

[CASE REPORT]

An Autopsy Case of Anaplastic Pancreatic Cancer Complicated by Refractory Eosinophilic Peritonitis

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Abstract:

Tumor-associated blood eosinophilia (TABE) and tumor-associated tissue eosinophilia (TATE) are rare in solid cancers, and reports of eosinophilic peritonitis in these cases are extremely limited. We present the case of a 68-year-old man with pancreatic and lung cancer who developed severe eosinophilia and eosinophilic peritonitis. Despite corticosteroid therapy, refractory ascites led to a decline in performance status. An autopsy confirmed undifferentiated pancreatic adenocarcinoma with TABE, TATE, and lung cancer. This case underscores the diagnostic challenges of multiple primary malignancies with eosinophilia and highlights the need for individualized management strategies for tumor-associated eosinophilic disorders.

Key words: undifferentiated pancreatic adenocarcinoma, tumor-associated blood eosinophilia, tumor-associated tissue eosinophilia

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Introduction

In cancer patients, an increase in the peripheral blood eosinophil count may occur, a condition referred to as tumor-associated blood eosinophilia (TABE) (1). As TABE can be associated with organ dysfunction as a paraneoplastic syndrome, understanding its pathophysiology is essential. Additionally, increased eosinophil infiltration within tumor tissues may be observed in patients with cancer, a condition known as tumor-associated tissue eosinophilia (TATE). TATE has been suggested to be a prognostic factor in certain cancer types (2).

Anaplastic pancreatic ductal carcinoma, on the other hand, is an extremely rare pancreatic tumor, accounting for only 0.18% of invasive pancreatic ductal carcinomas (3). No standardized treatment has been established and the prognosis remains extremely poor.

TABE and TATE caused by solid tumors are relatively rare, with only a few reported cases being complicated by eosinophilic peritonitis. We herein report an autopsy case and review the relevant literature.

Case Report

A 68-year-old man with a history of type 2 diabetes mellitus and a smoking history of 20 cigarettes per day from 18 to 36 years of age developed progressive abdominal distension in April 20XX. In May, he experienced fatigue and constipation. He visited a local physician, where laboratory tests revealed a markedly elevated white blood cell count of 74,000/ μ L. Due to these abnormal laboratory findings, he was referred to our hospital for further evaluation and was admitted in May 20XX.

On admission, the patient was alert and had no acute distress. His vital signs were stable, with a temperature of 36.8°C, blood pressure of 113/61 mmHg, heart rate of 74 bpm, and oxygen saturation of 97% on room air. Physical examination revealed a distended, soft, and non-tender abdomen without palpable masses or signs of peritoneal irritation. No abnormalities were noted on cardiac or pulmonary examination; breath sounds were clear bilaterally, and heart sounds were normal without murmurs. However, bilateral lower limb edema was observed.

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Table. Laboratory Tests on Admission.

Hematology		Blood chemistry	
WBC	74,130 / μ L	TP	7.1 g/dL
Neutro	32.5 %	ALB	3.3 g/dL
Stab	8.5 %	Glb	3.8 g/dL
Seg	24 %	A/G	0.87
Mono	3 %	T-Bil	1.1 mg/dL
Eosino	62.5 %	ALP	238 U/L
Baso	0 %	AST	23 U/L
Lymph	2 %	ALT	28 U/L
RBC	500 $\times 10^4$ / μ L	LDH	392 U/L
Hb	14.7 g/dL	γ -GTP	387 U/L
PLT	12.7 $\times 10^4$ / μ L	ChE	103 U/L
		BUN	22.9 mg/dL
		Cre	1.21 mg/dL
		UA	6.7 mg/dL
		AMY	20 U/L
		T-Chol	117 mg/dL
		BS	128 mg/dL
		Na	134.6 mmol/L
		K	4.6 mmol/L
		Cl	101.8 mmol/L
		Ca	9.5 mg/dL
Coagulation		Serology	
PT	75 %	CRP	2.39 mg/dL
INR	1.23	AFP	1.3 ng/mL
APTT	34.4 s	CEA	1.8 ng/mL
		CA19-9	15.9 U/mL
		PSA	0.276 ng/mL
		IgE RIST	520 IU/mL

