

Deglutition-Induced Cough Revealing an Esophagotracheal Fistula Complicating Hodgkin Lymphoma: A Case Report

Review began 10/13/2026

Review ended 10/27/2026

Published 10/29/2026

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DOI: 10.7759/cureus.118799

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Abstract

Acquired esophago-respiratory fistulas are rare and potentially life-threatening complications, most often of neoplastic origin. They are most commonly associated with esophageal or lung cancer, whereas their occurrence in the setting of Hodgkin lymphoma is exceptional. We report a case of a 37-year-old male followed for recurrent or progressive Hodgkin lymphoma under second-line chemotherapy with ICE (ifosfamide, carboplatin, and etoposide) regimen, admitted for a three-month history of chronic cough systematically triggered by swallowing (Ono's sign). Physical examination revealed severe malnutrition (BMI = 16 kg/m²) along with bulky cervical and mediastinal lymphadenopathy. Chest computed tomography (CT) demonstrated an esophagotracheal communication at the T3-T4 level within necrotic mediastinal masses. Endoscopy could not be performed because of respiratory instability. Swallowing-induced cough should prompt investigation for an esophago-respiratory fistula, particularly in patients with mediastinal tumor involvement, even in the absence of dysphagia. Chest CT allows direct identification of the fistulous communication and assessment of the underlying lesion and its complications, especially when endoscopy cannot be performed.

Categories: Hematology, Oncology, Gastroenterology

Keywords: computed tomography, esophago-tracheal fistula, hodgkin lymphoma, mediastinal lymphadenopathy, ono's sign, swallowing-induced cough, tumor necrosis

Introduction

Acquired esophagotracheal fistulas constitute an abnormal communication between the esophagus and the respiratory tract, exposing patients to the risk of aspiration pneumonia, severe malnutrition, mediastinitis, and sepsis [1]. In adults, malignant conditions account for the majority of etiologies [1]. We report a case of a patient with progressive Hodgkin lymphoma complicated by an esophagotracheal fistula revealed by a swallowing-induced cough, and discuss the underlying pathophysiological mechanisms, diagnostic approach, and therapeutic options in light of the published literature.

Case Presentation

A 37-year-old male had been followed in the hematology department since 2022 for classic Hodgkin lymphoma, diagnosed by immunohistochemical study of a biopsy from a right supraclavicular lymph node. He initially received two cycles of ABVD (doxorubicin, bleomycin, vinblastine, and dacarbazine) chemotherapy (October-November 2023) without radiotherapy, with clinical disappearance of the cervical lymph node. A PET-CT scan was requested to assess the response but was not performed, and the patient was subsequently lost to follow-up. Complete remission was therefore not documented, and it remains uncertain whether the subsequent presentation represented relapse after clinical response or progression of persistent disease. He presented again in June 2026 with progressive lymphadenopathy, including a large fistulized cervical lymph node, whose immunohistochemical study (CD30, CD15, PAX5) confirmed recurrent or progressive mixed-cellularity Hodgkin lymphoma, prompting initiation of a new line of chemotherapy. At the time of presentation, the patient was receiving the ICE (ifosfamide, carboplatin, and etoposide) regimen as second-line therapy, administered as a three-day cycle every three weeks; the most recent cycle had been completed in August 2026.

The patient was admitted for a three-month history of chronic cough systematically triggered by swallowing, particularly upon ingestion of liquids and semi-liquids, without associated dysphagia or odynophagia, accompanied by dyspnea at rest and recurrent pleural effusions requiring several therapeutic thoracenteses. On admission, physical examination showed a respiratory rate of 26 breaths/minute, an oxygen saturation of 86% on room air, a BMI of 16 kg/m² reflecting severe malnutrition, and multiple cervical, supraclavicular,

How to cite this article

Gracien M, Ekomisa Boyele F, Allen K, et al. (October 29, 2026) Deglutition-Induced Cough Revealing an Esophagotracheal Fistula Complicating Hodgkin Lymphoma: A Case Report. Cureus 18(10): e118799. DOI 10.7759/cureus.118799

and axillary lymphadenopathy.

Initial laboratory investigations (Table 1) revealed anemia with a hemoglobin level of 8.2 g/dL, leukocytosis at 10,690 cells/mm³ predominantly composed of neutrophils (10,050 cells/mm³), and lymphopenia at 500 cells/mm³. The platelet count was normal at 262,000 cells/mm³.

