

Figure 1 Correlation between number of circulating endothelial stem cells (ESC) expressed as a percentage of the total number (CD34+) of circulating stem cells, and high-resolution CT scan of the chest score of pulmonary emphysema.

1 s/forced vital capacity (FEV $_1$ /FVC) ratio 56.8 (2.7)%) whereas 39.5% (n = 15) had normal lung function (FEV $_1$ /FVC ratio 77.1 (1.4)%).

The presence and the severity of pulmonary emphysema was determined using highresolution CT (HRCT) scans of the chest with density mask and the National Emphysema Treatment Trial Research Group score (0-4).4 The mean (SD) HRCT score was 1.7 (0.4). Blood sampling and flow cytometry were performed as previously described.<sup>5</sup> Briefly, quantification of peripheral blood CD34+ cells was performed with double labelling with FITC-anti-CD45 and phycoerythrinanti-CD34 monoclonal antibodies (Becton Dickinson, Milan, Italy) on a FACSCalibur flow cytometer (Becton Dickinson) according to standardised procedures. Enumeration of endothelial stem cells was performed as CD34 + cells co-expressing CD133 and VEGF-2. It was performed on immunomagnetically purified peripheral blood CD34+ cells (Miltenyi Biotech, Bologna, Italy) by triple labelling with peridinin chlorophyll protein-conjugated anti-CD34, phycoerythrin-conjugated anti-CD133 (Miltenyi Biotech) and unconjugated anti-VEGFR-2 (Santa Cruz Biotechnology, Milan, Italy), followed by FITC-conjugated swine anti-rabbit (Dako, Milan, Italy) as secondary reagent.

We found a significant correlation between the absolute number of circulating CD34+ cells and the absolute number of circulating endothelial stem  $(r^2 = 0.593, p < 0.0001; see figure 1 in online$ supplement). There was also a significant correlation between the percentage of circulating endothelial stem cells and the number of pack-years smoked ( $r^2 = 0.42$ , p<0.05; see figure 2 in online supplement). No significant correlation was found between total and endothelial stem cell numbers and HRCT score of pulmonary emphysema (figure 1), lung function data or smoking status (current vs ex-smokers). These data indicate that the number of circulating endothelial stem cells is not related to the presence and/or severity of the pulmonary emphysema or the presence or absence of COPD.

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### Competing interests None.

**Ethics approval** The study was approved by the local ethics committee of the University Hospital of Ferrara and written informed consent was obtained from each participant in accordance with the principles outlined in the Declaration of Helsinki.

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### **REFERENCES**

- Kasahara Y, Tuder RM, Cool CD, et al. Endothelial cell death and decreased expression of vascular endothelial growth factor and vascular endothelial growth factor receptor 2 in emphysema. Am J Respir Crit Care Med 2001;163:737—44.
- Yamada M, Kubo H, Kobayashi S, et al. Bone marrowderived progenitor cells are important for lung repair after lipopolysaccharide-induced lung injury. J Immunol 2004;172:1266—72.
- Global Initiative for Chronic Obstructive Lung
  Disease (GOLD). Global strategy for the diagnosis,
  management and prevention of chronic obstructive
  pulmonary disease. NHLBI/WHO workshop report. NIH
  Publication No 2701A, March 2001 (last update 2008).
  http://www.goldcopd.com (accessed 9 June 2009).
- National Emphysema Treatment Trial (NETT)
   Research Group. Patients at high risk of death after lung-volume-reduction surgery. N Engl J Med 2001;345:1075—83.
- Valgimigli M, Rigolin GM, Fucili A, et al. CD34+ and endothelial progenitor cells in patients with various degrees of congestive heart failure. Circulation 2004;110:1209—12.

## **Images in Thorax**

# Massive thymic hyperplasia presenting with respiratory insufficiency in a 2-year-old child

A 2-year-old boy presented with fever, dry cough and dyspnoea for 3 days. Breath sounds were absent and thoracic percussion revealed dullness in the lower two-thirds of the left hemithorax. A chest x-ray showed almost complete opacification of these areas (figure 1). The boy was successfully treated with antibiotics for lower airways infection but tachypnoea persisted and the x-ray showed no change after 6 weeks. A CT scan was performed and showed a heterogeneous mass in the anterior mediastinum, predominantly on the left side, occupying most of the left hemithorax. This lesion showed areas of lower attenuation interspersed with areas of soft tissue attenuation (figure 2). The possibility of a germinative cell tumour was considered so  $\alpha$ -fetoprotein and  $\beta$ -human chorionic gonadotropin were measured but were found to be within normal limits. A percu-



**Figure 1** Chest x-ray showing opacification of the lower two-thirds of the left hemithorax. Part of the left upper lobe is still aerated.

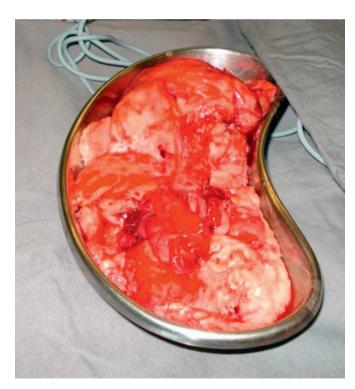
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Figure 2 CT scan of the chest. (A) Mediastinum and (B) lung windows showing a heterogeneous mass with areas of low attenuation interspersed with areas of soft tissue attenuation in the anterior mediastinum, occupying most of the left hemithorax.





taneous biopsy of the lesion was performed using a 14-gauge Tru-cut needle and fragment analysis was compatible with thymic tissue. Despite orientation for oral corticosteroids, the child did not receive the medication and the family did not come back for follow-up. Six months later the boy developed intense dyspnoea, cyanosis and other signs of respiratory insufficiency and was brought to our hospital. A new chest x-ray showed complete opacification of the left hemithorax with mediastinal shift towards the right. Surgery was indicated based on the respiratory condition, with removal of 830 g of homogeneous pale tissue. Microscopy findings showed preservation of the normal thymic architecture. The appearance was compatible with true thymic hyperplasia (figure 3). Postoperative recovery was uneventful and the patient was discharged from the intensive care unit on the third postoperative day with a normal respiratory pattern.



**Figure 3** Homogeneous whitish tissue removed during surgery, compatible with thymic hyperplasia.

Massive true thymic hyperplasia is a rare benign entity. Although no specific data have been published, it is believed that its incidence in Brazil is the same as that reported in other parts of the world. It can be asymptomatic or can present with symptoms resulting from airway and lung parenchymal compression. True thymic hyperplasia corresponds to thymic enlargement without alteration of normal histology and architecture. It is different from thymic hyperplasia associated with myasthenia gravis and autoimmune diseases where lymphofollicular hyperplasia occurs, usually not accompanied by a marked increase in thymic size. 12

Anterior mediastinal masses are usually asymptomatic but occasionally produce symptoms related to compression of adjacent structures. These lesions infrequently can cause acute lifethreatening respiratory compromise. Although rare, true thymic hyperplasia should be considered in the differential diagnosis of anterior mediastinal masses in children and young adolescents. Learning points

- 1. True thymic hyperplasia should be considered in the differential diagnosis of anterior mediastinal masses in children and young adolescents.
- 2. Anterior mediastinal masses can occasionally produce symptoms related to compression of adjacent structures.

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## **REFERENCES**

- Linegar AG, Odell JA, Fennell WM, et al. Massive thymic hyperplasia. Ann Thorac Surg 1993;55:1197—201.
- Rice HE, Flake AW, Hori T, et al. Massive thymic hyperplasia: characterization of a rare mediastinal mass. J Pediatr Surg 1994;29:1561—4.

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