and accurate information are essential to a curative surgical outcome.

References

Idiopathic Pulmonary Vein Thrombosis: A Rare Cause of Massive Hemoptysis
Gerard R. Alexander, MBChB, Anu Reddi, FCS (Cardio), and Darshan Reddy, MBChB
Department of Cardiothoracic Surgery, Inkosi Albert Luthuli Central Hospital, University of KwaZulu-Natal, Durban, South Africa

The case history of an adult female with massive hemoptysis due to idiopathic left inferior pulmonary vein thrombosis necessitating lower lobectomy is presented with a review of the current literature.

Massive hemoptysis in our environment is almost always due to inflammatory lung disease (ie, active tuberculosis or bronchiectasis) and very rarely other causes. Irrespective of the cause, when the disease is localized to the lobe or lung in an otherwise suitable surgical candidacy, after attention to contributory factors, lung resection is the preferred modality of treatment, with few exceptions.

A 47-year-old African female was referred to our thoracic surgical unit with a history (3 days previously) of massive hemoptysis associated with left chest pain and mild dyspnea. There was no surgical history noted. Clinical examination was noncontributory, except for digital clubbing, decreased air entry, and crackles in the left base posteriorly. The chest roentgenogram (Fig 1) showed homogenous opacification of the left lower lobe with concomitant high-resolution computed tomographic scan (HRCT) features of air-space consolidation of the same lobe (Fig 2). Laboratory investigation revealed anemia with a hemoglobin level of 8.3 g/dL, a leukocytosis of 30.07 × 10^9/L, an ESR of 91, albumin of 33 g/L, a normal urea, electrolytes, and coagulation profile. She was deemed suitable for management by lung resection and a left lower lobectomy was undertaken with relative urgency. Thoracotomy was immediately preceded by bronchoscopy, which confirmed our suspicion of bleeding from the left lower lobe. No other pathology was noted endoscopically. At operation the lobe was consolidated and congested. The inferior pulmonary vein was anatomically normal in site and size, and except for the acute thrombus internally, the vessel was structurally normal. In addition, there was no other obvious pathology (eg, inflammatory fibrosis) in close proximity to the vessel, whereas division of the pulmonary artery and bronchus was unremarkable. Macroscopic cut-sections of the lung parenchyma demonstrated red hepatization with thrombosis of the pulmonary venous system. The postoperative course was uneventful. On histology of the resected lobe, features of a recent hemorrhagic infarction were seen. There was no evidence of tumor in the pulmonary veins. In view of the operative and micro-
scopic findings, an echocardiogram was undertaken that showed a normal left atrium with no evidence of a thrombus. In addition, the mitral valve leaflet tips were mildly thickened with normal mobility, the left ventricular contractility was good, and the pulmonary systolic pressure was 31 mm Hg. At the time of this writing, a work-up for an underlying pro-thrombotic state was ongoing.

Comment

Based on our scouring of published articles, spontaneous or idiopathic pulmonary vein thrombosis (PVT), as a cause of massive hemoptysis necessitating lung resection is unrecorded in the Anglophone scientific literature. As the entity is relatively unknown and inadequately described in traditional texts, the review that follows attempts to address some pertinent areas of interest (the notation “spontaneous” or “idiopathic” is prefixed to PVT in this case, as no precipitating cause could be identified).

The PVT has been documented after pulmonary resection [1], lung transplantation [2], and radiofrequency ablation for atrial fibrillation [3]. Direct extension of the tumor into proximal pulmonary venules with subsequent stasis and thrombosis of larger distal veins may mimic primary PVT [4].

The pathophysiology of PVT is analogous to that of mitral stenosis [5, 6]. A significant decrease in the mitral valve area causes an increase in the left atrioventricular gradient and pulmonary venous pressure with compensatory pulmonary arteriolar vasoconstriction. “Leaky capillaries,” interstitial edema, and increased uptake by lymphatics through the mechanism of Starling forces occur. Elevated pulmonary artery pressure, right ventricular end-diastolic pressure, and right ventricular dilatation may ultimately result.

