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CASE REPORT

# Simultaneous Lymphatic and Alveolar Leak: High-Output Non-Traumatic Chylothorax with Bilateral Fluidopneumothorax and Pneumomediastinum in a 27-Year-Old Woman Treated by Thoracoscopic Duct Ligation and Mechanical Pleurodesis

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**ABSTRACT**

**Background:** Chylothorax accounts for a small minority of pleural effusions, and the non-traumatic form is rarer still, usually indolent and usually low output. The simultaneous occurrence of high-output chylothorax and a spontaneous air-leak syndrome in an immunocompetent young adult has not been described.

**Objective:** To report such a case, to quantify the metabolic and immunological cost of the leak, and to argue that a single mechanical trigger can breach the alveolar and the lymphatic conduits together.

**Case presentation:** A 27-year-old woman with no trauma, thoracic surgery or malignancy developed progressive dyspnoea and recurrent massive pleural effusion requiring three thoracenteses, approximately 5,000 mL in 11 days. Tube thoracostomy then drained about 2,500 mL of serosanguineous, milky-pink fluid on the first day. Pleural fluid triglycerides were 1,774 mg/dL with cholesterol 130 mg/dL, a ratio of 13.6, lymphocyte predominance and a positive Rivalta test. Computed tomography showed bilateral fluidopneumothorax, pneumomediastinum, extensive subcutaneous emphysema and bilateral pneumonia, although lung sliding had been preserved bilaterally on admission ultrasonography. Lymphopenia of 0.63 $\times 10^9$/L, albumin of 2.20 g/dL, rising haemoglobin and deterioration to pH 7.33 with $PaO_2$ 52 mmHg accompanied a daily loss of approximately 44 g of triglyceride. Thoracoscopic duct ligation with mechanical pleurodesis reduced drainage to 180–250 mL/day and the patient was discharged on day 28.

**Conclusion:** High output does not imply trauma, and a non-traumatic cause does not imply an indolent course. Failure of conservative treatment is better defined by the metabolic and immunological ledger than by drainage volume alone.

## 1. Introduction

Chylothorax is the accumulation of chyle in the pleural space following disruption or obstruction of the thoracic duct or of one of its tributaries. It is an uncommon cause of pleural effusion, and the non-traumatic variety is uncommon even within that small group. Contemporary data make the point starkly. In a retrospective cohort of 40 consecutive patients with non-traumatic chylothorax managed at a tertiary referral hospital between 2020 and 2024, the median age was 61 years, cancer accounted for 67.5 % of cases, and mortality at 90 days and one year was 37.5 % and 55 % respectively; the highest one-year mortality, 83.3 %, was seen in those with solid-organ malignancy.[1] Non-traumatic chylothorax is therefore, in the modern literature, largely a disease of older adults with cancer and a poor prognosis. A 27-year-old woman with no malignancy, no thoracic operation and no trauma sits outside that description on every axis, and the reported cases that do resemble her are individually rare: recurrent bilateral idiopathic disease refractory to parenteral nutrition, octreotide and duct embolisation,[2] idiopathic bilateral disease that failed lymphaticovenular anastomosis and ended fatally three years later,[3] chylothorax as the presenting feature of systemic lupus erythematosus,[4] of cancer of unknown primary,[5] of follicular lymphoma,[6] and as a post-acute sequela of SARS-CoV-2 infection.[7]

Two heuristics dominate the bedside approach to such a patient, and both of them failed here. The first is that a very high output points to a traumatic or postoperative cause, because it is the surgical series that supply almost all the high-volume cases; the second is that a non-traumatic mechanism produces a slow, indolent accumulation with indistinct symptoms. Neither is a rule. Published thresholds are also less firm than they appear: outputs above 1,000 mL/day are conventionally described as high output in adults, yet a recent cohort of chylothorax after minimally invasive lung resection used 500 mL/day to separate high- from low-volume disease within a stepwise protocol,[8] and a review of 2,942 pulmonary resections concluded explicitly that the criteria for surgical treatment of chylothorax are not consistent between centres.[9] The point is made most directly by a 17-year retrospective cohort of consecutive patients who developed postoperative chylothorax after oesophageal or lung resection, whose authors state plainly that the optimal therapeutic recommendations are not well defined and rest on retrospective series, and who set out to build a rational algorithm from their own data for that reason.[10]

The second trap is analytical rather than conceptual. Chyle is not reliably milky. Lipoprotein electrophoresis with demonstration of chylomicrons remains the reference standard for a chylous effusion, and the triglyceride threshold used in its place varies between studies; when the appearance of the fluid is not diagnostic, only biochemistry resolves the question.[11] A fluid that is serous or serosanguineous rather than white is therefore repeatedly misassigned to another category, and reports continue to

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describe the same pitfall from the opposite direction, in effusions that look milky but are not chylous at all.[12] The two conditions most often confused with chylothorax have entirely different implications: pseudochylothorax, in which cholesterol is high and triglyceride low in a long-standing loculated collection,[13] and the leakage of enteral feed into the pleural space, which can be identified only by looking for formula-specific markers.[14]

A third element of this case belongs to a separate literature. Pneumomediastinum, subcutaneous emphysema and pneumothorax in the absence of trauma are explained by the Macklin effect: rupture of marginal alveoli, with air then tracking centrally along the bronchovascular sheaths. A systematic review of seven studies and 979 patients found the Macklin sign in 4–22 % of patients with coronavirus disease 2019, associated with barotrauma in 89.8 % of cases and preceding barotrauma by three to eight days in 94.2 %.[15] Its clinical counterpart is well described in young adults: a single-centre series of 87 patients with pneumomediastinum,[16] a retrospective study of 40 patients in which the Macklin effect was demonstrable in 38,[17] a woman in her mid-twenties who developed pneumomediastinum with extensive subcutaneous emphysema after nothing more than an upper respiratory tract infection,[18] and a young man in whom pneumomediastinum, pneumopericardium and bilateral pneumothoraces coexisted and still resolved without intervention.[19] The same mechanical vocabulary — sustained Valsalva, violent expiratory effort — recurs in the reports of chylothorax without trauma: a bilateral chylothorax attributed to a tonic-clonic seizure six days earlier,[20] and a two-year-old boy in whom a bout of heavy coughing was the only identifiable cause.[21] That the same mechanical events appear in both literatures is the observation on which this report turns.

What has not been reported, to the best of our knowledge, is the coincidence of these two processes in one immunocompetent young adult with no trauma and no thoracic operation: a high-output chylothorax of approximately 2,500 mL/day whose fluid was serosanguineous rather than milky, together with bilateral fluidopneumothorax, pneumomediastinum and extensive subcutaneous emphysema. The aim of this report is threefold. First, to document that combination in detail and to show how the diagnosis was reached from biochemistry alone when the appearance of the fluid pointed away from it. Second, to quantify what such an output actually costs the patient in lipid, energy, protein and lymphocytes, and to argue that this ledger, rather than the volume in the bottle, is what should define failure of conservative treatment. Third, to advance the hypothesis that one mechanical trigger — paroxysmal cough in the setting of bilateral pneumonia — can breach the alveolar and the lymphatic conduits in parallel, and that the sequence of imaging in this patient supports rather than contradicts that reading.

The claim of novelty rests on a documented search rather than on impression. PubMed was searched through the NCBI E-utilities on 30 July 2026, with citation metadata checked against the Crossref REST API, using terms in four domains combined with each other and with "chylothorax": non-traumatic, spontaneous, idiopathic and high-output chylothorax; thoracic duct ligation, pleurodesis, lymphangiography and duct embolisation; pneumomediastinum, subcutaneous emphysema, Macklin effect and iatrogenic pneumothorax; and the metabolic and immunological consequences of chyle loss. The search was limited to human studies published in English between 2021 and 2026. On that basis we identified no report in which high-output non-traumatic chylothorax and a spontaneous air-leak syndrome occurred together in an immunocompetent adult without trauma or thoracic surgery. The restriction to English materially qualifies the claim, because a substantial case-report literature on chylothorax is published in Chinese, Japanese and Spanish and was not searched.

## 2. Case Presentation

A 27-year-old woman with no previous respiratory disease presented with progressive shortness of breath that began on 6 October 2025. The breathlessness was continuous, present both at rest and on exertion, and only partially relieved by supplemental oxygen. She reported positional improvement when lying on her left side, and an occasional cough productive of scant white sputum. There was no haemoptysis, no pleuritic chest pain, no fever, no prolonged cough, no weight loss and no night sweats, and no recent history suggestive of acute infection. She had never undergone thoracic surgery, had sustained no chest trauma, and was taking no regular medication. The record documents no chest trauma, no thoracic operation and no regular medication; it is silent on smoking, on inhalational recreational drug use, on recent vomiting or retching, and on features of connective tissue disease, and none of these was recorded either as present or as absent. The complete sequence of admissions, procedures and physiological milestones described below is set out in Figure 1, which should be read alongside this narrative.

As shown in Figure 1, she was first admitted to a district hospital from 6 to 12 October 2025, during which a thoracentesis was performed and approximately 1 L of pleural fluid was evacuated. Symptoms persisted and she was readmitted to the same hospital from 13 to 17 October for recurrent dyspnoea, undergoing two further thoracenteses that drained about 2 L each. Because the fluid re-accumulated rapidly and respiratory symptoms did not settle, she was referred to Prof. dr. I.G.N.G. Ngoerah General Hospital, where she was admitted from 17 October to 2 November 2025 and a chest tube with water-seal drainage was inserted. In the 11 days before referral, therefore, approximately 5,000 mL of pleural fluid had been formally removed on three occasions, as summarised in the events above the axis in Figure 1.

On arrival at the tertiary centre she was alert and haemodynamically stable with a Glasgow Coma Scale score of 15. The blood pressure was 148/80 mmHg, the pulse 98 beats/min, the respiratory rate 22 breaths/min and the axillary temperature 36.3 °C. Oxygen saturation was 88 % on room air, rising to 96 % with oxygen delivered by face mask at 5 L/min. There was no pallor, jaundice or cervical lymphadenopathy. Chest expansion was symmetrical without intercostal retraction. Vocal fremitus was reduced over the lower lung fields, percussion was dull in the corresponding areas, and auscultation revealed decreased vesicular breath sounds with rhonchi and no wheeze. Cardiac examination was normal. The abdomen was non-distended with normal bowel sounds and mild epigastric tenderness, without guarding or organomegaly. The extremities were warm and well perfused, with normal capillary refill and no oedema. There was no peripheral lymphoedema and no clinical ascites.