WBC: white blood cell, Neutro: neutrophil, Stab: stab neutrophil (band neutrophil), Seg: segmented neutrophil, Mono: monocyte, Eosino: eosinophil, Baso: basophil, Lymph: lymphocyte, RBC: red blood cell, Hb: hemoglobin, PLT: platelet, BUN: blood urea nitrogen, Cre: creatinine, UA: uric acid, AMY: amylase, T-Chol: total cholesterol, BS: blood sugar, Na: sodium, K: potassium, Cl: chloride, Ca: calcium, TP: total protein, ALB: albumin, Glb: globulin, A/G: albumin/globulin ratio, T-Bil: total bilirubin, ALP: alkaline phosphatase, AST: aspartate aminotransferase, ALT: alanine aminotransferase, LDH: lactate dehydrogenase, γ -GTP: gamma-glutamyl transferase, ChE: cholinesterase, PT: prothrombin time, INR: international normalized ratio, APTT: activated partial thromboplastin time, CRP: C-reactive protein, AFP: alpha-fetoprotein, CEA: carcinoembryonic antigen, CA19-9: carbohydrate antigen 19-9, PSA: prostate-specific antigen, IgE RIST: immunoglobulin E radioimmunosorbent test

Laboratory tests on admission revealed a WBC count of 74,130/ μ L, with marked eosinophilia (62.5%). The troponin I level was mildly elevated at 42.5 pg/mL. Serum tumor markers, including carcinoembryonic antigen (CEA), cytokeratin 19 fragment, carbohydrate antigen 19-9 (CA19-9), duodenal pancreatic cancer antigen 2 (DUPAN-2), and sialylated pancreatic antigen-1 (Span-1), were within the normal limits (Table).

Contrast-enhanced computed tomography (CT) of the chest and abdomen revealed multiple abnormalities. A par-

tially solid ground-glass nodule (GGN) was observed in the left lower lobe (Fig. 1a). Additionally, an enlarged mediastinal lymph node was observed at station 7 (Fig. 1b). In the abdomen, a tumor-like lesion extended from the hepatic hilum to the pancreatic head and duodenum and was accompanied by massive ascites (Fig. 1c). Bilateral adrenal masses were also detected (Fig. 1d).

The patient presented with severe abdominal distension prompting an ascitic tap for symptomatic relief. Paracentesis was performed, and ascitic fluid analysis revealed a total cell count of 688/ μ L, consisting of 475/ μ L of mononuclear cells and 213/ μ L of polymorphonuclear cells. A cytological analysis did not identify malignant cells, but demonstrated marked eosinophilic infiltration, with eosinophils comprising 50% of the total cell count.

Given the marked eosinophilia in the blood, leukemia was suspected. However, a chromosomal analysis and bone marrow biopsy ruled out leukemia. Tumor-associated eosinophilia secondary to a solid tumor was considered, and contrast-enhanced chest and abdominal CT were performed. Chest CT revealed GGN and lymphadenopathy, suggesting primary lung cancer. Abdominal CT showed tumor-like lesions around the intestines, ascites, and bilateral adrenal masses, raising the suspicion of gastrointestinal cancer with peritoneal and adrenal metastases. The abdominal mass lesion was conglomerated with the pancreatic head, duodenum, and bile duct.

These findings suggest that the underlying solid tumor was responsible for eosinophilia. Given the patient's declining performance status (PS) due to massive ascites, urgent diagnostic evaluation and therapeutic intervention are necessary.

Upper gastrointestinal endoscopy on hospital day 9 revealed diffuse mucosal erythema extending from the esophagus to the duodenum with an elevated lesion in the duodenal bulb (Fig. 2a). A forceps biopsy identified adenocarcinoma cells within the lymphatic vessels, consistent with lymphangitic carcinomatosis (Fig. 2b). Immunohistochemistry revealed napsin A-negative, cytokeratin 7 (CK7)-positive, and thyroid transcription factor-1 (TTF-1)-positive tumor cells (Fig. 2c). Lower gastrointestinal endoscopy on hospital day 10 revealed no evidence of advanced cancer from the ascending colon to the rectum but revealed mild diffuse erythema, suggestive of non-infectious colitis. Biopsies from 10 sites along the ascending to transverse colon demonstrated mild mucosal eosinophilia, but showed no malignant findings. At this stage, the primary site of the gastrointestinal cancer remained uncertain. However, the immunohistochemistry results suggest that lung cancer may be of primary origin.

