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Case Report

A case of pulmonary tumor embolism diagnosed with respiratory distress immediately after FDG-PET/CT scan ☆☆☆

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ABSTRACT

Acute distress immediately following an ¹⁸F-fluorodeoxyglucose positron emission tomography/computed tomography (FDG-PET/CT) scan is an exceedingly rare event. We report a case whose condition was suddenly deteriorated in the nuclear medicine laboratory, and whose diagnosis was confirmed by FDG-PET/CT. A 67-year-old woman with left renal cell carcinoma (RCC) suddenly complained of dyspnea and tachycardia just after undergoing FDG-PET/CT. PET/CT images showed increased FDG uptakes in the left renal vein, inferior vena cava, right atrium, and bilateral hila. She was diagnosed with a massive tumor embolism from the inferior vena cava to both pulmonary arteries, and urgently underwent tumor embolism. FDG-PET/CT was helpful for diagnosing the tumor embolism and differentiating it from bland thromboembolism in this patient with RCC.

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Introduction

Since ¹⁸F-fluorodeoxyglucose positron emission tomography/computed tomography (FDG-PET/CT) is usually used for

staging and follow-up of cancer patients, it is mostly performed for patients with relatively stable conditions. However, the patient's condition may change suddenly during or right after FDG-PET/CT examination, and by chance, very rarely, PET/CT image may show the cause. Urgent and treatable

Abbreviations: CECT, contrast-enhanced computed tomography; CD, cluster of differentiation; FDG-PET/CT, ¹⁸F-fluorodeoxyglucose positron emission tomography/computed tomography; IHC, immunohistochemical; IVC, inferior vena cava; RCC, renal cell carcinoma; SUVmax, maximum standardized uptake value; TFE3, transcription factor E3; TTF, thyroid transcription factor.

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Fig. 1 – CT images 5 days before the embolism. Axial CECT, scanned 5 days before the FDG-PET/CT, showed a left renal mass with a long diameter of 78 mm (B and C, black arrowheads) encasing the left renal vein (C, black arrow) and spreading to the infra-/supradiaphragmatic IVC (A and B, black arrows) and left (D, black arrow) ovarian vein. Tumor emboli are depicted in black, and bland thrombi within the bilateral ovarian veins are gray in the schema of this condition (F).

findings can also be incidentally found even in asymptomatic patients [1].

Pulmonary embolism is one of the causes of sudden chest pain or dyspnea in patients with renal cell carcinoma (RCC). Tumors including RCC that invade inferior vena cava (IVC) can develop pulmonary embolisms. This condition causes very unstable hemodynamics and is often fatal.

We report a patient complaining of acute distress immediately after PET/CT scan and incidentally identified tumor embolus from IVC to the pulmonary artery by PET/CT image. We also present the contrast-enhanced CT (CECT) findings before and after the embolism and discuss the role of FDG-PET/CT in the diagnosis.

Case report

A 67-year-old woman without a past medical history went to a hospital complaining of gross hematuria. Screening ultrasound detected a left renal tumor with massive venous encasement. She was referred to our hospital for surgery. Pre-operative CECT showed a well-defined left renal tumor, with tumor thrombus in the left renal vein, extending into the IVC and left ovarian vein (Fig. 1A–D). Filling defects were also found in the right ovarian vein and the distal left ovarian vein (Fig. 1C and E, gray arrows). No evidence of lymph node or distant metastasis was found.

An FDG-PET/CT scan was performed 5 days after the CECT for the purpose of metastatic survey. The patient complained of sudden shortness of breath immediately after stepping off the table after the PET/CT scan. Her blood pressure was 103/61 mm Hg, but her heart rate was increased to 146/min. The PET/CT image showed FDG accumulation not only in the known left renal tumor but also in the left renal vein to the left ovarian vein and the infradiaphragmatic IVC, where a caval tumor thrombus was shortened compared to 5 days earlier (Fig. 2C, D, and E, black arrows). Moderate to high FDG accumulation was also found in both the hila (maximum standardized uptake value [SUVmax] = 8.8, Fig. 2A and E, white arrowheads)

and right atrial cavity (SUVmax = 5.6, Fig. 2B and E, white arrows). We suspected acute/subacute massive tumor embolism in bilateral pulmonary arteries and the right atrium.