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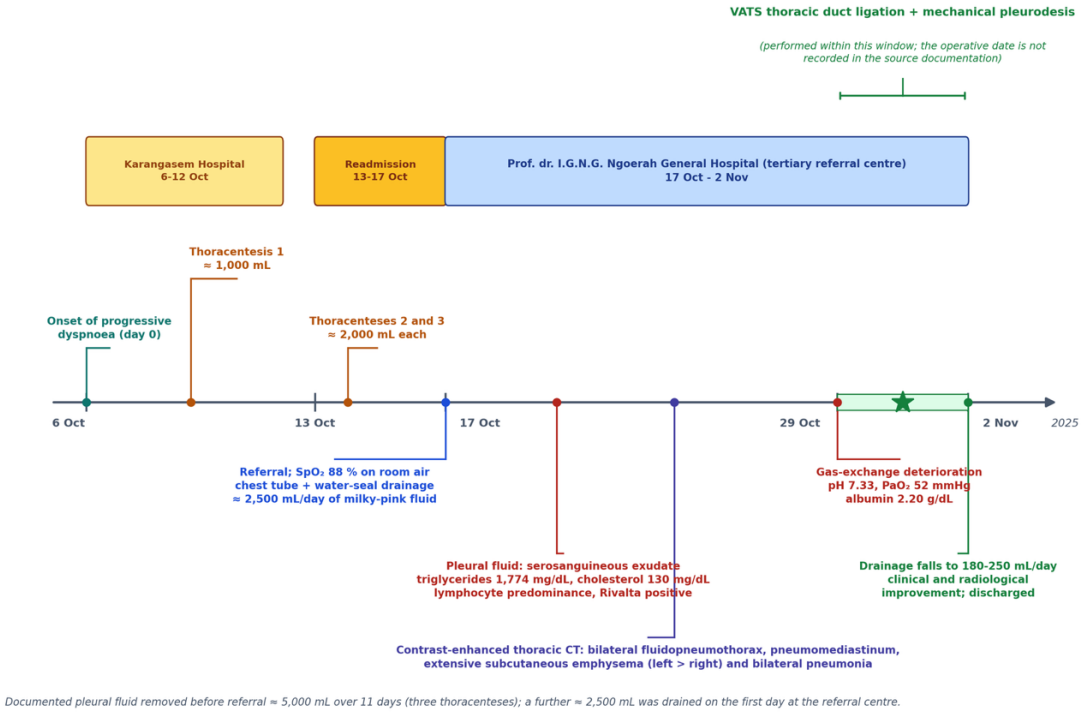


**Figure 1.** Clinical timeline of the illness episode. Day 0 is 6 October 2025, the day progressive dyspnoea began. The amber bands mark the two admissions to the referring district hospital and the blue band the 17-day admission to the tertiary referral centre. Events above the axis are the pleural procedures performed before referral; events below the axis are the diagnostic and physiological milestones of the tertiary admission. The green bracket marks the window within which video-assisted thoracoscopic thoracic duct ligation and mechanical pleurodesis were performed; the operative date itself is not recorded in the source documentation and has therefore not been assigned. Approximately 5,000 mL of pleural fluid was formally removed in the 11 days before referral, and a further 2,500 mL on the first day after tube thoracostomy.

Bedside thoracic ultrasonography performed on arrival confirmed predominantly right-sided pleural fluid, and — a point of some importance for the sequence of events discussed later — lung sliding was preserved bilaterally at that moment, which argues strongly against a large pneumothorax on either side at the time of that examination. The examination was a single bedside study performed to assess pleural fluid rather than to exclude air, and it is operator- and window-dependent, so a small or loculated anterior collection cannot be excluded on that basis. Laboratory testing showed a leucocytosis of 16.53 $\times 10^9$/L falling to 13.69 $\times 10^9$/L over the course of the admission, with neutrophil predominance and a reduced absolute lymphocyte count. Haemoglobin and platelet counts were reported as remaining normal, the platelet count moving from 287 to 253 $\times 10^9$/L and remaining within the reference interval on both occasions. Arterial blood gas analysis initially showed adequate oxygenation, followed later by hypoxaemia. Serum biochemistry demonstrated mildly raised transaminases, normal renal function and hypoalbuminaemia. The serial values, the reference intervals printed on the laboratory report and the derived neutrophil-to-lymphocyte ratio are set out in Table 1.

Two features of Table 1 deserve emphasis before the pleural fluid results are considered. The first is the behaviour of the lymphocyte count. As detailed in Table 1, the absolute lymphocyte count was 0.63 $\times 10^9$/L on 17 October and 0.60 $\times 10^9$/L twelve days later, so that while the total leucocyte count and the neutrophil count both fell towards their reference intervals, the lymphopenia did not correct at all; the derived neutrophil-to-lymphocyte ratio fell only from 24.7 to 21.5. The second is the haemoglobin. The value rose from 14.9 to 17.6 g/dL, an increase of 2.7 g/dL or 18 %, over a period during which the patient was losing litres of protein-rich fluid each day. As the footnote to Table 1 records, the reference interval printed on the report, 13.5–17.5 g/dL, is the interval for adult men; measured against the conventional interval for adult women the final value is frankly raised, and it is best read as haemoconcentration rather than as a normal result. These two trajectories, together with the arterial blood gas and biochemical values, are displayed graphically in Figure 2.

Figure 2A makes the dissociation between the myeloid and lymphoid compartments visible at a glance: the neutrophil bars fall towards their upper reference limit while the lymphocyte bars remain far below the lower limit on both occasions. Figure 2B plots the arterial blood gas values against their own reference intervals and shows the reversal of the respiratory picture — an initial respiratory alkalosis, pH 7.46 with a $PaCO_2$ of 41 mmHg and a supranormal $PaO_2$ of 104 mmHg on 17 October, replaced twelve days later by acidaemia with a pH of 7.33, a $PaCO_2$ of 46 mmHg and a $PaO_2$ of 52 mmHg on 29 October. Figure 2C shows the albumin of 2.20 g/dL lying far to the left of its reference interval while the haemoglobin marker has moved to the right of the female interval, and also displays the mildly raised transaminases, aspartate aminotransferase 39 U/L and alanine aminotransferase 84 U/L. Figure 2D, discussed further below, sets the drainage volumes against the thresholds conventionally used to define high-output disease.

**Table 1.** Serial laboratory findings during the tertiary-centre admission, with the reference intervals printed on the laboratory report.

| Parameter | 17 Oct 2025 | 29 Oct 2025 | Reference interval |
|---|---|---|---|
| White blood cell count (×10⁹/L) | 16.53 | 13.69 | 4.1–11.0 |
| Neutrophils (×10⁹/L) | 15.55 | 12.87 | 2.5–7.5 |
| Lymphocytes (×10⁹/L) | 0.63 | 0.60 | 1.0–4.0 |
| Neutrophil-to-lymphocyte ratio* | 24.7 | 21.5 | Not reported* |
| Haemoglobin (g/dL) | 14.9 | 17.6 | 13.5–17.5† |
| Platelet count (×10⁹/L) | 287 | 253 | 150–440 |
| Arterial pH | 7.46 | 7.33 | 7.35–7.45 |
| $PaCO_2$ (mmHg) | 41 | 46 | 35–45 |
| $PaO_2$ (mmHg) | 104 | 52 | Not stated‡ |
| Aspartate aminotransferase (U/L) | 39 | Not repeated | <34 |
| Alanine aminotransferase (U/L) | 84 | Not repeated | <35 |
| Serum albumin (g/dL) | 2.20 | Not repeated | 3.5–5.0 |
| Serum creatinine and urea | Within reference interval | Within reference interval | Reported as normal |

***Notes:*** Values printed in bold red lie outside the reference interval. *The neutrophil-to-lymphocyte ratio was derived by the authors from the reported absolute counts (15.55 ÷ 0.63 and 12.87 ÷ 0.60) and was not issued by the laboratory. †The interval printed on the report, 13.5–17.5 g/dL, is the interval for adult men; measured against the conventional interval for adult women (12.0–15.5 g/dL) the value of 17.6 g/dL on 29 October represents frank haemoconcentration rather than a normal result. ‡The laboratory report did not state a reference interval for $PaO_2$, and the fraction of inspired oxygen accompanying each arterial sample was not recorded. Renal function was reported as normal without numerical values, and these are therefore not tabulated.

Analysis of the pleural fluid provided the diagnosis. Two descriptions of the same fluid appear in the record and they do not agree: the formal laboratory description was of a serosanguineous exudate, whereas the fluid drained at the bedside was described as milky-pink. The discrepancy is not an inconsistency to be smoothed over but the very diagnostic pitfall this report concerns, and both descriptions are reported here as they were recorded. A total of roughly 2,500 mL was evacuated on the first day through the water-seal drain. The triglyceride concentration was markedly raised at 1,774 mg/dL, equivalent to approximately 20 mmol/L, with a total cholesterol of 130 mg/dL, and cytological examination showed a predominance of lymphocytes. The Rivalta test was positive; this is a qualitative bedside method in which a drop of pleural fluid is added to dilute acetic acid, and a persisting precipitate indicates a protein-rich exudate. Pleural fluid protein, lactate dehydrogenase and glucose were not measured and no paired serum values are available, so Light's criteria could not be applied and the exudative classification rests on this qualitative test alone. Microbiological evaluation, including blood cultures, sputum cultures and molecular testing for Mycobacterium tuberculosis, was entirely negative. Pulmonary function testing revealed a severe restrictive ventilatory impairment without airflow obstruction. These results, the drainage volumes before and after operation, and — equally importantly — the investigations that were not undertaken, are presented together in Table 2.