By hospital day 12, the patient had elevated troponin I levels, massive eosinophilic ascites, and eosinophilic colitis, raising concerns about eosinophilic myocarditis and peritonitis. Prednisolone 80 mg (1 mg/kg) was then administered. Despite frequent paracentesis, ascitic fluid cytology on hospital day 16 confirmed malignancy and identified a combi-

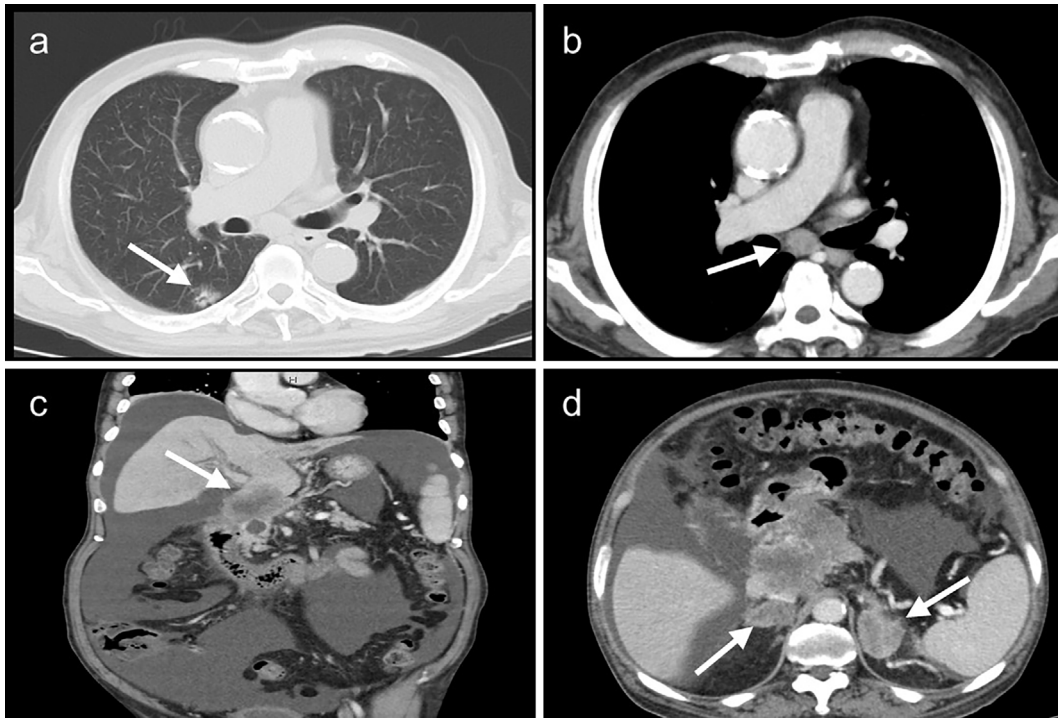


Figure 1. Contrast-enhanced chest and abdominal computed tomography (CT) images at the initial visit. a) In the left lower lobe, a part-solid ground-glass nodule (GGN) measuring 18×15 mm is identified. b) Enlarged mediastinal lymph node measuring 30×18 mm can be seen at station 7. c) A tumor-like lesion measuring 60×40 mm can be seen extending from the hepatic hilum to the pancreatic head and duodenum, accompanied by massive ascites. d) Bilateral adrenal masses measuring 45×28 mm and 53×30 mm can be seen in the right and left adrenal glands, respectively.

