EJCRIM
European Journal
of Case Reports
in Internal Medicine

PULMONARY TUMOUR EMBOLISM FROM GASTRIC ADENOCARCINOMA CAUSING RESPIRATORY AND CARDIAC FAILURE MAY NOT BE IDENTIFIED ON STANDARD ANTE-MORTEM IMAGING

Konstantīns Ščerbakovs[1,2], Valentīna Mihejeva[1,2], Sergejs Dubencovs[3], Evelīna Stunda[3], Dace Žentiņa[1,2]

[1] Department of Lung Diseases and Thoracic Surgery, Pauls Stradins Clinical University Hospital, Riga, Latvia
[2] Riga Stradins University, Riga, Latvia
[3] Department of Pathology, Pauls Stradins Clinical University Hospital, Riga, Latvia

Corresponding author's e-mail: scerbakovs.k@gmail.com

Received: 26/02/2025
Accepted: 11/03/2025
Published: 26/03/2025

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: The deceased patient's legal representative has given an informed consent.
This article is licensed under a Commons Attribution Non-Commercial 4.0 License

How to cite this article: Ščerbakovs K, Mihejeva V, Dubencovs S, Stunda E, Žentiņa D. Pulmonary tumour embolism from gastric adenocarcinoma causing respiratory and cardiac failure may not be identified on standard ante-mortem imaging. *EJCRIM* 2025;**12**:doi:10.12890/2025_005294

ABSTRACT

Pulmonary tumour embolism is severe complication of malignancy, often presenting with progressive dyspnoea and respiratory failure. Due to their non-specific symptoms and absence of clear imaging findings, diagnosis is frequently delayed or made post-mortem. This is a case of previously undiagnosed gastric adenocarcinoma presenting with rapidly worsening respiratory symptoms: hypoxaemia, pulmonary hypertension and heart failure. Standard imagining – computed tomography angiography and echocardiography – provide indirect evidence, definitive diagnosis of pulmonary tumour embolism confirmed post-mortem. This case highlights the diagnostic challenges associated with a pulmonary tumour embolism and underscores the importance of early suspicion of malignancy-related complications in cases of progressive respiratory and cardiac failure without radiographic evidence of pulmonary embolism.

KEYWORDS

Pulmonary tumour embolism, lymphangitic carcinomatosis, gastric adenocarcinoma

LEARNING POINTS

- Standard diagnostic imaging lacks specificity and fails to detect pulmonary tumour embolism. Computed tomography angiography and echocardiography provide indirect evidence, but definitive diagnosis often requires histopathological confirmation, frequently post-mortem.

INTRODUCTION

Pulmonary embolism is a common and potentially lethal disease in active cancer patients. Pulmonary tumour embolism and pulmonary lymphangitic carcinomatosis are rare complications of malignancies, particularly gastric adenocarcinoma, often presenting with dyspnoea and pulmonary hypertension. Pulmonary lymphangitic carcinomatosis involves tumour infiltration of pulmonary

EFIM
European Federation of Internal Medicine

Publisher
smc media srl

lymphatics, while pulmonary tumour embolism is characterised by tumour emboli in the pulmonary vessels[1-4]. Pulmonary tumour thrombotic microangiopathy is a rare form of pulmonary tumour embolism clinically indistinguishable from pulmonary thromboembolic disease; it is characterised by fibro cellular intimal proliferation of small pulmonary arteries and arterioles in patients with intraluminal tumour embolization[5]. All conditions have poor prognosis, with survival often measured in months. Diagnostic imaging lacks specificity, and definitive diagnosis typically requires biopsy[1-4].

CASE DESCRIPTION

A middle-aged male was admitted to the rural hospital with progressive dyspnoea, cough and night sweats for three months. He was a non-smoker, worked as an electrician, and had no occupational or environmental exposures. The patient's family history was significant for an unspecified metastatic malignancy in his mother and a diagnosis of bladder cancer in his father. An outpatient chest computed tomography (CT) showed non-specific bronchiolitis, but abdominal CT revealed lymphadenopathy around the pancreas and stomach *(Fig. 1)*. Upon hospital admission, the patient had elevated D-dimer levels and decreased oxygen saturation. Pulmonary embolism was ruled out with lung CT angiography, but interstitial changes were noted. Magnetic resonance imaging of the abdomen ruled out pancreatic involvement and confirmed pathological lymphadenopathy around the pancreas and stomach. On esophagogastroduodenoscopy, suspicious gastric mucosal infiltration was visualised; a biopsy was taken. Serial blood tests showed progressive leukocytosis, and elevated inflammatory markers and D-dimer levels *(Table 1 and 2)*.