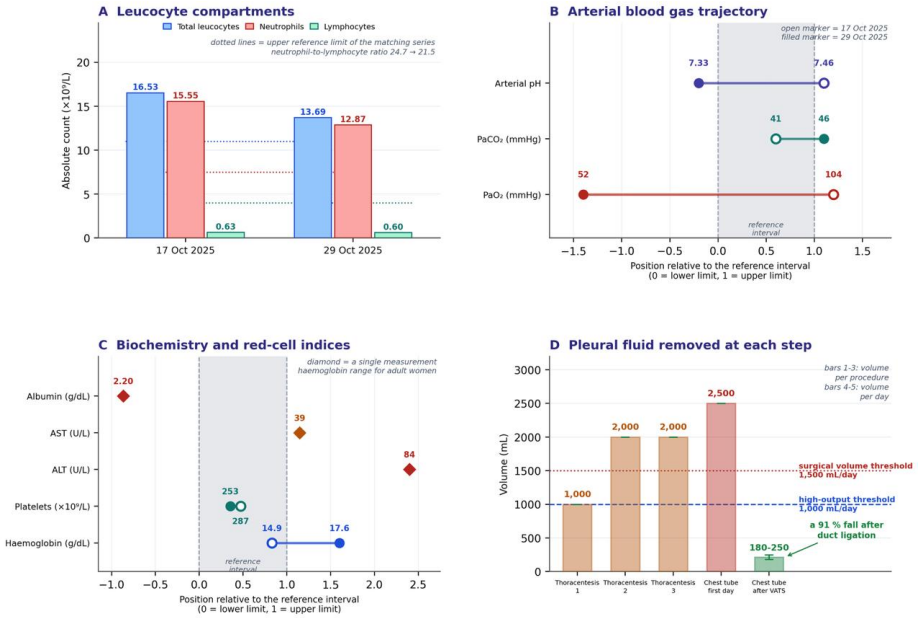


**Figure 2.** Serial physiology and drainage volumes. Normalisation in panels B and C uses the reference intervals printed on this laboratory's report, with the single exception of the haemoglobin interval, for which the interval for adult women has been substituted and is flagged as such; reference intervals differ between laboratories and these panels should not be transferred directly to another setting. (A) Absolute leucocyte counts on 17 and 29 October; the dotted lines mark the upper reference limit of the series of the same colour. Neutrophilia coexisted with a profound lymphopenia that did not correct, so that the neutrophil-to-lymphocyte ratio remained above 21 throughout. (B) Arterial blood gas values plotted against their own reference intervals, normalised so that 0 is the lower limit and 1 the upper limit of each interval; open markers are the values of 17 October and filled markers those of 29 October. The initial respiratory alkalosis with supranormal oxygenation gave way to combined hypoxaemia and carbon dioxide retention. (C) Biochemistry and red-cell indices on the same normalised scale; albumin fell far below the lower reference limit while haemoglobin rose above the interval for adult women. (D) Volume of pleural fluid removed at each step. Bars 1 to 3 are volumes per procedure and bars 4 and 5 volumes per day. The two horizontal lines mark the conventionally quoted high-output threshold of 1,000 mL/day and the volume threshold of 1,500 mL/day at which operation is often advised; neither is a consensus definition.

**Table 2.** Pleural fluid analysis, microbiological and physiological investigations, drainage volumes, and the investigations that were not performed.

| Domain | Parameter | Result | Interpretation |
|---|---|---|---|
| Pleural fluid | Appearance | Serosanguineous; described as milky-pink at the bedside | Not classically milky — a recognised source of misdiagnosis[11] |
| | Effusion type | Exudative (Rivalta test only) | Protein, lactate dehydrogenase and glucose were not measured, so Light's criteria could not be applied |
| | Triglycerides (mg/dL) | 1,774 | 16.1 times the 110 mg/dL diagnostic threshold[8] |
| | Total cholesterol (mg/dL) | 130 | Well below the concentrations reported in pseudochylothorax[13] |
| | Triglyceride-to-cholesterol ratio* | 13.6 | Greater than 1, consistent with chyle[12] |
| | Predominant cell type | Lymphocytes | Expected in chyle[22] |
| | Rivalta test | Positive | Exudate |
| Microbiology | Blood culture | No growth | No bacteraemia documented |
| | Sputum culture | No growth | No pathogen isolated |
| | Molecular test for Mycobacterium tuberculosis | Negative | Tuberculous pleuritis not supported |
| Pulmonary function | Ventilatory pattern | Severe restrictive impairment | Consistent with a large effusion and loss of lung volume |
| | Airflow obstruction | Not present | No coexisting obstructive defect |
| Bedside imaging | Thoracic ultrasonography on arrival | Predominantly right-sided pleural fluid; lung sliding preserved bilaterally | Argues strongly against a large pneumothorax at that moment; a single operator-dependent study cannot exclude a small anterior collection |
| Drainage | Chest-tube output on the first day (mL/day) | ≈ 2,500 | High output by any published definition[8] |
| | Chest-tube output after operation (mL/day) | 180–250 | A fall of approximately 91 % |
| Not performed | Pleural fluid flow cytometry and lymphocyte immunophenotyping | Not performed | A lymphoproliferative cause was therefore not formally excluded[6,23] |
| | Lipoprotein electrophoresis or chylomicron detection | Not performed | Not required — the triglyceride concentration was unequivocal[11] |
| | Paired serum triglyceride and cholesterol | Not performed | Pleural-fluid-to-serum ratios could not be computed[12] |
| | Pleural fluid protein, lactate dehydrogenase and glucose | Not performed | Light's criteria could not be applied |
| | Parietal pleural biopsy at thoracoscopy | Not recorded | Tissue was obtainable at no additional risk and would have addressed the lymphoma question[6] |
| | Lymphoscintigraphy, lymphangiography or magnetic resonance ductography | Not performed | The site of leakage was never localised, as in most reported series[1] |

***Notes:*** *Derived by the authors as 1,774 ÷ 130. The timing of pulmonary function testing in relation to drainage was not recorded in the source documentation, and the severity of the restrictive defect should therefore be read as descriptive rather than as a measurement at a defined point in the illness. Values printed in bold red are abnormal or exceed a diagnostic threshold.

Table 2 records that the triglyceride-to-cholesterol ratio in the pleural fluid, derived by the authors as 1,774 ÷ 130, was 13.6, and that the triglyceride concentration was 16.1 times the 110 mg/dL threshold conventionally used to define a chylous effusion. Taken with the lymphocyte predominance, these values establish a high-output chylothorax without recourse to lipoprotein electrophoresis. The lower block of Table 2 lists what was not done: no flow cytometry or lymphocyte immunophenotyping of the pleural fluid, no paired serum lipids and therefore no pleural-fluid-to-serum ratios, and no lymphatic imaging of any kind. The consequences of those omissions are considered in the Discussion.

Serial chest radiographs showed persistent pleural effusion with reduced lung expansion. A representative radiograph from the tertiary admission, with the right-sided chest drain in place, is shown in Figure 3.

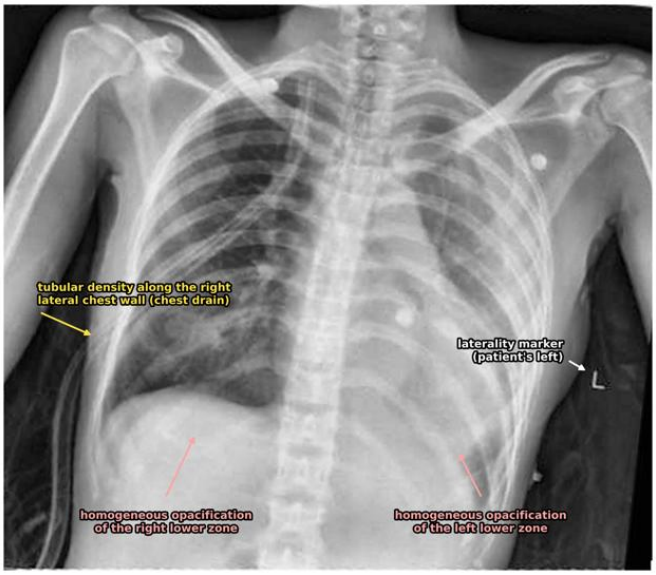


**Figure 3.** Anteroposterior chest radiograph obtained during the tertiary admission, with the right-sided chest drain in place. The image has been rotated to correct the tilt of the original capture, cropped to the radiographic field and contrast-normalised; no anatomical content has been altered and no patient identifier was present on the source image. There is homogeneous opacification of both lower zones with loss of the normal hemidiaphragmatic outlines and generally reduced lung volumes, the appearances reported at the time as persistent pleural effusion with reduced lung expansion. A tubular density runs along the right lateral chest wall in the expected position of the intercostal drain. The metallic marker on the patient's left confirms the orientation of the film.

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As shown in Figure 3, there is homogeneous opacification of both lower zones with loss of the normal hemidiaphragmatic outlines and generally reduced lung volumes, and a tubular density runs along the right lateral chest wall in the expected position of the intercostal drain. Contrast-enhanced computed tomography of the thorax was then performed and demonstrated left-sided fluidopneumothorax — the coexistence of fluid and air within the same pleural space — together with right pneumothorax, pneumomediastinum, bilateral pneumonia and extensive subcutaneous emphysema involving both hemithoraces, the left more than the right. The right-sided chest tube was visualised with its distal tip directed cranially. The two axial levels available to us are reproduced in Figure 4.

Figure 4A shows aerated right lung with visible vessels alongside an avascular air collection lying medially against the mediastinum, while the left hemithorax is uniformly opacified; gas outlines the chest wall on both sides and lifts the superficial soft tissues away from the thoracic cage. Figure 4B, obtained at a level at which almost the whole cross-section of the thorax is dense and non-aerated, shows a paramediastinal air collection and allows the displaced superficial fascial plane to be traced well outside the expected chest-wall contour. The juxtaposition of the two levels shown in Figure 4A and Figure 4B, and the discrepancy between the right-predominant fluid seen on admission ultrasonography and the left-predominant fluidopneumothorax seen on this later study, are examined in the Discussion.

Conservative management during the tertiary admission consisted of continuous water-seal pleural drainage and supplemental oxygen. The source documentation records no dietary intervention, no medium-chain-triglyceride or fat-restricted diet, no parenteral nutrition and no somatostatin analogue, and it should be assumed that these measures were not used rather than that they were used and not recorded. Because high-volume drainage persisted and gas exchange deteriorated as documented in Table 1 and Figure 2B, definitive intervention was undertaken. Video-assisted thoracoscopic surgery was performed with ligation of the thoracic duct, followed by mechanical pleurodesis. The operative documentation available to us does not record the level at which the duct was ligated, the method of occlusion, whether any intraoperative technique was used to identify the duct, or whether a parietal pleural biopsy was taken; no biopsy result appears anywhere in the record. The operative date is not recorded in the source documentation and has therefore not been assigned in Figure 1, where, as shown in the green bracket at the top of the figure, the procedure is marked only by the window within which it must have taken place. After operation the patient improved substantially: drainage from the water-seal system fell to 180–250 mL/day of serosanguineous fluid, a reduction of approximately 91 % from the 2,500 mL/day recorded on the day the tube was inserted, as plotted in the last two bars of Figure 2D. Clinical and radiological improvement followed, and she was discharged on 2 November 2025, 28 days after the onset of dyspnoea. No recurrence was documented during the remainder of the admission. Follow-up ends at discharge: no post-discharge imaging, drainage record or clinic review is available to us, so the follow-up interval beyond discharge is zero days.