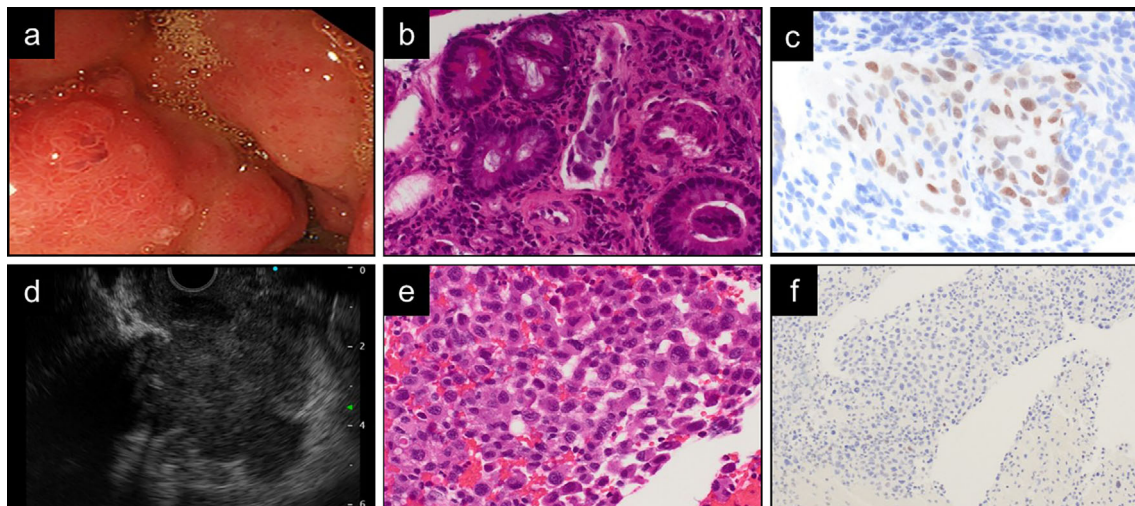


Figure 2. a) Elevated lesion in the duodenal bulb observed on esophagogastroduodenoscopy (EGD). b) Pathological findings: adenocarcinoma cells within the lymphatic vessels, consistent with lymphangitic carcinomatosis (Hematoxylin and Eosin staining, ×400). c) Adenocarcinoma cells positive for thyroid transcription factor-1 (TTF-1) observed using the 8G7G3/1 antibody (×400). d) Hyperechoic lesion identified by endoscopic ultrasound-guided tissue acquisition (EUS-TA). e) Adenocarcinoma with extensive necrosis (×400). f) The lesion is mostly negative for TTF-1 (×200).

nation of carcinoma and eosinophilic peritonitis.

Further evaluation of the pulmonary lesions was conducted. On hospital day 17, endobronchial ultrasound-guided transbronchial needle aspiration of mediastinal lymph node

#7 was performed. A histopathological examination confirmed adenocarcinoma with extensive eosinophilic infiltration around tumor cells (Fig. 3a). Immunohistochemistry identified Napsin A-positive, CK7-positive, and TTF-1-

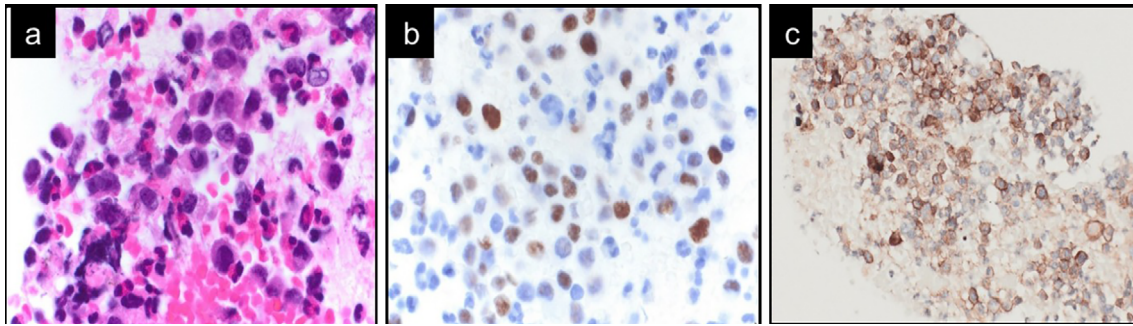


Figure 3. a) Adenocarcinoma with extensive eosinophilic infiltration around the tumor cells seen on histopathology (×1,000). b) Adenocarcinoma cells positive for thyroid transcription factor-1 (TTF-1) seen using the 8G7G3/1 antibody (×1,000). c) The programmed death-ligand 1 (PD-L1) expression rate (total number of PD-L1-expressing tumor cells/total number of tumor cells) showing a tumor proportion score (TPS) of ≥50% in lung cancer (×400).