During hospitalisation, the patient developed sinus tachycardia (130 bpm) and ECG changes (T-wave inversion in V1–V3), suggestive of cardiovascular strain. Serial cardiac markers were initially normal, and the patient denied experiencing chest pain. However, within a week his respiratory failure worsened, with oxygen saturation dropping to 62%. He was transferred to the university hospital in a critical condition and initiated on high-flow nasal cannula therapy. Upon admission to the university hospital, blood tests showed elevated troponin and, indicating right heart strain, persistent leucocytosis and thrombocytopenia, and worsening inflammatory markers (C-reactive protein, procalcitonin) *(Table 3 and 4)*. An additional chest CT was performed, showing diffuse thickening of small airways and signs of bronchiolitis, with no significant changes compared to the previous scan *(Fig. 2)*. Therapy with anticoagulants continued.

Echocardiography findings were:

- right heart overload and pulmonary hypertension (estimated pulmonary artery pressure: 65–70 mmHg);
- right ventricular dilation and dysfunction (tricuspid annular plane systolic excursion 13 mm);
- preserved left ventricular function (left ventricular ejection fraction 60%);
- no pericardial effusion.

Following cardiologist consultation, tadalafil (a phosphodiesterase type 5 inhibitor) was initiated. Despite receiving intensive care, the patient's respiratory failure continued to worsen. The final pathology report confirmed the diagnosis of gastric adenocarcinoma (Grade 3, T3N2M0), and the oncology board recommended surgery. However, due to severe respiratory compromise, surgery was contraindicated. Non-invasive ventilation was

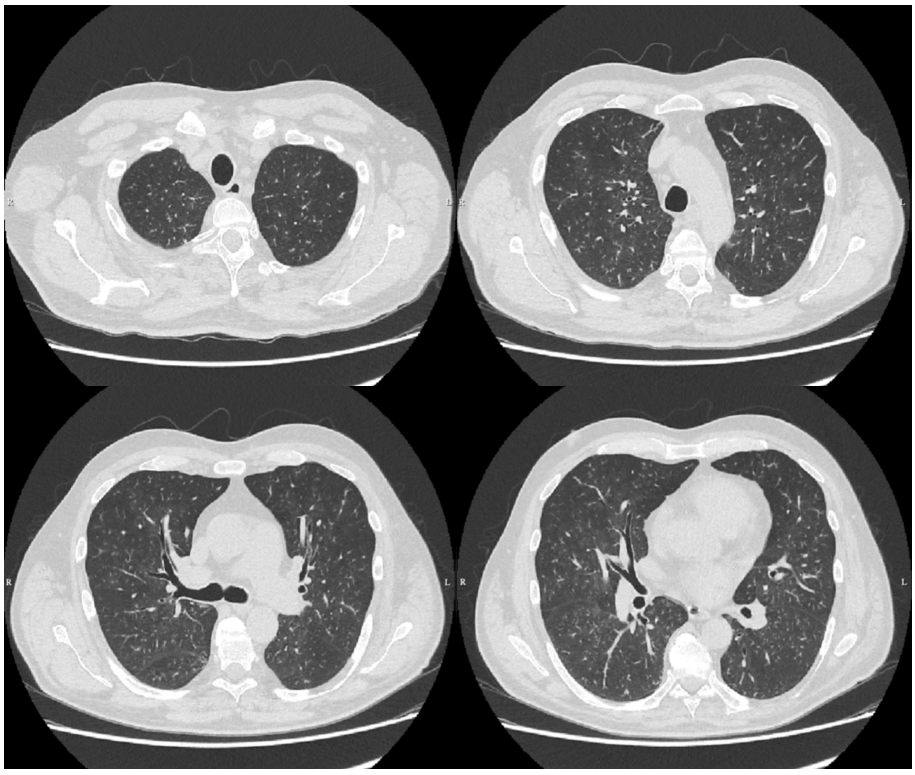


Figure 1. Outpatient lung CT a month before hospitalisation – diffusely thickening of small airways, signs of bronchiolitis.