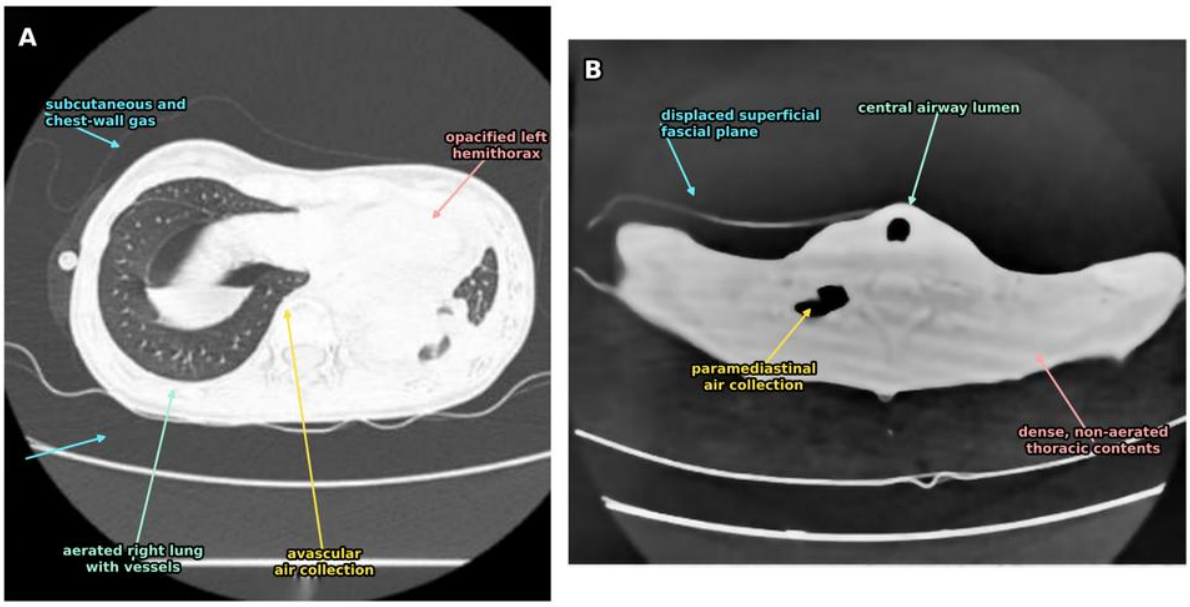


**Figure 4.** Axial contrast-enhanced computed tomography of the thorax at two levels, displayed in a lung window. (A) An image exported from the reporting workstation. The right hemithorax contains aerated lung with visible vessels, and an avascular air collection lies medially against the mediastinum; the left hemithorax is uniformly opacified. Gas outlines the chest wall on both sides, lifting the superficial soft tissues away from the thoracic cage. (B) A second level, photographed from the reporting monitor, which is the only record of this level available to the authors; the image has been converted to greyscale, corrected for the illumination gradient of the screen and cleaned of the pointer artefact, without altering anatomical content. At this level almost the whole cross-section of the thorax is dense and non-aerated, a paramediastinal air collection is present, and the displaced superficial fascial plane can be traced well outside the expected chest-wall contour. The formal report described left-sided fluidopneumothorax, right pneumothorax, pneumomediastinum, bilateral pneumonia and extensive subcutaneous emphysema involving both hemithoraces, left more than right.

## 3. Discussion

### *Confirming chyle when the fluid is not milky*

The single most consequential decision in this admission was to send the pleural fluid for lipid analysis at all. Everything about its appearance argued against chylothorax. It was recorded as a serosanguineous exudate, not as the white or opalescent fluid that the word chyle evokes, and the bedside description of milky-pink captures a fluid whose colour was being driven as much by blood as by lipid. The literature is unambiguous that this is where the diagnosis is lost. Because a macroscopic assessment of the fluid is insufficient,

lipoprotein electrophoresis with demonstration of chylomicrons remains the reference standard for chylous effusion, and the triglyceride threshold used as a surrogate varies between studies; a chemiluminescent assay for apolipoprotein B48, the structural apolipoprotein of the chylomicron, has recently been shown to perform at least as well as triglyceride measurement, with threshold values of 2.45, 0.25 and 19.00 µg/mL for the maximal Youden index, for a sensitivity above 95 % and for a specificity above 95 % respectively, and in that study both triglyceride concentration and the triglyceride-to-cholesterol ratio were significantly higher in chylous effusions.[11] The mirror-image error is equally well documented: a milky yellow effusion after lung cancer surgery in which the diagnosis rested on a pleural-to-serum triglyceride ratio of 3.62, a pleural-to-serum cholesterol ratio of 0.43, a cholesterol-to-triglyceride ratio of 0.32 and positive Sudan III staining, and whose authors concluded that integrating these ratios reduces false-negative results in non-milky effusions.[12]

Against that background the biochemistry in this patient was not marginal but emphatic. As detailed in Table 2, the triglyceride concentration of 1,774 mg/dL is 16.1 times the 110 mg/dL threshold, and the triglyceride-to-cholesterol ratio of 13.6 is more than an order of magnitude above unity. The cholesterol of 130 mg/dL is the value that closes off the main alternative. In a reported pseudochylothorax complicating treated pleuropulmonary tuberculosis, the cholesterol was 300 mg/dL with triglycerides of only 90 mg/dL in a thick, chocolate-coloured fluid that had been present intermittently for more than a decade;[13] our patient's numbers are the exact inverse of that pattern, and her illness had lasted eleven days. Lymphocyte predominance on cytology adds independent support, because chyle is a lymphocyte-rich fluid: in a study of 18 neonates with chylothorax and paired samples of blood and chyle, leucocyte, lymphocyte and T-cell counts were all significantly higher in chyle than in blood.[22] The one differential that biochemistry alone cannot exclude, the accidental instillation of an enteral feed into the pleural space, was excluded on clinical grounds, since the patient had no feeding tube; where that possibility is live, gas chromatography-mass spectrometry for a formula-specific marker such as tricaprylin is required, as an autopsy series has shown.[14] The full differential, together with the pleural fluid profile expected of each entity and the status of each possibility in this patient, is set out in Table 3.

**Table 3.** Differential diagnosis of a recurrent, rapidly re-accumulating lipid-rich pleural effusion in a young adult with no history of trauma or thoracic surgery, and the status of each possibility in this patient.

| Diagnostic possibility | Expected pleural fluid profile | Features sought clinically | Status in this patient |
|---|---|---|---|
| True chylothorax from disruption of the thoracic duct or a tributary | Triglycerides >110 mg/dL, triglyceride-to-cholesterol ratio >1, chylomicrons demonstrable, lymphocyte predominance | Milky or serosanguineous fluid, rapid re-accumulation after drainage | Confirmed — triglycerides 1,774 mg/dL, ratio 13.6, lymphocyte predominance[8,12] |
| Pseudochylothorax (cholesterol effusion) | Cholesterol markedly raised, triglycerides low, cholesterol crystals, long-standing loculated collection | Previous tuberculous or rheumatoid pleuritis, pleural thickening | Excluded — cholesterol only 130 mg/dL with triglycerides of 1,774 mg/dL and an acute course[13] |
| Malignant chylothorax, most often lymphoma | Chylous fluid, sometimes with atypical lymphoid cells | Lymphadenopathy, constitutional symptoms, flow cytometry, cross-sectional imaging | Not formally excluded — no lymphadenopathy or constitutional symptoms, but flow cytometry was not performed[1,5] |
| Parapneumonic effusion or empyema | Neutrophil predominance, low pH and glucose, positive culture | Fever, purulent fluid, systemic sepsis | Not the dominant process — lymphocyte predominance and sterile cultures, although bilateral pneumonia coexisted on computed tomography |
| Tuberculous pleuritis | Lymphocyte-rich exudate, high adenosine deaminase, positive nucleic acid amplification test | Weight loss, night sweats, contact history | Excluded — molecular testing negative and no constitutional symptoms |
| Leakage of enteral feed into the pleural space | Milky fluid containing formula-specific markers such as tricaprylin | Recent nasogastric or nasojejunal tube placement | Excluded — the patient had no enteral feeding tube[14] |
| Chylothorax complicating a systemic autoimmune disease such as systemic lupus erythematosus | Chylous fluid with serositis of other membranes | Rash, arthritis, cytopenias, autoantibody profile | Not suggested clinically; autoantibody testing is not documented in the record[4] |
| Congenital or acquired lymphatic malformation | Chylous fluid with abnormal lymphatic channels on lymphatic imaging | Lymphoedema, chylous ascites, lytic bone lesions | Not suggested — no lymphoedema or ascites; lymphatic imaging was not performed[3] |
| Drug-induced chylothorax | Chylous fluid with a clear temporal relationship to a culprit agent | Exposure to tyrosine-kinase inhibitors or other implicated drugs | Excluded — no relevant drug exposure[1] |
| Idiopathic chylothorax after exclusion of the above | Chylous fluid with no demonstrable cause | A complete negative work-up including lymphatic imaging | The default label if a mechanical cause is not accepted; reported idiopathic cases are often refractory and recurrent[2] |

***Notes:*** The middle two columns describe what would be expected of each diagnosis rather than what was found in this patient. Rows printed in bold red are the possibilities that remained live at the end of the admission.

As detailed in Table 3, and specifically in the two rows printed in bold red, two possibilities remained live when the patient was discharged. One is true chylothorax, which is established. The other is malignant chylothorax, which is not excluded, and that gap is discussed below. As detailed in Table 3, the remaining seven possibilities can be dismissed on the evidence available: the biochemistry excludes pseudochylothorax, the negative molecular test and the absence of constitutional symptoms make tuberculous pleuritis very unlikely, the lymphocyte predominance and sterile cultures argue against a primarily parapneumonic process, and there was no feeding tube, no relevant drug exposure, no lymphoedema and no clinical evidence of a systemic autoimmune disease.