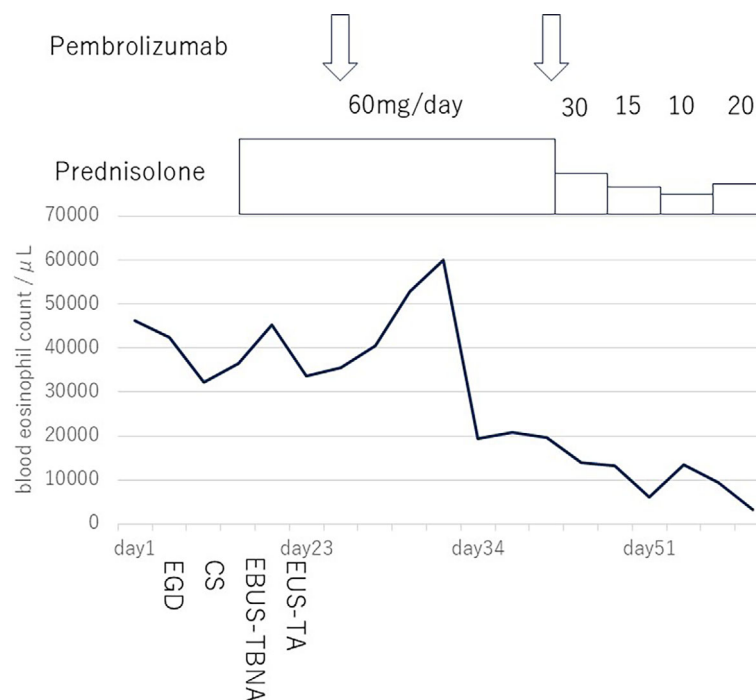


Figure 4. Changes in peripheral blood eosinophil count and treatments/examinations during hospitalization. EGD: esophagogastroduodenoscopy, CS: colonoscopy, EBUS-TBNA: endobronchial ultrasound-guided transbronchial needle aspiration, EUS-TA: endoscopic ultrasound-guided tissue acquisition. Administration of pembrolizumab (arrows).

positive tumor cells (Fig. 3b) with a programmed death-ligand 1 (PD-L1) tumor proportion score >50% (Fig. 3c). Genetic testing using an AmoyDx multipanel analysis detected no driver mutations.

Given the identical immunohistochemical staining patterns between mediastinal lymph nodes and duodenal biopsies, primary lung cancer with intestinal, adrenal, and peritoneal metastases was strongly suspected. However, as gastrointestinal metastases from lung cancer are rare, a more definitive sample is required. Endoscopic ultrasound-guided tissue acquisition (EUS-TA) was performed on hospital day 21. A 40-mm hypoechoic lesion (Fig. 2d) without blood flow was detected. Owing to stenosis of the duodenal bulb, observa-

tion was performed through the duodenal bulb to identify the tumor, followed by EUS-TA.

While awaiting histopathological results, the patient's hypoalbuminemia worsened owing to persistent massive ascites, leading to a decline in PS from 1 to 2 by hospital day 21. Although peripheral eosinophil counts decreased following the initiation of prednisolone (Fig. 4), ascites remained refractory, necessitating repeated paracentesis. Due to rapid deterioration of the patient's PS, there was insufficient time to await pathological results, and treatment for advanced lung cancer was initiated. Given the patient's PS score of 2, cytotoxic chemotherapy was deemed inappropriate, and pembrolizumab monotherapy (200 mg) was initiated on hos-