Test	Reference	14.05.2024	19.05.2024	23.05.2024	27.05.2024
White blood cells (×10³/µl)	4–10	7.28	7.33	11.55	15.38
Red blood cells (×10⁶/µl)	4.5–5.8	4.66	4.06	4.30	4.44
Haemoglobin (g/dl)	13–17.5	16.0	14.0	14.6	15.3
Haematocrit (%)	40–50	44.4	39.8	41.4	43.3
Mean corpuscular volume (fl)	80–100	95.3	98.0	96.3	97.5
Mean corpuscular haemoglobin (pg)	27–33	34.3	34.5	34.0	34.5
Mean corpuscular haemoglobin concentration (g/dl)	31.5–36	36.0	35.2	35.3	35.3
Platelets (×10³/µl)	150–400	185	138	130	154
Red cell distribution width – SD (fl)	37.8–46.1	44.3	46.6	46.5	47.4
Red cell distribution width – CV (%)	12.3–14.3	12.9	13.1	13.1	13.2
Mean platelet volume (fl)	6–11	8.8	9.3	9.7	10.4
Platelet large cell ratio	18.5–42.3	15.7	18.9	22.0	27.7
Platelet distribution width (%)	11–18	8.9	9.8	10.3	10.9
Plateletcrit (%)	0.15–0.5	0.16	0.13	0.13	0.16

Table 1. Complete blood count parameters.

Test	Reference	14.05.2024	19.05.2024	23.05.2024	27.05.2024
Creatinine (µmol/l)	62–106	72.8	63.1	64.5	71.0
Glomerular filtration rate (ml/min/1.73 m²)	100–150	101	120	117	104
Sodium (mmol/l)	136–145	138	139.2	135.7	137.3
Potassium (mmol/l)	3.5–5.1	3.98	3.5	4.1	3.6
C-reactive protein (mg/l)	0–5	63.94	14.8	38.3	31.3
Troponin T (ng/l)	24.95	-	-	33.71	-
D-dimer (ng/ml)	50–500	8,070	5,620	4,260	-

Table 2. Biochemical markers.

initiated, followed by mechanical ventilation, but the patient deteriorated and passed away. Autopsy findings were gastric adenocarcinoma with tumour emboli in pulmonary blood vessels *(Fig. 3 and 4)*. In the available-for-autopsy tissue samples no evidence of thrombosis within the lymphatic vessels was observed; however, based on the clinical course, the possibility of lymphangitic carcinomatosis cannot be ruled out.

DISCUSSION

Malignancy is a well-known risk factor for thrombotic complications, particularly pulmonary tumour embolism, which can cause pulmonary hypertension and right heart strain through the intravascular spread of tumour cells. Tumour cells disseminate via angioinvasive pathways, leading to pulmonary tumour microembolisation or lymphangitis carcinomatosis – both recognised complications of malignancy. In these conditions, metastatic cells spread into the pulmonary blood vessels and lymphatic system, respectively, without directly infiltrating the lung parenchyma or forming solid tumour masses, which differentiates them from pulmonary metastases[4]. Overlap syndromes regularly occur and terminology is often used interchangeably in the literature. Both conditions present with progressive dyspnoea and respiratory failure, often leading to diagnostic delays due to non-specific initial presentations. Prognosis for pulmonary tumour embolism and pulmonary lymphangitic carcinomatosis remains

Test	Reference	29.05.2024	31.05.2024	01.06.2024
White blood cells (×10^9/l)	4–10	↑ 13.6	↑ 12.1	↑ 15.5
Red blood cells (×10^{12}/l)	4.5–5.8	4.67	4.62	4.84
HGB (Haemoglobin) g/l	130–175	162	160	168
Haematocrit (%)	40–50	45.6	45.1	46.9
Mean corpuscular volume (fl)	80–100	97.8	97.7	96.8
Mean corpuscular haemoglobin (pg)	27–33	↑ 34.7	↑ 34.7	↑ 34.6
Mean corpuscular haemoglobin concentration (g/l)	320–360	355	355	358
Red cell distribution width (%)	11.5–14.5	↑ 14.9	↑ 14.6	↑ 14.9
Platelets (×10^9/l)	150–400	↓ 132	↓ 120	↓ 121
Mean platelet volume (fl)	7.5–11.5	8.6	8.8	8.9
Neutrophils (%)	40–75	↑ 90.7	-	-
Lymphocytes (%)	20–45	↓ 4.8	-	-
Monocytes (%)	2–10	4.4	-	-
Eosinophils (%)	1–5	↓ 0.0	-	-
Basophils (%)	0–1	0.1	-	-
Neutrophils (×10^9/l)	2–7.5	↑ 12.3	-	-
Lymphocytes (×10^9/l)	1–3	↓ 0.6	-	-
Monocytes (×10^9/l)	0.2–1	0.6	-	-
Eosinophils (×10^9/l)	0.02–0.5	0.0	-	-
Basophils (×10^9/l)	0–0.1	0.0	-	-