### *Non-traumatic did not mean low output: quantifying the leak*

It is often stated that non-traumatic mechanisms produce a slow accumulation of chyle with indistinct symptoms, and that high-volume disease belongs to the postoperative and traumatic categories. In this patient the opposite held. Approximately 2,500 mL of fluid was evacuated on the first day after tube thoracostomy, and roughly 5,000 mL had already been removed by three thoracenteses over the preceding eleven days. As shown in Figure 2D, that daily figure sits two and a half times above the conventionally quoted 1,000 mL/day threshold for high-output disease and well above the 1,500 mL/day volume at which operation is often advised. It also sits five times above the 500 mL/day threshold that a recent cohort study used to separate high-

from low-volume chylothorax after minimally invasive lung resection.[8]

As shown in Figure 3, the radiograph gives the qualitative counterpart of those numbers: both lower zones are homogeneously opacified with the hemidiaphragmatic outlines lost, and the drain visible along the right lateral chest wall in Figure 3 was, on the day the image was taken, removing more fluid than most patients with a pleural effusion produce in a week. Volume, however, is a poor description of what such a leak does to a patient, and the more informative currency is the quantity of substrate lost. Table 4 sets out the arithmetic in full.

As detailed in Table 4, a triglyceride concentration of 1,774 mg/dL is 17.74 g/L, so that an output of 2,500 mL/day corresponds to the loss of approximately 44 g of triglyceride each day — on the assumption that the concentration measured on a single sample applied to the whole of that day's output, which is unlikely to be exactly true because chyle composition varies with enteral intake — an energy loss of about 400 kcal/day at the conventional 9 kcal per gram, together with roughly 3.3 g of cholesterol. Those figures are the reason the albumin of 2.20 g/dL and the lymphopenia of 0.63 $\times 10^9$/L in Table 1 are not incidental observations but the expected consequence of the drainage volume. They also put the pre-referral period into perspective: Table 4 shows a mean documented drainage of only about 455 mL/day over those eleven days, but this is a lower bound that counts only the fluid formally removed at three procedures and ignores everything that re-accumulated between them. The true leak rate before referral was almost certainly close to the 2,500 mL/day measured once continuous drainage was established, and the apparent escalation on transfer is an artefact of measurement rather than a change in the disease.

**Table 4.** Quantities derived by the authors from the reported clinical values, with the arithmetic shown in full.

| Derived quantity | Reported inputs | Derivation | Result |
|---|---|---|---|
| Pleural fluid triglyceride concentration in mass units | 1,774 mg/dL | 1,774 mg/dL × 10 = 17,740 mg/L | 17.74 g/L |
| Daily triglyceride loss at the recorded output | 2,500 mL/day and 17.74 g/L | 2.5 L × 17.74 g/L = 44.35 g | ≈ 44 g/day |
| Energy equivalent of that lipid loss | 44.35 g/day of triglyceride | 44.35 g × 9 kcal/g = 399 kcal | ≈ 400 kcal/day |
| Daily cholesterol loss at the recorded output | 130 mg/dL and 2,500 mL/day | 2.5 L × 1.30 g/L | ≈ 3.3 g/day |
| Triglyceride-to-cholesterol ratio in the pleural fluid | 1,774 and 130 mg/dL | 1,774 ÷ 130 | 13.6 |
| Multiple of the diagnostic triglyceride threshold | 1,774 and 110 mg/dL | 1,774 ÷ 110 | 16.1 × |
| Documented pleural fluid removed before referral | 1,000 mL, 2,000 mL and 2,000 mL | sum of the three thoracenteses | ≈ 5,000 mL |
| Mean documented drainage rate before referral | 5,000 mL over 11 days | 5,000 ÷ 11 | ≈ 455 mL/day (a lower bound only) |
| Duration of repeated pleural drainage before the documented deterioration | First thoracentesis between 6 and 12 October; deterioration 29 October | 29 October minus 12 October, and minus 6 October | 17–23 days |
| Reduction in drainage after thoracic duct ligation | 2,500 mL/day before, 180–250 mL/day after | (2,500 – 250) ÷ 2,500 and (2,500 – 180) ÷ 2,500 | 90–93 % |
| Neutrophil-to-lymphocyte ratio on arrival | 15.55 and 0.63 $\times 10^9$/L | 15.55 ÷ 0.63 | 24.7 |
| Neutrophil-to-lymphocyte ratio on 29 October | 12.87 and 0.60 $\times 10^9$/L | 12.87 ÷ 0.60 | 21.5 |
| Rise in haemoglobin over 12 days | 14.9 and 17.6 g/dL | 17.6 – 14.9 | +2.7 g/dL (+18 %) |
| Arithmetic bound on the $PaO_2/FiO_2$ ratio on 29 October | $PaO_2$ 52 mmHg; $FiO_2$ between 0.21 (room air) and 0.40 (5 L/min face mask) | 52 ÷ 0.40 and 52 ÷ 0.21 | between ≈ 130 and ≈ 250 — a bound, not a measurement |
| Interval from the onset of dyspnoea to the documented deterioration | 6 October to 29 October | day count | 23 days |
| Total duration of the illness episode | 6 October to 2 November | inclusive day count | 28 days |

***Notes:*** Every value in the final column was computed by the authors from the figures in Tables 1 and 2; none was issued by the laboratory. The energy equivalent uses the conventional value of 9 kcal per gram of fat, and both lipid rows assume that the concentration measured on a single sample applied to the whole of that day's output, which is unlikely to be exactly true because chyle composition varies with enteral intake. No $PaO_2/FiO_2$ ratio is asserted: the inspired oxygen fraction accompanying the arterial sample was not recorded, so the row gives the arithmetic bound between the room-air and face-mask limits, and gas exchange was severely impaired on either assumption. The mean drainage rate before referral is a lower bound because it counts only fluid that was formally removed and ignores the fluid that re-accumulated between procedures.

There is a physiological corollary in Table 1 that is easy to overlook. Losing litres of protein-rich fluid daily while the intravascular volume is only partially replaced produces haemoconcentration, and this is what the rise in haemoglobin from 14.9 to 17.6 g/dL, an increase of 18 % over twelve days as calculated in Table 4, most plausibly represents. Read naively against the male reference interval printed on the report, the final haemoglobin looks reassuringly normal. Read against the interval for adult women, and in the context of the albumin, it is a marker of contraction of the plasma volume. Figure 2C was constructed specifically to make that distinction visible.

### *One mechanical trigger, two conduits*

Why should a healthy 27-year-old woman leak chyle at 2,500 mL/day and air into both pleural spaces at the same time? The most economical explanation is that a single mechanical event, or a repeated one, breached two conduits at once. Figure 5A sets out that hypothesis schematically.

As shown in Figure 5A, the proposed trigger is paroxysmal cough in the setting of the bilateral pneumonia documented on computed tomography, with the repeated Valsalva manoeuvres that accompany a violent cough. Such an effort raises the alveolar transmural pressure gradient and the intrathoracic and intra-abdominal pressures in the same instant. The left limb of Figure 5A is the Macklin sequence and accounts for the air: rupture of marginal alveoli, air tracking centrally along the bronchovascular sheaths to produce pneumomediastinum, and then decompression into the subcutaneous tissues and the pleural spaces. That sequence is not speculative. A systematic review of seven studies and 979 patients found the Macklin sign associated with barotrauma in 124 of 138 cases, or 89.8 %, and preceding barotrauma by three to eight days in 65 of 69 cases, or 94.2 %, and concluded that it anticipates barotrauma rather than merely accompanying it.[15] In a retrospective study of 40 patients with spontaneous pneumomediastinum, the Macklin

effect was demonstrable in 38, most commonly as gas accumulation around the mediastinum near the pulmonary hilum, and persistent coughing was the presenting symptom in 17.5 %.[17] The clinical phenotype produced by this mechanism in young adults is exactly what our patient displayed: in a single-centre series of 87 patients with pneumomediastinum, radiological subcutaneous emphysema was present in 49 % of spontaneous and 74 % of secondary cases, and secondary cases were significantly more likely to have an associated pneumothorax, 58 % versus 19 %, an associated pleural effusion, 18 % versus 0 %, and a chest tube, 58 % versus 18.9 %.[16] Our patient had all four of those features, which places her firmly in the secondary rather than the primary group, and secondary pneumomediastinum in that series carried a 10 % in-hospital mortality against zero for the spontaneous form. Two further reports show how little mechanical provocation is required: a woman in her mid-twenties developed pneumomediastinum with extensive subcutaneous emphysema after a five-day sore throat without even a cough,[18] and a 25-year-old man presented with pneumomediastinum, pneumopericardium and bilateral pneumothoraces after a respiratory infection and was managed conservatively to near-resolution by day six.[19] A retrospective review of 63 paediatric patients makes the same distinction that matters here: none of those with primary spontaneous pneumomediastinum developed complications, whereas 16.7 % of those with a secondary cause did, and 54.2 % required at least one additional intervention.[24]

The right limb of Figure 5A accounts for the chyle. The thoracic duct drains into a fixed outflow at the left venous angle, and a sudden rise in intra-abdominal pressure transmitted through the cisterna chyli produces a flow surge against that fixed outflow. The best published analogue is a woman in her thirties who developed acute left cervical swelling followed by large bilateral chylous effusions and respiratory failure requiring ventilatory support, in whom a significant tonic-clonic seizure six days earlier was proposed as the cause, the bilaterality suggesting injury at more than one level of the duct; the authors noted that no such case had previously been reported.[20] The paediatric literature supplies the cough-specific version: a previously healthy two-year-old boy admitted after a bout of heavy coughing with extensive opacification of the right lung, in whom pleural fluid analysis gave the unexpected diagnosis of chylothorax and in whom a fat-free diet and two weeks of continuous drainage failed before octreotide produced complete resolution within 15 days.[21] The obstetric literature supplies the purest Valsalva analogue of all: a 29-year-old primigravida with a protracted second stage of labour who developed dyspnoea, chest pain and subcutaneous emphysema of the face, neck and anterior chest wall immediately after delivery, in whom the chest radiograph confirmed pneumomediastinum and conservative management sufficed.[25] Cough-induced chylothorax and effort-induced pneumomediastinum are therefore both established entities. What this case adds is their coincidence, which the shared mechanism predicts but which does not appear to have been documented. One weakness of the hypothesis must be conceded at this point rather than left to the limitations. The cough documented in our patient was described as occasional and productive of scant white sputum, which is a considerably milder history than the violent, discrete mechanical events reported in the seizure- and cough-associated cases above. The schematic in Figure 5A should therefore be read as depicting the mechanism we hypothesise rather than a mechanical event of documented severity; what the record supports is repeated coughing in the setting of bilateral pneumonia over more than three weeks, so the argument rests on cumulative rather than single-event strain.