Parameters	Patient value	Reference range
Hemoglobin (g/dL)	8.2	13–17 (male)
White blood cell count (/mm³)	10,690	4,000–10,000
Neutrophils (/mm³)	10,050	1,500–7,000
Lymphocytes (/mm³)	500	1,000–4,000
Platelets (/mm³)	262,000	150,000–400,000
C-reactive protein (CRP) (mg/L)	40	<5
Albumin (g/L)	20	35–50
Urea (g/L)	0.18	0.15–0.45
Creatinine (mg/L)	3.22	7–13
Lactate dehydrogenase (LDH) (IU/L)	640	120–250
Sodium (Na⁺) (mmol/L)	138	135–145
Potassium (K⁺) (mmol/L)	3.8	3.5–5.0
Chloride (Cl⁻) (mmol/L)	105	98–107
Aspartate aminotransferase (ASAT) (IU/L)	25	10–40
Alanine aminotransferase (ALAT) (IU/L)	32	7–56
Total bilirubin (mg/dL)	0.6	0.2–1.2
Beta-2 microglobulin (mg/L)	3	0.8–2.4

TABLE 1: Laboratory findings on admission.

Inflammatory markers showed an elevated CRP of 40 mg/L and severe hypoalbuminemia at 20 g/L, reflecting a marked biological inflammatory syndrome and a catabolic state. Serum creatinine was 3.22 mg/L, which is low and likely related to reduced muscle mass in the context of severe malnutrition, and urea of 0.18g/L. Serum electrolytes were unremarkable, with sodium (Na⁺) at 138 mmol/L, potassium (K⁺) at 3.8 mmol/L, and chloride (Cl⁻) at 105 mmol/L. Liver function tests were normal, with an aspartate aminotransferase (AST) level of 25 IU/L, an alanine aminotransferase (ALT) level of 32 IU/L, and a total bilirubin level of 0.6 mg/dL. Finally, lactate dehydrogenase (LDH) was elevated at 640 IU/L, and beta-2-microglobulin was increased at 3 mg/L, reflecting the intensity of cellular turnover and systemic inflammatory activity.

Cervicothoracic CT, performed on day 2, demonstrated extensive lymphomatous progression with bulky necrotic mediastinal lymphadenopathy, pulmonary parenchymal involvement, bilateral pleural effusions, and heterogeneous, irregular thickening of the thoracic esophagus between D2 and D4. A direct communication between the esophagus and the trachea was visualized at the D3-D4 level (Figure 1). The associated necrotic mediastinal lymphadenopathy, esophageal wall thickening, and bilateral pleural effusion are shown in Figure 2.

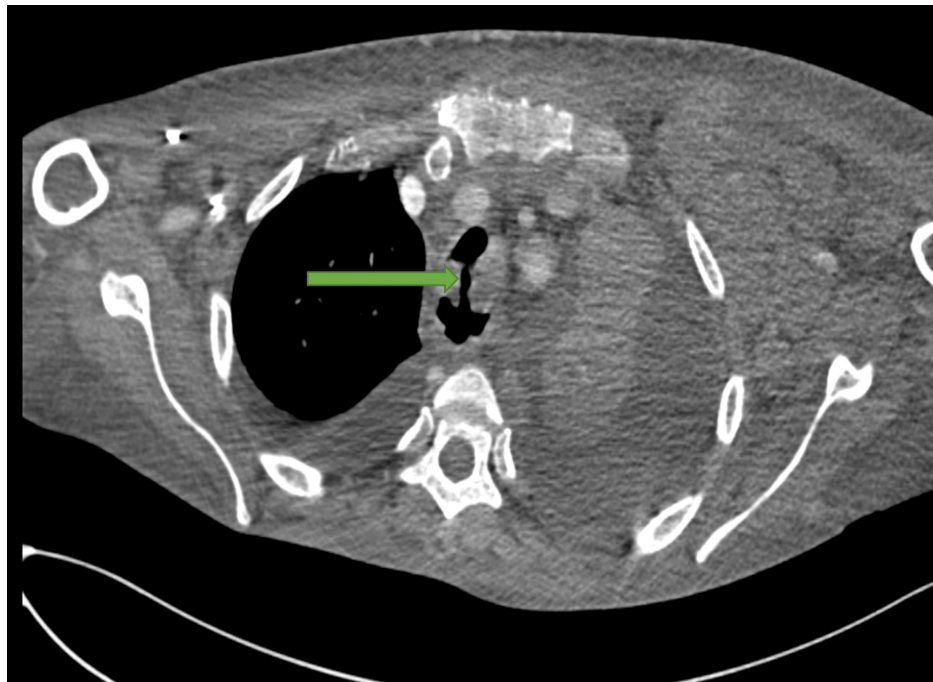


FIGURE 1: Mediastinal window of a chest CT scan (axial view) showing a tracheoesophageal fistula at the T3-T4 level (green arrow).

