton and poor visualization of the distal appendicular skeleton and kidneys.[44] Superscan appearances have also been obtained with several of the ^{68}Ga-labeled somatostatin analogs currently in development for the staging of neuroendocrine tumors. Tan reports a ^{68}Ga-DOTATATE PET/CT superscan, accompanied by metaiodobenzylguanidine and classic bone superscan, in a case of metastatic pheochromocytoma.[45] Skeletal superscan presentations of metastatic pancreatic neuroendocrine tumor have been reported with both ^{68}Ga-DOTATATE and ^{68}Ga-DOTANOC PET/CT.[15,46]

To illustrate a typical case of metabolic skeletal superscan, we present an 18-F-FDG PET/CT superscan in which intense radiotracer sequestration by lesions throughout the skeleton and soft tissues in a patient with Burkitt lymphoma caused a reduction in physiologic uptake by the brain, myocardium, and kidneys to levels similar to the blood pool [Figure 2]; this is to our knowledge the

first reported 18-F-FDG PET/CT skeletal superscan in a case of Burkitt lymphoma. Our patient, notably, underwent pancreaticoduodenectomy for a presumed primary neoplasm of the pancreas before receiving a histological diagnosis of lymphoma and undergoing PET/CT scanning. This case emphasizes the utility of 18-F-FDG PET/CT imaging in the initial staging of oncologic patients as the diffuse skeletal and soft tissue involvement was not as apparent on conventional imaging modalities This is of special interest because several studies have demonstrated low sensitivity of 18-F-FDG PET for detection of bone marrow involvement in lymphomas;[47] it is possible that this low sensitivity is in part caused by interpretation of the diffuse, homogeneous, and symmetric skeletal activity characteristic of a superscan as an absence of metastatic foci in the bones.

Discussion

These reports highlight the importance of awareness of the 18-F-FDG PET/CT superscan to the nuclear medicine physician and radiologist specializing in the interpretation of nuclear studies. First, as we note above, it is possible for a superscan to lead to a false-negative interpretation. Second, the great reduction and occasional near-absence of brain activity we and others have observed may lead to dismissal of a positive scan as artifactual, possibly leading to repeating the scan or performing additional unnecessary studies. Third, the reduction or absence of brain uptake seen in ^{18}F-FDG-PET/CT superscans may also provide prognostic information: in a series of sixty-five patients with confirmed non-Hodgkin's lymphoma, Hanaoka *et al*. found a significant negative correlation between brain FDG uptake and total glycolytic volume, a measure of tumor burden.[48] Finally, knowledge of those cases in which a superscan appearance has been reported can help the interpreter to narrow the differential diagnosis for a patient whose imaging demonstrates a similar appearance.

Conclusion

Knowledge of the 18-F-FDG PET/CT superscan, its reported pathological causes, its benign imitators, and its clinical implications is important to the nuclear medicine physician or radiologist specializing in the interpretation of nuclear studies to avoid several diagnostic pitfalls.

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Conflicts of interest

There are no conflicts of interest.

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