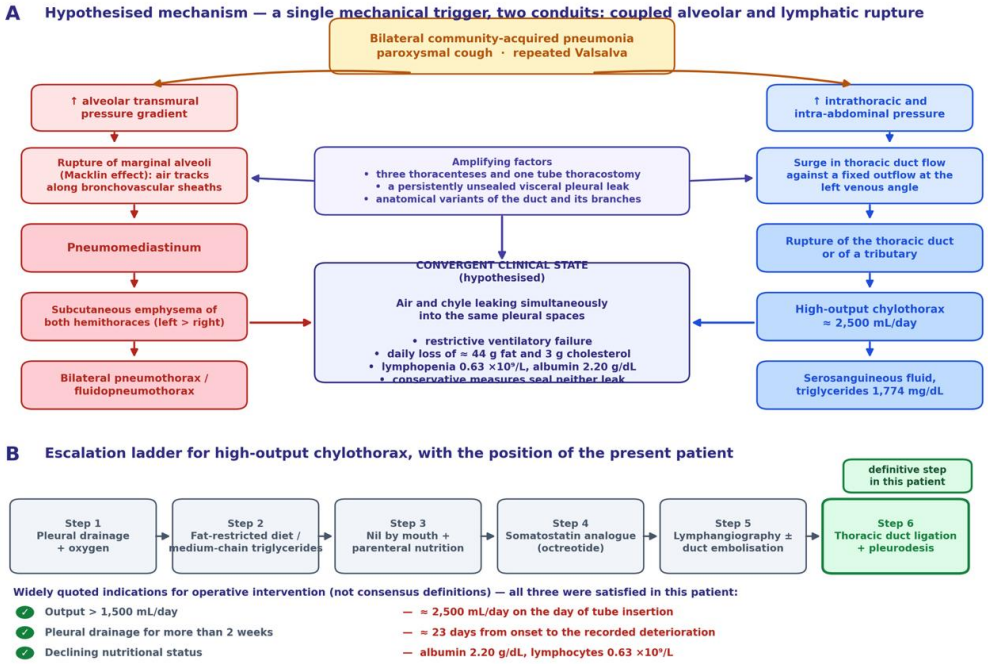


**Figure 5.** (A) The hypothesised mechanism advanced in this report; the arrows denote a proposed rather than a demonstrated causal sequence. A single mechanical trigger — paroxysmal cough in the setting of bilateral pneumonia, with the repeated Valsalva manoeuvres that accompany it — raises the alveolar transmural pressure gradient and the intrathoracic and intra-abdominal pressures at the same moment, and can therefore breach the alveolar and the lymphatic conduits in parallel. The left limb is the Macklin sequence that accounts for the pneumomediastinum, the subcutaneous emphysema and the bilateral pneumothoraces; the right limb accounts for the chylothorax. The two limbs converge on a single clinical state in which neither leak can be sealed by conservative measures. (B) The escalation ladder for high-output chylothorax with the position of the present patient marked. The three indications for operation shown at the foot of the panel are widely quoted rather than formally standardised, and all three were satisfied before the patient came to operation.

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The chronology of imaging in this patient supports the hypothesis in an unexpected way. As recorded in Table 2, bedside thoracic ultrasonography on arrival on 17 October showed predominantly right-sided pleural fluid with lung sliding preserved bilaterally. Preserved lung sliding argues strongly against a large pneumothorax at the point of examination, although a single bedside study performed to assess fluid rather than air, and dependent on operator and window, cannot exclude a small or loculated anterior collection. On the most defensible reading, the bilateral air collections displayed in Figure 4A and Figure 4B were not present in that volume on admission and developed during it. Two influences plausibly contributed, and they are shown as the amplifying factors in the centre of Figure 5A. The first is procedural: by the time of the computed tomography the patient had undergone three thoracenteses and one tube thoracostomy. Even in expert hands pleural puncture is not free of risk; a systematic review and meta-analysis of three randomised trials including 417 patients found that ultrasound guidance was successful in 195 of 202 patients, 96.5 %, against 189 of 215, 87.9 %, with landmark technique, and trended towards lower complication rates with an odds ratio of 0.18, although the confidence interval was wide.[26] Four pleural interventions in eleven days in a lung already prevented from re-expanding is a plausible route for air to enter the pleural space. The second influence is that the cough and the pneumonia, and therefore the Macklin mechanism itself, continued during the admission, which is precisely the pattern the systematic review describes when it reports that the Macklin sign precedes clinically apparent barotrauma by three to eight days.[15] The apparent discrepancy between right-predominant fluid on admission and left-predominant fluidopneumothorax on the later study is resolved by the same reasoning: the right-sided tube was actively draining that hemithorax, leaving air on the right and residual fluid, together with air, on the undrained left.

One anatomical caveat belongs here. The assumption that a single duct injury produces a unilateral effusion is not safe. Fluorescence thoracoscopy during right lung cancer surgery identified, in four patients, a collateral branch arising from the thoracic duct at the level of the azygos vein arch, running along the superior edge of that arch from posterior to anterior and then turning upward in the anterior mediastinum with a complex branching pattern, branches which are easily injured and can cause postoperative chylothorax.[27] Variant anatomy of this kind provides a straightforward explanation for how one mechanical insult might produce bilateral chylous collections, as in the seizure-associated case,[20] and why the site of a leak is so often never found.

### *The gap in the aetiological work-up*

Honesty requires that the principal weakness of this work-up be stated plainly rather than buried. Lymphoma is the commonest non-traumatic cause of chylothorax, and flow cytometry of the pleural fluid was not performed. As recorded in Table 2 and in the third row of Table 3, that omission means a lymphoproliferative cause was never formally excluded. The literature shows why this matters. In the tertiary cohort of 40 patients, haematological malignancy accounted for 22.5 % of non-traumatic chylothorax and solid-organ malignancy for 45 %.[1] Diagnosis can be genuinely elusive: in a patient in her seventies with subtotal right pleural effusion shown to be chylous, mediastinal, abdominal and retroperitoneal lymphadenopathy had been present without progression for six years, initial tests were inconclusive, and only video-assisted thoracoscopic mediastinal lymph node dissection and biopsy established follicular lymphoma.[6] In another patient a chylous effusion containing adenocarcinoma cells led to a diagnosis of cancer of unknown primary after systemic examination failed to identify the tumour, and death followed twelve months later.[5] Chylous effusions in lymphoproliferative disease are also often refractory: in 17 such patients with effusions unresponsive to chemotherapy and conservative therapy, lymphangiography showed chylo-lymphatic reflux in 47 %, leakage in 11.8 %, combined leakage and reflux in 17.6 % and lymphatic obstruction in 11.8 %, with resolution in 15 of 17 after lymphangiography with or without embolisation.[23] Chylothorax can equally be the presenting manifestation of a systemic inflammatory disease, as in a patient with systemic lupus erythematosus whose chylothorax resolved with immunosuppression, recurred as corticosteroids were tapered and then responded to rituximab,[4] and it has been described as a post-acute sequela of SARS-CoV-2 infection.[7]

Several features argue against malignancy in this patient: her age, the absence of lymphadenopathy on examination and on contrast-enhanced computed tomography of the thorax, the absence of weight loss or night sweats, the abrupt onset in the context of an acute respiratory illness, and the durable response to mechanical treatment of the leak. None of these is decisive. The appropriate conclusion is not that malignancy has been ruled out but that it has not been ruled out. The diagnosis this report offers is therefore not idiopathic chylothorax but chylothorax of undetermined aetiology, in which a mechanical cause is the most economical explanation and a lymphoproliferative cause has not been excluded. In any similar patient pleural fluid flow cytometry should be obtained at the first opportunity, before drainage volumes fall and the opportunity is lost. The same applies to lymphatic imaging. In the tertiary cohort, lymphatic imaging was performed in only 20 % of patients and identified a chyle leak in just one,[1] which suggests both that it is underused and that its yield in non-traumatic disease is modest; newer approaches such as magnetic resonance ductography and intranodal computed tomographic lymphangiography may change that calculus, but they were not available here.

### *The metabolic and immunological ledger*

The strongest argument for early operation in this patient was never the volume in the bottle. It was the ledger of what that volume contained. Two recent primary studies quantify the same signature we observed. In the study of 18 neonates with chylothorax, three patients, 16.7 %, had absolute lymphopenia with particular depletion of T-cell and natural killer cell subsets, five, 27.8 %, had hypogammaglobulinaemia, immunoglobulin G and M levels were significantly lower in chyle than in blood, and 15 of the 18, 83.3 %, developed late-onset sepsis, most often bacterial.[22] In a descriptive cohort of 23 children with chylous effusion after cardiac surgery, 83 % were lymphopenic and 74 % hypoalbuminaemic at the time of diagnosis, with hypocalcaemia in nearly half and hypophosphataemia in 26 %, and the longer the interval from surgery to the onset of the effusion, the lower the albumin and the high-density lipoprotein cholesterol.[28] Our patient's albumin of 2.20 g/dL and absolute lymphocyte count of 0.63 $\times 10^9$/L, as detailed in Table 1, place her squarely within that pattern.

The clinical consequence of an absolute lymphocyte count in this range is not theoretical. In a retrospective cohort of 286 adults admitted for burn or inhalation injury, 62, 21.7 %, were lymphopenic on admission using a threshold of 1.0 $\times 10^9$/L, and admission lymphopenia was associated with an increased risk of pneumonia and acute kidney injury.[29] Trajectory matters as much as the single value: in a retrospective cohort of 2,149 patients with sepsis, externally validated in a further 2,388 and extended prospectively to 1,056, latent class mixed modelling of serial lymphocyte counts identified four phenotypes, of which a high-declining group comprising 3.8 % of patients carried the highest severity and mortality at 25.9 %, while a stable-low group

accounted for 23.8 %; lymphocyte trajectory phenotype was an independent predictor of 28-day mortality.[30] Our patient's counts of 0.63 and 0.60 ×10⁹/L twelve days apart are the stable-low pattern, and they occurred while she also had radiologically documented bilateral pneumonia. The neutrophil-to-lymphocyte ratio adds a further dimension: in a secondary analysis of 812 older patients hospitalised with community-acquired pneumonia, 30-day mortality reached 18.3 % in the highest quartile of that ratio, and adding the ratio to the CURB-65, CRB-65, A-DROP and SMART-COP scores improved discrimination.[31] The values of 24.7 and 21.5 recorded in Table 1 and Table 4 are far above the range in which that analysis found risk to accelerate. This is the sense in which the chylothorax was not simply a drainage problem: it was continuously removing the lymphocytes that the coexisting pneumonia required. The inference should be stated with its limits. Those cohorts establish that lymphopenia and a high neutrophil-to-lymphocyte ratio are prognostic markers in burn injury, sepsis and community-acquired pneumonia; none of them demonstrates that correcting a chyle leak improves outcome by correcting lymphopenia, and no study has shown an outcome benefit from earlier operation on immunological grounds. The practical recommendation that follows is therefore about what to measure and what to weigh, not about a proven causal chain.