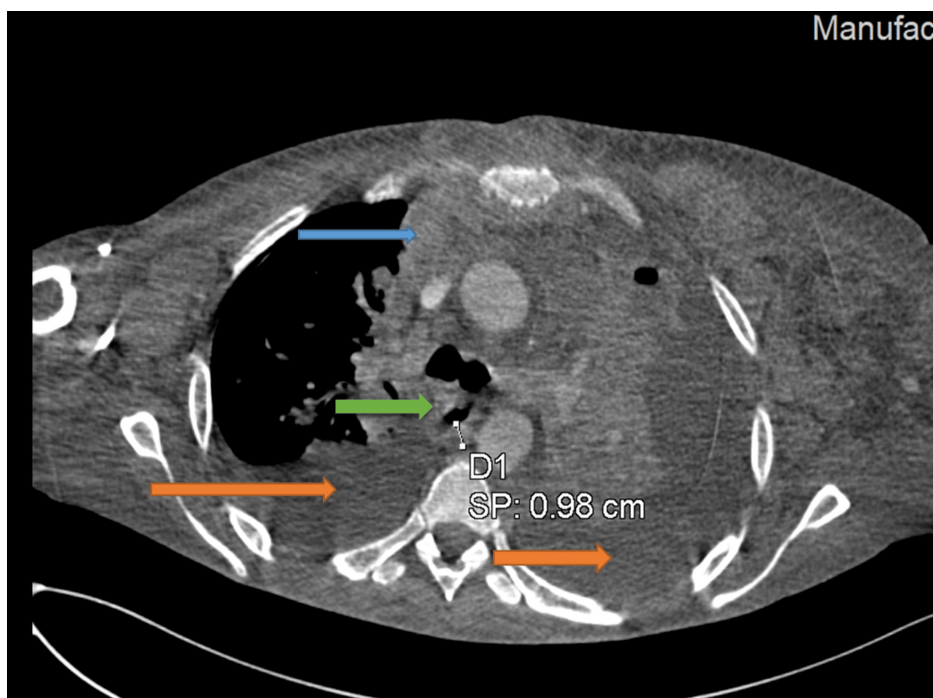


FIGURE 2: Mediastinal window of a chest CT scan (axial section) showing necrotic mediastinal lymphadenopathy (blue arrow), esophageal wall thickening measuring 10 mm (green arrow), and bilateral pleural effusion (orange arrows).

The diagnosis of esophagotracheal fistula, presumably secondary to locoregional progression of the lymphoma, was established based on the concordance of clinical and CT findings. The patient was kept nil per os and received parenteral nutrition, oxygen therapy (initially 5 L/min via nasal cannula, subsequently increased to 10 L/min via a non-rebreather mask), and empiric antibiotic therapy with amoxicillin-

clavulanic acid. Given the advanced progressive malignancy, severe malnutrition, and worsening respiratory status, admission to the intensive care unit was not feasible in our hospital setting, and invasive mechanical ventilation was not instituted. Following multidisciplinary assessment, endoscopic evaluation and esophageal stent placement were deemed unfeasible because of the patient's respiratory instability and poor general condition. Despite supportive measures, the patient's respiratory status progressively deteriorated, and he died on day 10 of hospitalization.

The timeline of the case is summarized in Table 2.

Day	Event
About 3 months before day 1	Onset of cough triggered by swallowing
Before day 1	In 2022: mixed-cellularity classic Hodgkin lymphoma diagnosed on a right supraclavicular lymph node biopsy. October-November 2023: two cycles of ABVD, without radiotherapy; clinical disappearance of the cervical lymph node; PET-CT scan requested but not performed; lost to follow-up. June 2026: recurrent or progressive disease confirmed by immunohistochemistry, with positivity for CD30, CD15, and PAX5; second-line ICE; last cycle completed 3 days before admission.
Day 1 (admission)	Chronic cough triggered by swallowing, dyspnea at rest, recurrent pleural effusions. Respiratory rate 26 breaths/minute, SpO ₂ of 86% on room air, BMI = 16 kg/m ² , multiple cervical, supraclavicular, and axillary lymphadenopathy. Laboratory work-up presented in Table 1.
Day 2	Cervicothoracic CT: esophagotracheal fistula at the D3-D4 level (Figures 1, 2). Nil per os, parenteral nutrition, oxygen therapy (5 L/min via nasal cannula, then up to 10L/min via non-rebreather mask), and empiric amoxicillin-clavulanic acid. Endoscopic exploration not feasible because of respiratory instability.
Day 3	Multidisciplinary assessment by pulmonology, thoracic surgery, anesthesia-intensive care; esophageal stent placement judged not feasible because of respiratory instability and poor general condition; ICU admission not feasible in our hospital setting; supportive care continued.
Days 4-9	Progressive respiratory deterioration occurred despite supportive measures.
Day 10	Respiratory worsening followed by death from respiratory distress, seven days after the decision.