Emergency CECT was immediately performed, which showed that the tumor thrombus in IVC was decreased in size compared to 5 days earlier and the stump of it seemed to be fragmented (Fig. 3C, D, and E, black arrows). The CECT also showed a blurred mass in the right atrium (Fig. 3B and E, white arrows) and filling defects in the left distal main pulmonary artery and bilateral segmental branches (Fig. 3A, white arrowheads). Her systolic blood pressure was occasionally down to 80 mm Hg, suggested a preshock condition. The cardiovascular surgery team performed an emergency open heart embolectomy, and an IVC filter was placed. The tumor specimen predominantly had a papillary structure and contained atypical cells with clear cytoplasm (Fig. 4A). Immunohistochemical (IHC) staining demonstrated positive staining for CD10 and negative staining for carbonic anhydrase IX as markers of clear cell renal carcinoma. IHC staining was also positive for α -methylacyl CoA racemase and negative for cytokeratin 7 as markers of papillary renal carcinoma. Anti-TFE3 antibody (Fig. 4B) and cathepsin K (Fig. 4C) were positive. TTF1 showed poor dyeability. Although the tumor might be an Xp11.2 translocation/TFE3 gene fusion RCC based on the IHC staining, we did not perform either fluorescence in situ hybridization or reverse transcription polymerase chain reaction to identify TFE3 gene translocation for a final diagnosis.

This patient survived as a result of the operation, and it was planned for her to receive a nephrectomy and thrombectomy in another hospital.

Discussion

RCC with IVC tumor thrombus occasionally causes micro or macro pulmonary tumor embolism [2,3]. Many cases with tumor embolism caused by malignant tumors have been reported to date. Other tumors known to cause tumor embolism include Wilms' tumor [4], renal sarcoma [5], renal peripheral

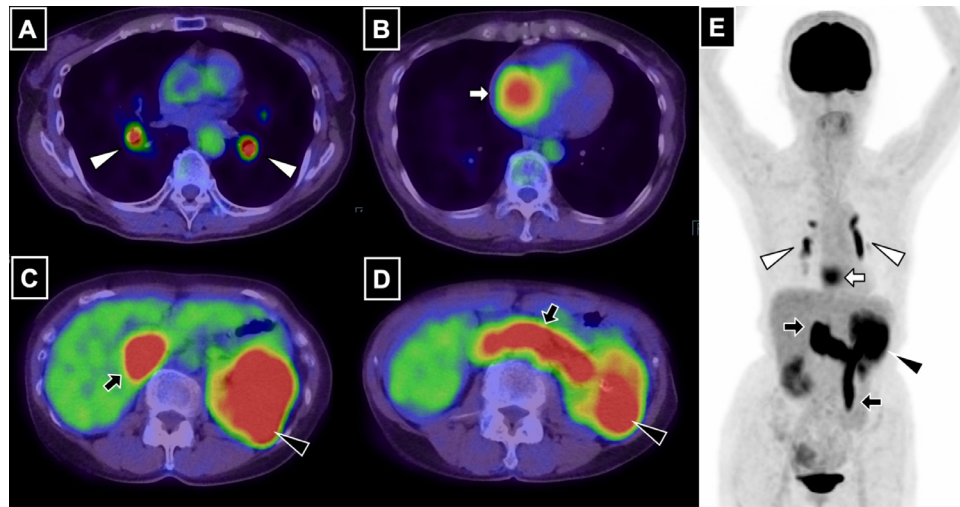


Fig. 2 – PET/CT images just after the dyspnea. Axial FDG-PET/CT fusion images (A–D) and maximum intensity projection image (E) show massive tumor emboli. FDG accumulation in the left renal tumor was continuously observed in the left renal vein and extended into an infradiaphragmatic IVC and the left ovarian vein (C, D, and E, black arrows). FDG uptakes in the left distal pulmonary artery and bilateral branches (A and E, white arrowheads) and right atrial cavity (B and E, white arrows) were also presented.



Fig. 3 – CT images 1 hour after the embolism. Axial (A–D) and coronal (E) contrast-enhanced CT images obtained right after the FDG-PET/CT are shown. The left renal tumor (C and D, black arrowheads) and tumor thrombus extending to the infradiaphragmatic IVC (C and D, black arrows) can be observed. Bilateral filling defects of pulmonary arterial branches (A, white arrowheads) and of the right atrial cavity (B and E, white arrows) were also identified. Tumor emboli are depicted in black, and bland thrombi within the bilateral ovarian veins are in gray in the schema of this condition (F).

primitive neuroectodermal tumor [6], renal angiomyolipoma [7], hepatocellular carcinoma [8], cardiac lymphoma [9], intravenous retroperitoneal leiomyosarcoma [10], and soft tissue and bone sarcomas [11]. According to a previous study, less than 6% of RCC patients with IVC tumor thrombosis developed a perioperative pulmonary embolism, but the mortality rate of this condition was reported to be 60%–75% [3]. Treatment of acute massive tumor embolism is often unsuccessful and fatal. Pulmonary tumor macroembolism sometimes occurs with subacute to chronic onset, and tumor thrombectomy itself can cause pulmonary embolism as a complication [3].

