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Use of an Alpha-1 Adrenoreceptor Agonist in the Management of Recurrent Refractory Idiopathic Chylothorax

Parthipan Sivakumar, BM; and Liju Ahmed, MBBS

A 70-year-old woman presents with recurrent idiopathic chylothorax refractory to both medical and surgical treatment. To our knowledge, this is the first reported case where midodrine, an alpha-1 receptor agonist, was used as an adjunctive therapy for idiopathic chylothorax resulting in both a radiographic and clinical response. CHEST 2018; 154(1):e1-e4

KEY WORDS: adrenergic agents; chyle; chylothorax; midodrine; pleural effusion

A 70-year-old woman presented to the hospital with worsening fatigue and breathlessness. She had a medical history of breast cancer treated with surgery and chemotherapy 17 years prior to her presentation.

Case Report

Her chest radiograph demonstrated a right pleural effusion (Fig 1A), and she underwent an ultrasound-guided thoracocentesis yielding milky white fluid with a triglyceride level of 20.29 mmol/L, consistent with a chylothorax. However, CT scan failed to identify a cause, with no evidence of breast cancer recurrence. During a protracted 7-month admission, she underwent multiple interventions. This included total parenteral nutrition, octreotide, multiple ascitic drains for chyloperitoneum, and failed attempts at local anaesthetic thoracoscopic talc pleurodesis on both sides. Pleural biopsies were benign, and three attempts at CT lymphangiography failed to identify a source for the leak. She therefore underwent thoracic duct ligation and at surgery the cisterna chyli was seen to herniate through the diaphragm (Fig 2). Despite ligation, she required a further laparoscopic resection of the paraaortic and coeliac truck lymphatics to stop the leak and she was discharged home. (Fig 1B).

Eight months later, her breathlessness returned. Chest radiograph confirmed a new left-sided pleural effusion with increase in size of the right-sided collection (Fig 3A). Pleural aspiration of the left side confirmed a chylous effusion. There was no evidence of ascites on this occasion. Repeat CT imaging again failed to demonstrate a cause and confirmed that her breast cancer remained in remission.

She was admitted to hospital for left-sided thoracoscopy and iodine pleurodesis. This was unsuccessful with an average output of > 300 mL/d from her left-sided chest drain. Talc slurry pleurodesis via the existing drain was attempted 48 h later; however, the drain

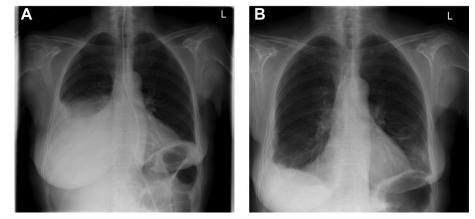
DOI: https://doi.org/10.1016/j.chest.2018.02.005

AFFILIATIONS: From the Department of Thoracic Medicine, Guy's and St Thomas' NHS Foundation Trust, London, England.

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CORRESPONDENCE TO: Parthipan Sivakumar, BM, Respiratory Department, Guy's and St Thomas' NHS Foundation Trust, Westminster Bridge Rd, London, SE1 7EH, England; e-mail: deepan. sivakumar@kcl.ac.uk

Figure 1 – A, Chest radiograph during the initial admission with rightsided pleural effusion. B, Chest radiograph on discharge from initial admission.



output did not decrease. Octreotide was not used because of the lack of response during her previous admission. Based on a case report of treatment success with the alpha-1 agonist, midodrine, in postoperative thoracic duct injury,¹ oral midodrine was commenced, uptitrating the dose to a maximum of 10 mg four times daily. After 48 h of therapy, the drain output reduced to 150 mL/d, the drain was removed, and she was discharged with close outpatient follow-up. She continued midodrine in the community for 3 weeks with ongoing improvement in her symptoms. Serial chest radiographs (Figs 3B-D) demonstrated regression of the pleural effusions. She remained normotensive and did not experience any other side effects of therapy. The midodrine was then discontinued after 3 weeks. Although 2 weeks later there was some evidence of radiographic deterioration (Fig 3E), this remained stable at her 6-month follow-up (Fig 3F), where she remained symptom free.

Discussion

Disruption or obstruction of the thoracic duct or its tributaries may result in the leakage of chyle into the



Figure 2 – Views at thoracoscopic surgery—the cisterna chyli herniating through the diaphragm.

pleural space. Of chylous effusions, 50% are nontraumatic, with idiopathic chylothorax accounting for 6% of nontraumatic cases.² Although management of nontraumatic chylothorax is centred around treatment of the underlying condition, idiopathic chylothorax presents a unique and complex challenge. Optimal medical and surgical management is essential, centred around dietary therapy (excluding long-chain triglycerides), pleurodesis, and thoracic duct ligation. Several case reports support the use of octreotide, a somatostatin analogue; however, the mechanism of this is poorly understood and it may reduce lymphatic flow by inhibiting gastric, pancreatic, and biliary secretions.³

Several series report the successful use of intravenous etilefrine, an alpha-1 and beta-1 adrenoreceptor agonist, in the management of postoperative chyle leak.⁴ On this basis, Liou et al¹ describe the use of midodrine, an oral, selective alpha-1 adrenoreceptor agonist, as a novel therapeutic agent in thoracic duct injury after esophagogastrectomy. Midodrine's relatively low side effect profile and selective alpha-1 agonist properties prompted our trial of this agent in this unique case after appropriate patient consent. Although we acknowledge that the previous pleurodesis attempts with talc and iodine may have had some effect on the chyle leak, the regression on both sides only occurred after adjunctive treatment with midodrine with slight radiographic deterioration after cessation which may suggest a therapeutic effect.

Because lymphatic transit through the thoracic duct is closely related to the portal circulation with thoracic duct distension, raised duct pressures, and increased lymph flow rates seen in portal venous hypertension,⁵ we postulate that modulation of portal blood flow with a potent splanchnic vasoconstrictor such as midodrine⁶ may reduce lymphatic formation and transport. This

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