Table 3. Complete blood count parameters.

Test	Reference	30.05.2024	31.05.2024	01.06.2024
Brain natriuretic peptide (pg/ml)	< 100	-	↑ 653.27	-
Creatine kinase-MB (ng/ml)	< 5	-	2.69	3.40
Troponin I (ng/l)	< 40	↑ 3,258.88	↑ 3,659.46	↑ 3,492.02
Haematocrit (%)	40–50	45.6	45.1	46.9

Table 4. Biochemical markers.

extremely poor, with survival ranging from weeks to months. Studies indicate that pulmonary lymphangitic carcinomatosis patients typically survive three months post-diagnosis, while pulmonary tumour embolism with tumour thrombotic microangiopathy provides an even shorter life expectancy[2-4]. In this case, the patient's prolonged dyspnoea, tachycardia and ischaemic ECG changes suggested increased pulmonary vascular resistance. Elevated D-dimer levels and hypoxaemia, despite the absence of visible pulmonary embolism, supported the diagnosis of pulmonary tumour embolism-related pulmonary hypertension. CT angiography fails to detect microscopic tumour emboli, and in this case, only subtle interstitial changes were noted. Echocardiography showed right ventricular dysfunction and pulmonary hypertension, strongly suggesting a pulmonary vascular involvement. Histological confirmation remains the gold standard but is rarely achieved ante-mortem. Gastric adenocarcinoma was diagnosed via biopsy, but pulmonary involvement was only confirmed post-mortem – pulmonary blood vessels contained tumour cells and organised thrombi.

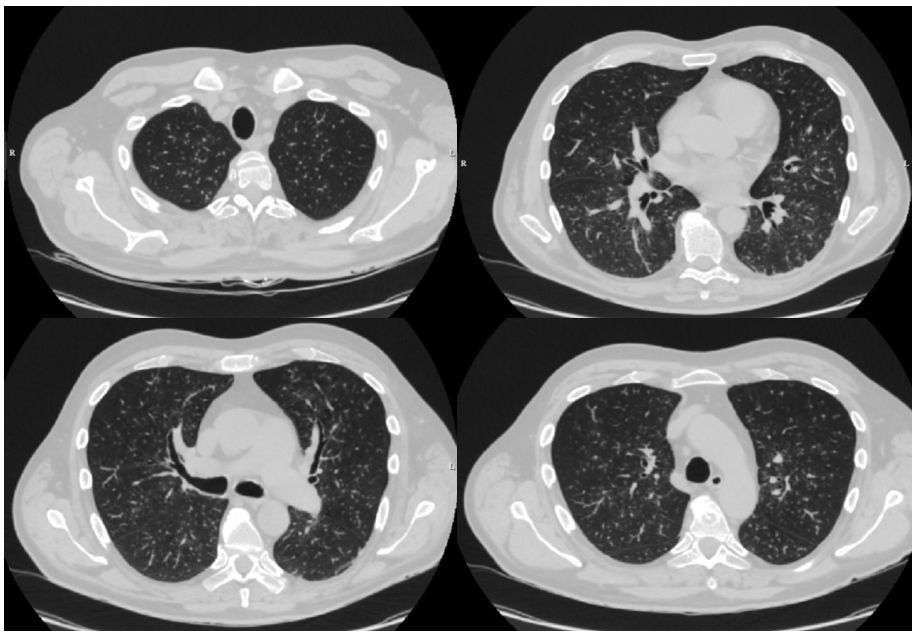


Figure 2. Lung CT at the university hospital (description above in the text).