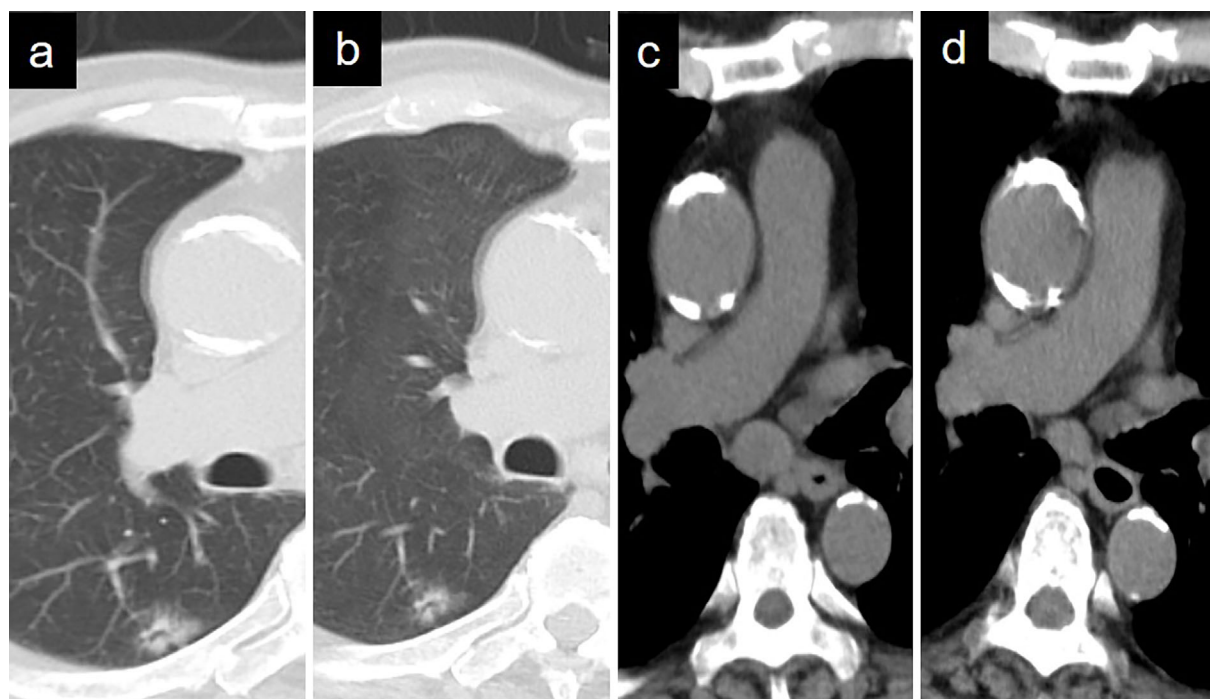


Figure 5. a, b) Ground-glass opacity (GGO) observed before pembrolizumab treatment and after two courses of treatment. c, d) Similarly, changes noted in the #7 mediastinal lymph node before and after two courses of pembrolizumab treatment.

pital day 24.

On hospital day 34, the histopathological results from EUS-TA contradicted those of the initial duodenal biopsy. Pathological findings revealed adenocarcinoma with extensive necrosis (Fig. 2e), and immunohistochemistry of the obtained tissue showed CK7-positive, Napsin A-negative and TTF-1-negative (Fig. 2f), suggesting poorly differentiated pancreatic ductal adenocarcinoma. As the patient's PS had already declined, chemotherapy for the pancreatic cancer was not feasible. Because mediastinal lymph node and adrenal metastases are also common metastatic sites of lung cancer, we judged that continuation of pembrolizumab therapy was feasible as treatment for advanced lung cancer, and therefore proceeded with the second course of administration.

On hospital day 52, the treatment response evaluation after two cycles showed regression of the primary lung tumor (Fig. 5a, 5b) and #7 mediastinal lymph node metastasis (Fig. 5c, 5d) but no improvement in the abdominal mass or ascites. The abdominal mass slightly increased in size to 83 × 81 mm and extended along the medial aspect of hepatic segment 1 and the hepatic portion of the inferior vena cava. The structures around the hepatic hilum, pancreatic head, and duodenum remain conglomerated. Subsequently, the patient transitioned to best supportive care. Deterioration of the patient's PS due to massive ascites progressed and the patient died on hospital day 63.

A pathological autopsy was performed, and the pancreatic head tumor was diagnosed as a pancreatic cancer with features of undifferentiated ductal carcinoma. The tumor exhibited direct invasion of the duodenum and hepatic serosa

along with peritoneal carcinomatosis, bilateral adrenal metastases, and omental metastases. Although TTF-1 staining was predominantly negative, focal positivity was observed. PD-L1 was detected in 100% of the tumor cells. Furthermore, there was extensive eosinophilic infiltration surrounding the undifferentiated pancreatic ductal carcinoma (Fig. 6a, b) as well as a significant eosinophilic component in the malignant ascites (Fig. 6c). In right lung segment 6, a minimally invasive adenocarcinoma (lepidic > papillary > acinar subtype) was identified. The immunohistochemical findings of mediastinal lymph node #7 were identical to those of the lung tumor. Eosinophilic infiltration was predominantly localized within the intravascular space of the alveolar septa, with minimal direct infiltration into the lung cancer itself, suggesting systemic manifestation of peripheral eosinophilia.

Discussion

The patient presented with eosinophilic peritonitis secondary to peritoneal carcinomatosis of a malignant tumor. The presence of refractory ascites posed a significant challenge for both the diagnosis and treatment. Additionally, the coexistence of lung and pancreatic cancers, particularly the rare histological subtype of undifferentiated pancreatic ductal carcinoma (accounting for only 0.18% of invasive pancreatic ductal carcinomas), further complicated the clinical diagnosis.