### *Deciding that conservative management has failed*

The three indications for operation shown at the foot of Figure 5B — an output above 1,500 mL/day, a requirement for pleural drainage beyond two weeks, and declining nutritional status — are quoted very widely, but as the review of 2,942 pulmonary resections observed, the criteria for surgical treatment are in fact not consistent across centres.[9] That study offers a more discriminating alternative: among 108 patients who developed postoperative chylothorax, of whom 79 were managed with a low-fat diet and 29 with total parenteral nutrition, the pleural fluid triglyceride level after two days of a low-fat diet predicted prognosis better than drainage volume did, and a threshold of 1.33 mmol/L gave a sensitivity of 100 % and a specificity of 80.6 %.[9] Our patient's concentration of 1,774 mg/dL, which is approximately 20 mmol/L, is an order of magnitude above that threshold of 1.33 mmol/L, although she was not on a defined low-fat diet at the time of sampling and the comparison is therefore indicative rather than exact.

As shown in Figure 5B, all three conventional indications were nonetheless satisfied. The output of approximately 2,500 mL/day on the day of tube insertion exceeded 1,500 mL/day. As computed in Table 4, pleural fluid had been drained repeatedly for between 17 and 23 days by the time of the documented deterioration on 29 October, the range reflecting the fact that the source record places the first thoracentesis somewhere between 6 and 12 October. Nutritional decline was documented by the albumin of 2.20 g/dL and the lymphopenia of 0.63 ×10⁹/L in Table 1. The counter-argument for continued conservative management deserves a fair hearing, and the literature does supply one. In a comparison of two management eras in paediatric cardiac surgery, a strategy of nil by mouth with total parenteral nutrition, extended milrinone and no chest-tube suction reduced the occurrence of massive chylothorax above 20 mL/kg/day from 51 % to 20 %, and although drainage volume, intubation time and length of stay all favoured the newer strategy, none of those differences reached significance.[32] Somatostatin analogues are the other conservative option, and the evidence for them is more equivocal than their widespread use suggests: in a translational study combining myography of 12 human thoracic ducts with a double-blinded randomised cross-over trial in 16 healthy adults, octreotide stimulated thoracic duct contraction rate concentration-dependently in the myograph, yet spontaneous lymphatic contractions and lymph pressure measured by near-infrared fluorescence did not differ from placebo in vivo.[33] In the paediatric cough-induced case, by contrast, octreotide succeeded where a fat-free diet and two weeks of drainage had failed.[21]

What tips the balance in an adult losing 44 g of fat and 400 kcal a day is the measurable cost of delay. A systematic review and meta-analysis of 22 studies of chylothorax after cardiothoracic surgery found that non-operative treatment carried a mean chest-tube duration of 16.1 days, with a 95 % confidence interval of 12.5 to 19.6, and a mean hospital stay of 23.7 days, with a confidence interval of 16.1 to 31.4, and that pulmonary complications, infections and arrhythmia were the commonest adverse events.[34] Set against a lymphopenic patient with bilateral pneumonia and a falling $PaO_2$, another two weeks of drainage is not a neutral choice.

### *Definitive management and why it worked*

Video-assisted thoracoscopic ligation of the thoracic duct with mechanical pleurodesis addressed both components of this patient's problem in one operation, and it is worth being explicit about which element did which. Ligation interrupted the lymphatic leak at its source. Pleurodesis addressed the air, because a leak of air into a pleural space that has been obliterated by adhesion of the visceral to the parietal pleura has nowhere to accumulate; sealing the space is a treatment for an air leak whose exact site has not been identified, and in a patient whose visceral pleural surface was leaking at an unknown point that was the more practical of the two available strategies. The outcome was unambiguous. As plotted in the final bars of Figure 2D and computed in Table 4, drainage fell from approximately 2,500 mL/day to 180–250 mL/day, a reduction of 90 to 93 %, and clinical and radiological improvement followed with discharge on 2 November.

The combination of ligation and pleurodesis is supported by the recent primary literature. In recurrent bilateral idiopathic chylothorax, total parenteral nutrition, octreotide and thoracic duct embolisation all failed in succession, and resolution was achieved only with thoracic duct ligation and talc pleurodesis.[2] A standardised technique for refractory post-oesophagectomy chylothorax combines pre-operative bilateral inguinal lymph node injection of indocyanine green, a provocative fatty meal through the nasogastric tube, near-infrared identification of the duct, mass ligation with silk and reinforcement with polymer clips, and talc pleurodesis to ensure pleural symphysis; the reported patient was discharged on the fifth postoperative day with no recurrence at 30 days.[35] Where thoracic-level surgery fails, the abdominal approach can still succeed: in a retrospective series of 14 patients with refractory chylothorax operated on between 2011 and 2025, half of whom had bilateral effusions and half of whom had a postoperative aetiology, open transabdominal ligation of the cisterna chyli produced a median postoperative stay of nine days over a median follow-up of 75 months.[36] The largest recent primary series puts the relative yield of each option in numbers. Among 17 oesophageal-resection and approximately 51 lung-resection patients treated over 17 years for postoperative chylothorax, curative therapy in the oesophageal group was achieved by total parenteral nutrition in 18 %, a medium-chain-triglyceride enteral diet in 18 % and surgical ligation of the thoracic duct in 35 %, while in the lung-resection group the most effective treatments were octreotide with midodrine in 29 %, surgical ligation in 23 % and lymphangiography with embolisation, alone or combined with octreotide, in 15 %; the authors concluded that every patient was ultimately cured by aggressive invasive procedures directed mostly at

interruption or ligation of the duct, while some responded to dietary and drug therapy alone.[10] Duct interruption is therefore not a last resort but the single most productive step in the sequence.

There is also an external benchmark for the result obtained in our patient. In a retrospective series of five patients — two of them with spontaneous rather than postoperative chylothorax — who underwent near-infrared, indocyanine-green-guided thoracoscopic duct ligation after inguinal nodal injection, the mean operative time was 62 minutes, the mean postoperative drainage was 229.6 mL/day, the mean duration of postoperative chest drainage was 6.2 days, the mean hospital stay was 17.8 days and no patient had a recurrence during follow-up.[37] Our patient's postoperative output of 180 to 250 mL/day brackets that reported mean of 229.6 mL/day almost exactly, which suggests that the residual drainage seen after successful duct ligation is a reproducible quantity rather than an idiosyncrasy of this case, and that it should not be mistaken for persistent chyle leakage. Our patient's course, summarised alongside the comparator literature in Table 5, illustrates the value of moving decisively down the sequence when the ledger rather than the volume is used to define failure; the two surgical rows of Table 5 give the outcome figures against which our own result should be read.

**Table 5.** The present case set against recent primary reports of non-traumatic chylothorax and of spontaneous air-leak syndrome.

| Report | Design | Population | Finding relevant to the present case |
|---|---|---|---|
| Chua and colleagues, 2026[1] | Retrospective cohort, 40 patients | Non-traumatic chylothorax at a tertiary centre; median age 61 years | Cancer accounted for 67.5 % of cases; 90-day and 1-year mortality were 37.5 % and 55 %; lymphatic imaging was performed in only 20 % |
| Lawrence and colleagues, 2025[20] | Case report | Woman in her thirties with bilateral chylothorax | A tonic-clonic seizure six days earlier was proposed as a purely mechanical cause of multilevel duct injury — the closest published analogue to the mechanism argued here |
| Anger and colleagues, 2023[21] | Case report with literature review | Two-year-old boy after a bout of heavy coughing | Cough alone ruptured a lymphatic channel; a fat-free diet and two weeks of drainage failed and the effusion resolved only after octreotide |
| Ng and colleagues, 2023[2] | Case report | Recurrent bilateral idiopathic chylothorax | Parenteral nutrition, octreotide and duct embolisation all failed; duct ligation with talc pleurodesis was curative |
| Çetinkaya and Batırel, 2025[36] | Retrospective case series, 14 patients | Refractory chylothorax after failed conservative or transthoracic therapy | Half had bilateral effusions; abdominal cisterna chyli ligation salvaged disease that thoracic-level surgery had not controlled |
| Son and colleagues, 2025[8] | Retrospective cohort | Chylothorax after minimally invasive lung resection | A threshold of 500 mL/day separated high- from low-volume disease within a stepwise conservative protocol, and nutritional indices tracked severity |
| Santos and colleagues, 2023[34] | Systematic review and meta-analysis, 22 studies | Chylothorax after cardiothoracic surgery | Non-operative management carried a mean chest-tube duration of 16.1 days and a mean hospital stay of 23.7 days — the measurable price of waiting |
| Belletti and colleagues, 2023[15] | Systematic review, 7 studies, 979 patients | Patients with the Macklin sign on computed tomography | Present in 4–22 % and associated with barotrauma in 89.8 %; it preceded barotrauma by 3–8 days in 94.2 % of cases |
| Jan and colleagues, 2025[18] | Case report | Woman in her mid-twenties after an upper respiratory tract infection | Pneumomediastinum with extensive subcutaneous emphysema followed airway irritation alone and resolved on conservative treatment within 24 hours |
| De Rose and colleagues, 2025[22] | Retrospective study, 18 neonates | Chylothorax, with paired samples of blood and chyle | Chyle was lymphocyte- and T-cell-rich; 16.7 % were frankly lymphopenic and 83.3 % developed late-onset sepsis |
| Silva and colleagues, 2024[28] | Descriptive cohort, 23 children | Chylous effusion after cardiac surgery | At diagnosis 83 % were lymphopenic and 74 % hypoalbuminaemic — the same biochemical signature seen here |
| Robinson and colleagues, 2026[10] | Retrospective cohort over 17 years, 17 oesophageal and about 51 lung resections | Postoperative chylothorax after oesophageal or lung resection | Curative therapy came from surgical duct ligation in 35 % of the oesophageal and 23 % of the lung group, octreotide with midodrine in 29 % of the lung group, and lymphangiography with embolisation in 15 %; the authors state that optimal recommendations are not well defined |
| Shao and colleagues, 2025[37] | Retrospective series, 5 patients | Thoracoscopic duct ligation guided by near-infrared indocyanine-green imaging, two patients with spontaneous chylothorax | Mean operative time 62 minutes, mean postoperative drainage 229.6 mL/day, mean chest drainage 6.2 days, mean stay 17.8 days, no recurrence — a direct benchmark for the 180–250 mL/day recorded here |
| Present report, 2026 | Case report | 27-year-old woman, no trauma, thoracic surgery or malignancy | High-output serosanguineous chylothorax of approximately 2,500 mL/day with bilateral fluidopneumothorax, pneumomediastinum and extensive subcutaneous emphysema, resolving after thoracoscopic duct ligation and mechanical pleurodesis |

***Notes:*** The comparator reports are a purposive selection from the verified reference base, chosen to represent each thematic domain of the argument, and are not an exhaustive enumeration of the relevant literature. The final row, printed in bold red, is the present case. Reports are ordered by theme rather than by date: cohort and series data on non-traumatic chylothorax first, then the mechanical-trigger case reports, then the surgical and air-leak literature, then the studies that quantify the metabolic and immunological consequences of chyle loss.