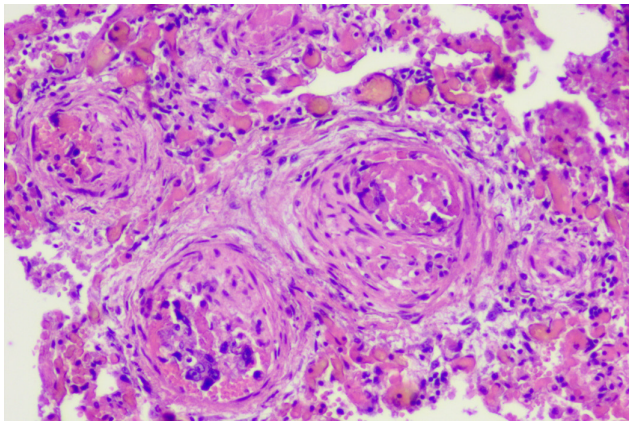


Figure 3. Pulmonary blood vessel showing occlusion with fresh and partially organised thrombus and tumour cells. Extravasated red cells in surrounding tissues (40×).

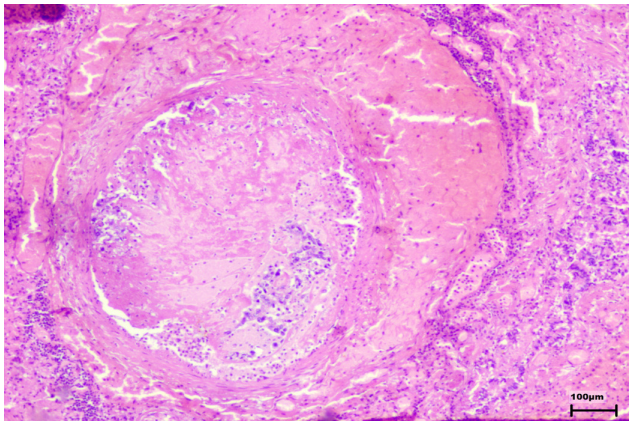


Figure 4. Pulmonary blood vessels with thickened walls containing tumour cells and organised thrombi (100×).

Management of pulmonary tumour embolism is largely supportive, focusing on oxygen therapy, anticoagulation and treatment of the underlying malignancy. Due to rapid disease progression, interventions are often palliative. In this case, surgical resection was recommended by the oncology board but was not feasible due to respiratory failure; the presence of pulmonary hypertension and right ventricular failure further worsens outcomes. Despite anticoagulation and pulmonary hypertension medications, high-flow nasal cannula therapy and subsequential mechanical ventilation, the patient's condition continued to worsen, ultimately leading to death.

CONCLUSION

This case highlights the diagnostic challenges associated with pulmonary tumour embolism. Standard imaging may fail to detect tumour emboli in the early stages – computed tomography angiography and echocardiography provides indirect evidence, and definitive diagnosis requires histopathologic confirmation, which is frequently made post-mortem. This case underscores the importance of early suspicion of malignancy in cases of progressive hypoxaemia and/or heart failure without radiographic evidence of pulmonary embolism.

REFERENCES

1. Khachekian A, Shargh S, Arabian S. Pulmonary lymphangitic carcinomatosis from metastatic gastric adenocarcinoma: case report. *J Am Osteopath Assoc* 2015;**115**:332–337.
2. Restrepo J, Carvajal-Fierro CA, Facundo H, González F, Ramírez AM, Beltran R, et al. Pleural and pulmonary dissemination patterns from gastric adenocarcinoma among patients with treated primary disease in Latin America. *Front Surg* 2022;**9**:969397.
3. Belhassine M, Papakrivopoulou E, Venet C, Mestdagh C, Schroeven M. Gastric adenocarcinoma revealed by atypical pulmonary lymphangitic carcinomatosis. *J Gastrointest Oncol* 2018;**9**:1207–1212.
4. Engel J, Auer J. Pulmonary tumour embolism and lymphangitis carcinomatosa: a case report and review of the literature. *J Cardiothorac Surg* 2022;**17**:105.
5. Kamouh A, Nelson A, Vats S, Powell R Jr, Missov E. Pulmonary tumor thrombotic microangiopathy associated with right ventricular rupture and hemopericardium: a case report. *J Cardiol Cases* 2014;**9**:230–232.