The patient also showed marked hypereosinophilia (HE). HE is defined as a persistent peripheral blood eosinophil count exceeding 1,500/μL for more than six consecutive

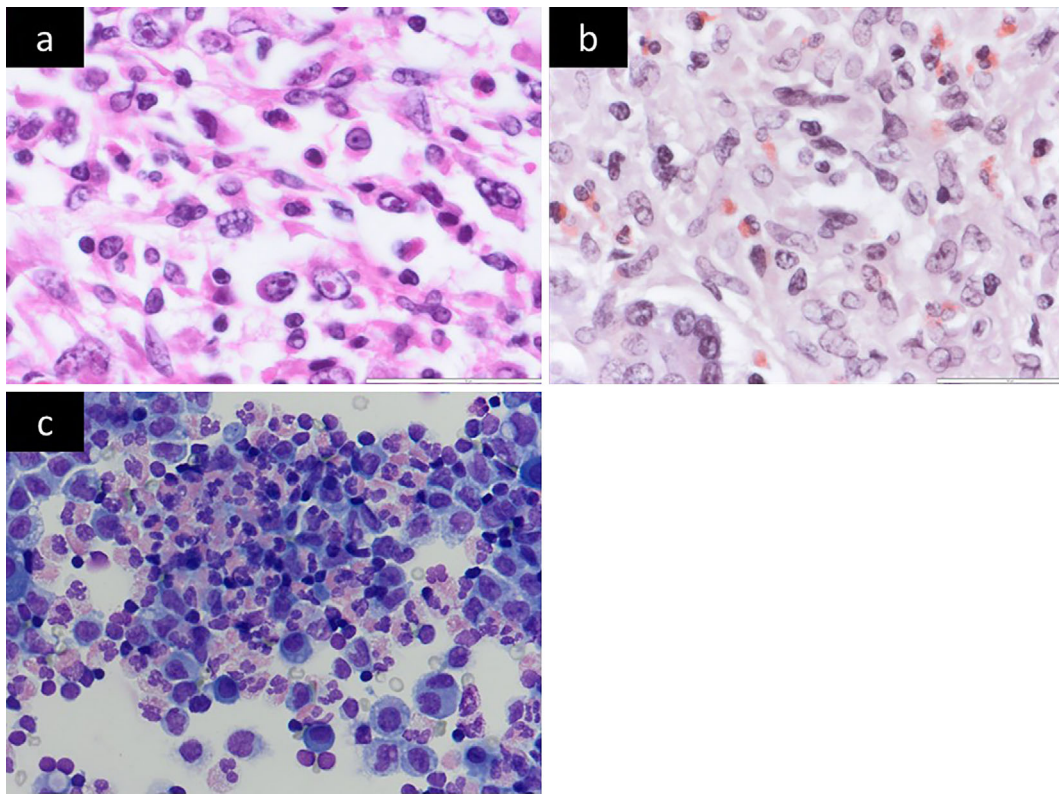


Figure 6. a) Pancreatic autopsy specimen showing scattered eosinophils admixed with pancreatic cancer cells [Hematoxylin and Eosin (H&E) staining, $\times 1,000$]. b) Pancreatic autopsy specimen showing abundant infiltration of eosinophils with orange granules [direct fast scarlet (DFS) staining, $\times 1,000$]. c) Ascitic fluid cytology showing marked eosinophilia (H&E staining, $\times 400$).

months (4). The primary treatment for HE involves addressing underlying diseases. However, when organ damage occurs, the condition is classified as hypereosinophilic syndrome (HES), for which corticosteroid therapy is recommended (5).

Initially, we considered the lung as the primary organ because mediastinal lymph node and adrenal metastases are frequently observed in lung cancer. This assumption was further supported by the negative results for pancreatic cancer-related tumor markers in the blood tests as well as the immunohistochemical findings of the duodenal bulb lesion, which partially matched those of lung cancer. Therefore, we regarded the abdominal mass as a metastatic lesion of lung cancer rather than of pancreatic cancer.