Because of its usual acute/subacute onset, pulmonary tumor embolism detected by FDG-PET/CT has rarely been reported. We experienced a patient with a massive pulmonary

artery and right atrial tumor embolism just after undergoing preoperative FDG-PET/CT in the nuclear medicine laboratory; this thromboembolism was unexpectedly detected by PET/CT imaging. Moreover, we accurately diagnosed the tumor embolism with FDG-PET/CT and CECT and performed an emergency operation without starting anticoagulation.

In the clinical settings of patients with RCC, differentiating tumor emboli from bland emboli is important. One of the ways to distinguish between a tumor embolus and a bland thrombus is to see if the lesion is enhanced in CECT. However, CT value measurement on the CECT was difficult in small pulmonary tumor thrombi and the blurring right atrial tumor thrombus in our case (Fig. 3A and B). On the other hand, PET/CT showed high FDG accumulation of the tumor

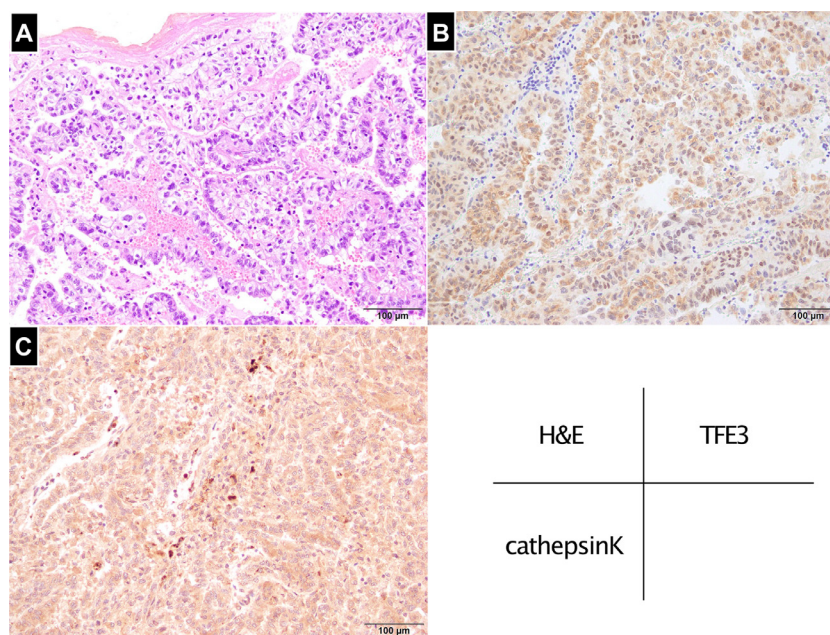


Fig. 4 – Pathological findings of the emboli. Histological images of specimens of the tumor emboli in the right atrial cavity and bilateral pulmonary arteries are presented (A-C). A papillary growth of clear tumor cells with bright cytoplasm was shown in H&E staining (A). The tumor cells were positive for TFE3 (B) and cathepsin K (C) in IHC.

thrombus same as the primary left renal tumor (Fig. 2A-E). Although bland deep venous thrombosis [12], acute pulmonary thromboembolism, and even normal pulmonary arteries can weakly accumulate FDG according to previous studies [13,14], the SUV of the tumor emboli in our case was quite high, which made it easy to differentiate it from a bland thrombosis [13]. Both CECT and FDG-PET/CT are better tools for differentiating these emboli from one another. When CECT cannot distinguish tumor emboli from bland thrombi, FDG-PET/CT might be more useful [14]. However, the FDG accumulation level of a tumor embolism might depend on the degree of differentiation and size of the tumor. FDG-PET/CT is rarely performed in emergencies with acute/subacute massive pulmonary arterial tumor embolisms.

IHC staining of the renal tumor in our case suggested Xp11.2 translocation/TFE3 gene fusion RCC [15], though the definitive diagnosis was not confirmed. Although CECT findings of Xp11.2 translocation/TFE3 gene fusion RCC are non-specific, CT findings of the renal tumor in our patient were atypical for clear cell RCC. CT showed a gradually enhanced pattern, but did not show an early enhancement and washout pattern. The IVC tumor thrombus showed homogeneous enhancement, and no necrosis was found pathologically. The SUVmax of the tumor in our patient was higher than that commonly found in clear cell RCC, though it was at a non-specific level.

Conclusions

We experienced a patient with a pulmonary tumor embolism detected by FDG-PET/CT. Attention should always be paid to

the development of pulmonary embolisms, even when FDG-PET/CT is performed, especially in patients with intravenous invasion of tumors. FDG-PET/CT is a potentially useful tool for differentiating tumor embolisms from bland macroemboli.

Patient consent

We obtained written informed consent for scientific use from the patient.

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