As set out in Table 5, this case can be placed beside thirteen recent primary reports. Three points emerge from that comparison. First, in the first row of Table 5, the demographic mismatch is complete: a median age of 61 years and cancer in two-thirds of patients against a 27-year-old with no malignancy identified.[1] Second, in the second and third rows of Table 5, the mechanical hypothesis advanced here has direct precedent for the lymphatic limb alone,[20] and in the rows for the Macklin systematic review and the two spontaneous pneumomediastinum reports it has direct precedent for the alveolar limb alone;[15,18] what is absent from Table 5, and from our reading of the literature, is any report in which both limbs are present in the same patient; the final row of Table 5, printed in bold red, is the present case and is the only entry in which they coincide. Third, in the last two comparator rows of Table 5, the biochemical signature of lymphopenia and hypoalbuminaemia is reproduced almost exactly, which suggests that the metabolic consequences of chyle loss are largely independent of the underlying cause.[22,28]

### *Limitations*

Seven limitations should be stated. First, and most importantly, flow cytometry of the pleural fluid was not performed, so a lymphoproliferative cause has not been formally excluded, as recorded in Table 2 and marked in bold red in the third row of Table 3. Second, no lymphatic imaging was undertaken, so the site of the leak was never localised and the mechanistic hypothesis in Figure 5A remains an inference from the clinical sequence rather than a demonstrated anatomical finding. Third, the images available to us were of limited technical quality: the radiograph in Figure 3 required rotation and contrast normalisation before it could be read, and one of the two computed tomography levels exists only as a photograph of a reporting monitor, so both had to be processed as declared and neither can be re-examined by a reader at the original resolution. Fourth, paired serum triglyceride and cholesterol concentrations were not measured, so the pleural-fluid-to-serum ratios that some authors regard as the more robust criterion could not be computed.[12] Fifth, several timings are absent from the source record: the exact date of the first thoracentesis, which is why Table 4 gives a range of 17 to 23 days for the duration of drainage, the operative date, which is why Figure 1 shows only a window, and the timing of pulmonary function testing relative to drainage, which is why the severe restrictive defect in Table 2 should be read descriptively. Sixth, the fraction of inspired oxygen accompanying each arterial sample was not recorded, so no $PaO_2/FiO_2$ ratio is asserted anywhere in this report; Table 4 gives instead an arithmetic bound, since the ratio cannot have exceeded 52 ÷ 0.21, approximately 250, if the sample was drawn on room air, and was of the order of 52 ÷ 0.40, approximately 130, if the face mask documented on admission was still in use. Gas exchange was severely impaired on either assumption. Seventh, follow-up ended with discharge on 2 November 2025, and since idiopathic and recurrent chylothorax can relapse long after apparently successful treatment,[2] the absence of documented recurrence within the admission cannot be taken as evidence of a durable cure. Four further caveats are worth noting. The derived quantities in Table 4 assume that the triglyceride concentration measured on a single sample applied to the whole of the day's output, which is unlikely to be exactly true. Pleural fluid protein, lactate dehydrogenase and glucose and the paired serum values were not measured, so Light's criteria could not be applied and the exudative label rests on the Rivalta test alone. No parietal pleural biopsy is recorded at thoracoscopy, although the patient was anaesthetised with the pleural cavity open and tissue could have been obtained at no meaningful additional risk; this is the most easily avoidable of the gaps listed here. And the mechanistic reasoning draws on studies of Macklin physiology conducted largely in ventilated patients with coronavirus disease 2019 rather than in spontaneously breathing patients with community-acquired pneumonia,[15] while the cough documented in our own patient was mild.

### *What this case changes at the bedside*

Three practical propositions follow. The first concerns testing: any recurrent, rapidly re-accumulating pleural effusion in a young adult should have triglyceride and cholesterol measured on the first sample, whatever the fluid looks like, because a serosanguineous appearance does not exclude chyle and the biochemistry costs nothing.[11] The second concerns aetiological completeness: when chyle is confirmed, flow cytometry and consideration of lymphatic imaging belong in the same episode of care, not in a later one, because malignancy dominates the non-traumatic causes and diagnosis can require thoracoscopic biopsy.[1,6] The third concerns timing: the decision to operate should be driven by an explicit ledger — daily lipid and energy loss, albumin, absolute lymphocyte count and gas exchange, as tabulated in Table 1 and Table 4 — rather than by drainage volume alone, because the published cost of prolonged conservative management is measured in weeks of drainage and hospital stay and in infectious complications.[9,34-37] The fourth concerns tissue: when a patient with confirmed chyle and an unestablished aetiology comes to thoracoscopy, parietal pleural tissue should be taken at that opportunity, because the patient is already anaesthetised with the pleural cavity open and a biopsy adds no meaningful risk while going some way towards the question that biochemistry cannot answer. No biopsy is recorded in our patient, and in retrospect that was the single most easily avoidable gap in the work-up. In a patient who is simultaneously leaking air, losing lymphocytes and fighting pneumonia, the ledger closes early.

## 4. Conclusion

This report describes a 27-year-old woman with no history of trauma, thoracic surgery or malignancy in whom a high-output non-traumatic chylothorax of approximately 2,500 mL/day occurred together with bilateral fluidopneumothorax, pneumomediastinum and extensive subcutaneous emphysema, and in whom video-assisted thoracoscopic thoracic duct ligation with mechanical pleurodesis reduced drainage by approximately 91 % and was followed by clinical and radiological recovery.

Four conclusions follow. First, the diagnosis rested entirely on biochemistry. The fluid was a serosanguineous exudate rather than a milky one, and a triglyceride concentration of 1,774 mg/dL with a cholesterol of 130 mg/dL, a triglyceride-to-cholesterol ratio of 13.6 and lymphocyte predominance, as detailed in Table 2, established chyle where appearance alone would have argued against it. Measuring pleural fluid lipids on the first sample of any recurrent effusion is a small investment against a large diagnostic loss.

Second, the two heuristics that a high output implies trauma and that a non-traumatic mechanism implies an indolent course were both false in this patient, and neither should be allowed to delay biochemical confirmation or definitive treatment.

Third, the case supports a unifying mechanical reading, set out in Figure 5A, in which repeated coughing in the setting of bilateral pneumonia raised alveolar and intra-abdominal pressures together and breached both conduits. Preserved bilateral lung sliding on admission ultrasonography argues that the air collections were not present in that volume on 17 October, implicating both the continuing Macklin mechanism and the four pleural procedures performed within eleven days. The leak site was never imaged and the documented cough was mild, so this remains a hypothesis — but it is the reading that requires the fewest independent coincidences.

Fourth, and most usable at the bedside, failure of conservative treatment in high-output chylothorax is better defined by an explicit metabolic and immunological ledger than by the volume in the drainage bottle. In this patient that ledger — approximately 44 g of triglyceride and 400 kcal lost each day, an albumin of 2.20 g/dL, an absolute lymphocyte count of $0.63 \times 10^9$/L that did not correct over twelve days, a neutrophil-to-lymphocyte ratio above 21, haemoconcentration, and a $PaO_2$ falling from 104 to 52 mmHg, as set out in Table 1 and Table 4 and displayed in Figure 2 — closed decisively in favour of early operation. Where such a patient also has radiologically documented pneumonia, the continuing loss of lymphocytes is an argument for intervention in its own right.

### *Declarations*

#### *Ethics approval and consent to participate*

This case report was prepared in accordance with the ethical standards of the institutional research committee and with the 1964 Declaration of Helsinki and its later amendments. Institutional approval for the publication of anonymised case material was obtained.

#### *Consent for publication*

Written informed consent for the publication of this case report and of the accompanying clinical and radiological images was obtained from the patient. No direct or indirect patient identifier is present in any of the images reproduced here; the source radiograph and computed tomography images were reviewed at high magnification and carried no burned-in demographic data.

#### *Reporting guideline*

This report was prepared in accordance with the CARE reporting guideline for case reports. A completed CARE checklist is available from the corresponding author.

#### *Evidentiary composition of the reference base*

All thirty-seven references report original patient data: case reports and case series, retrospective and prospective cohorts, systematic reviews with and without meta-analysis, a randomised cross-over trial, a diagnostic-accuracy study and a forensic autopsy report. Two are hybrid articles that pair original patient data with a literature review, and even if those two are set aside the base remains 94.6 % primary research. No pure narrative review and no practice guideline is cited, and every reference was published between 2021 and 2026.

#### *Follow-up*

Follow-up ends at discharge on 2 November 2025. No post-discharge imaging, drainage record or clinic review is available to the authors, and the absence of documented recurrence therefore refers only to the remainder of the admission.

#### *Availability of data and materials*

All data supporting the findings of this report are contained within the article. Further anonymised details are available from the corresponding author on reasonable request.

#### *Competing interests*

The authors declare that they have no competing interests.

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#### *Authors' contributions*

All authors participated in the clinical care of the patient or in the acquisition and interpretation of the clinical data, contributed to the drafting and critical revision of the manuscript, and approved the final version. All authors accept responsibility for the integrity of the work as a whole.

#### *Use of artificial intelligence*

All clinical data were extracted from the primary medical record by the authors, every reference was verified against its digital object identifier, and the authors reviewed, corrected and approved the entire manuscript and take full responsibility for its content.

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