Tumor markers have relatively high sensitivity for pancreatic cancer: CEA 30-60%, CA19-9 70-80%, DUPAN-2 50-60%, and Span-1 70-80% (6). Thus, we considered tumor markers to be useful for excluding pancreatic cancer, particularly in advanced cases. In addition, immunohistochemical staining of the duodenal bulb lesion was positive for TTF-1 and CK7, which is consistent with the pathological findings of lung cancer. Because TTF-1 positivity is rare in pancreatic cancer but is frequently observed in pulmonary adenocarcinoma (7, 8), this was considered a highly specific finding.

Furthermore, as a supporting factor, the presence of primary lung cancer had already been confirmed, and it is

known that even GGNs can occasionally metastasize to other organs (9). This also reinforces our assumption that the lung was the primary site.

From an epidemiological perspective, intestinal metastasis from lung cancer is rare (10), whereas adrenal metastasis occurs in approximately 10% of pancreatic ductal adenocarcinoma cases (11). Therefore, if we had attempted early tissue sampling of the abdominal mass, the diagnosis of pancreatic cancer might have been established at an earlier stage.

EUS-TA is available in only a limited number of institutions. Previous reports indicate that anaplastic pancreatic carcinoma often exhibits extensive necrosis, making it difficult to diagnose using EUS-TA, with many cases diagnosed only after surgical resection (12). Nevertheless, since its inclusion in the Japanese national health insurance system in 2010, EUS-TA has been widely adopted for pancreatic tumors, and it is expected that more cases of anaplastic pancreatic carcinoma will be diagnosed using EUS-TA in the future.

HE associated with malignancies, known as TABE, is primarily observed in hematologic malignancies but has also been reported in solid tumors, including lung, pancreatic, thyroid, head and neck, and renal cancers (6). Previous reports have described cases of both lung adenocarcinoma and undifferentiated pancreatic ductal carcinoma, similar to ours (7, 8). TABE is thought to result from tumor lysis or

metastasis and has been identified as a poor prognostic factor for cancer (6).

Conversely, TATE refers to eosinophilic infiltration of the tumor microenvironment. The prognostic implications of TATE vary among cancer types (2, 9-14), with some cancers, including lung cancer, showing an association with a favorable prognosis (2, 6, 15). Additionally, TABE and TATE have been suggested as potential predictive biomarkers for improved responses to immune checkpoint inhibitors (ICIs) (16).

In our case, autopsy revealed significant eosinophilic infiltration surrounding the pancreatic carcinoma, indicating a high likelihood of concurrent TATE and TABE. While the presence of TABE does not necessarily imply the presence of TATE (6, 17), reports have documented the coexistence of these phenomena in both pancreatic and lung cancers (8, 16). However, the impact of such coexistence on prognosis and its role as a predictive marker for ICI therapy remains unclear due to the limited available literature and the potential influence of tumor type (16).

Undifferentiated pancreatic carcinoma is characterized by a pronounced inflammatory response, including leukocytosis, elevated C-reactive protein levels, and fever, which are not typically observed in conventional pancreatic carcinomas (15). Strong eosinophilic inflammation induced by the tumor may have contributed to the coexistence of TATE and TABE.

The most challenging aspect of this case was the management of refractory ascites. The malignant ascites was found to be eosinophilic in nature, and a histological examination of the peritoneum demonstrated prominent eosinophilic infiltration around the pancreatic tumor, suggesting eosinophilic peritonitis secondary to peritoneal dissemination of the malignancy. Although clinical suspicion of HES prompted the initiation of prednisolone at 1 mg/kg, neither the peripheral eosinophil count nor ascitic fluid volume could be adequately controlled.

Eosinophilic peritonitis is generally considered responsive to corticosteroid therapy (18); however, most prior reports have involved cases associated with peritoneal dialysis (19, 20). The efficacy of corticosteroid therapy in eosinophilic peritonitis arising from carcinomatous peritoneal dissemination, particularly in TATE-related cases, remains unclear.

This case report highlights the rare occurrence of concurrent solid malignancies associated with TABE and TATE. HE can lead to organ dysfunction through eosinophil-mediated inflammation, which directly affects the PS. Managing this condition remains challenging because of the lack of established treatment guidelines. Therefore, individualized treatment strategies based on the tumor type, extent of organ involvement, and disease severity are crucial. Timely intervention is essential to prevent disease progression and optimize patient outcomes.

The authors state that they have no Conflict of Interest (COI).

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