TABLE 2: Timeline of the case.
ABVD: doxorubicin, bleomycin, vinblastine, and dacarbazine; ICE: ifosfamide, carboplatin, and etoposide; SpO2: oxygen saturation.

Discussion

In adults, acquired esophagotracheal fistulas are predominantly malignant in origin, with bronchial and esophageal cancers representing the most frequently reported etiologies [2]. By contrast, the association between Hodgkin lymphoma and an esophago-respiratory fistula remains extremely rare. In their literature review, Greven and Evans (1992) identified only 22 cases of esophageal fistulas associated with Hodgkin's disease, most occurring in the setting of active mediastinal disease [3]. Subsequent reports have confirmed this rarity, as illustrated by the cases described by Kassir et al., Westin et al., and Rigney et al. [4-6].

The pathophysiology of fistula formation in Hodgkin lymphoma remains incompletely understood and appears to involve several intertwined mechanisms. In our case, the heterogeneous, irregular thickening of the esophageal wall between D2 and D4, adjacent to the fistula, may reflect locoregional esophageal tumor infiltration [4]. Extrinsic lymph node compression or infiltration represents another possible mechanism: bulky mediastinal lymph node masses can compress the esophagus and trachea and progressively weaken their walls [4]. Tumor necrosis within large lymphomatous masses may further promote tissue destruction and the gradual formation of a fistulous tract. Finally, the contribution of chemotherapy should also be considered: rapid tumor shrinkage and treatment-induced necrosis could theoretically favor fistula formation. However, in the absence of comparative imaging documenting the status of the mediastinal masses before initiation of ICE chemotherapy, the relative contributions of lymphoma progression and chemotherapy-associated tissue necrosis to fistula formation cannot be established. The presence of progressive lymphoma and necrotic mediastinal masses provides a plausible mechanism for fistula development; however, a contribution from treatment-related tissue injury cannot be excluded [1,5].

The most suggestive clinical feature is immediate postprandial cough upon ingestion of liquids and semi-

liquids, without associated dysphagia or odynophagia. This presentation corresponds to Ono's sign and reflects the passage of esophageal content into the tracheobronchial tree [7]. Ono's sign is highly suggestive but not constant; it should be kept in mind that the absence of dysphagia does not rule out an esophago-respiratory fistula [1,7]. In our patient, the respiratory impairment was multifactorial, related to pulmonary parenchymal involvement and bilateral pleural effusions, with a possible contribution from aspiration through the fistula, which could not be confirmed.

Although esophagography and endoscopy (esophago-bronchoscopy) allow direct visualization of the fistulous tract and endoluminal assessment for possible stenting [2], chest CT occupies a central place [8]. It directly visualizes the esophagotracheal communication and reveals indirect signs such as esophageal wall thickening, infiltration of the mediastinal fat, and necrotic lymphadenopathy. In our case, CT allowed direct identification of the esophagotracheal communication in a setting where endoscopic exploration was not feasible because of respiratory instability [8].

Management aims to occlude the fistula (covered esophageal stent or double esophagotracheal stenting), treat infection, and control the underlying disease [2]. In Hodgkin lymphoma, chemotherapy may allow spontaneous or secondary closure of the fistula, provided tumor control is achieved, as demonstrated by Westin et al. and Rigney et al. [5,6]. Nevertheless, the precarious respiratory status and severe malnutrition (BMI = 16 kg/m²) of our patient rendered any endoscopic or anesthetic procedure high-risk, illustrating the complexity of the management, which should be discussed within a multidisciplinary team [9].

Conclusions

Esophagotracheal fistula is an exceptional yet potentially fatal complication of Hodgkin lymphoma. Swallowing-induced cough, particularly upon ingestion of liquids, should raise suspicion of an esophago-respiratory fistula, even in the absence of dysphagia. Chest CT plays a major diagnostic role by allowing direct visualization of the fistulous communication and assessment of its complications, particularly when endoscopic exploration is not feasible. Management should be prompt and multidisciplinary. In our patient, management consisted of supportive measures, including nil per os status, parenteral nutrition, oxygen therapy, and empiric antibiotic treatment. Endoscopic intervention and stent placement were not feasible because of respiratory instability and poor general condition. Despite supportive care, the patient died on day 10 of hospitalization. This fatal outcome should be interpreted in the context of advanced progressive malignancy, severe malnutrition, and significant respiratory compromise, rather than as an independent estimate of the prognosis of esophagotracheal fistula in Hodgkin lymphoma. Nevertheless, it illustrates the seriousness of this complication and the importance of early recognition.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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