In animal studies, Wyatt and colleagues [5] described the sequential changes in the lung after ligation of the pulmonary veins (ie, congestion, edema, extravasation of serum, and blood in the alveoli resulting in total consolidation).

Hurwitz and colleagues [6] documented the development of post-alveolar bronchial venous channels after ligation of the pulmonary veins. Oxygen saturation in the azygos veins was noted to be similar to that of systemic arterial oxygen content (right-to-right shunt). Histology at 3 to 6 months revealed complete resolution of the consolidation, possibly due to re-canalization of the thrombosed pulmonary vein. Other compensatory mechanisms also seen include the development of dense vascular adhesions between lung and the chest wall [6].

The clinical presentation of PVT may be nonspecific, with symptoms such as dyspnea, cough, hemoptysis, pleuritic chest pain, and radiographic features of consolidation of the lobe or lung [1]. After lobectomy, thrombosis commonly occurs early in the postoperative period [1]. In lung transplantation, 15% occurs within 48 hours postoperatively, with features of pulmonary edema, pulmonary infection, or infarction making distinction from acute rejection or reperfusion injury a vexation [2].

Peripheral embolization to cerebral, coronary, or limb circulations from fragmentation of thrombus within the pulmonary veins may result in well-recognized clinical features of organ ischemia. The source of the emboli may however produce a clinical dilemma [7].

The diagnosis of PVT, as noted in previous publications, is almost always tentative and established retrospectively after lung resection or autopsy. However, it may be suspected from a radiographic series that shows...
Inadvertent Total Spinal Anesthesia After Intercostal Nerve Block Placement During Lung Resection

Babar B. Chaudhri, FRCS, Alistair Macfie, FRCA, and Alan J. Kirk, FRCS

Department of Cardiothoracic Surgery, Royal Infirmary of Edinburgh, Edinburgh, and West of Scotland Heart and Lung Centre, Golden Jubilee National Hospital, Glasgow, United Kingdom

Intercostal nerve block is a recognized way of providing analgesia at thoracotomy. There is a rare association between intercostal nerve block and the complication of total spinal anesthesia. This may arise inadvertently by injection into a dural cuff extending outside the intervertebral foramen. We report our experience with a patient who sustained this life-threatening complication. The patient required postoperative ventilation until the neurologic deficits resolved. The operator must be aware that intercostal nerve block runs the rare but potentially fatal risk of total spinal block.

(Ann Thorac Surg 2009;88:283–4) © 2009 by The Society of Thoracic Surgeons

Dissemination of local anaesthetic agent is possible from the site of injection away from its intended target and may result in serious complications [1, 2]. We report a case of total spinal anesthesia after placement of intrathoracic intercostal nerve blocks at the time of lung resection.

A 66-year-old man was scheduled for left thoracotomy and anatomic lung resection for a left lower lobe mass that was highly suspicious of non-small cell lung cancer (NSCLC). A computed tomography (CT) scan of the chest showed a 2- × 2-cm mass in the periphery of the left lower lobe. There was no evidence of mediastinal lymphadenopathy. A CT scan of the upper abdomen was unremarkable, and a CT scan of the brain showed no intracerebral lesions. The patient’s forced expiratory volume in 1 second was 2.1 L and forced vital capacity was 2.8 L. There was no significant past medical history.

Temazepam was given as premedication, and anesthesia was induced with isoflurane and vecuronium. Intubation was performed with a medium-sized, right-sided Robertshaw double-lumen tube. An epidural catheter was placed, and 4 mL of 0.5% bupivacaine was administered before induction. Anesthesia was maintained with isoflurane, remifentanil, and vecuronium.

Surgical entry into the chest was by a standard left thoracotomy through the bed of the fifth rib. The serratus

References


© 2009 by The Society of Thoracic Surgeons
Published by Elsevier Inc

Accepted for publication Sept 29, 2008.
Address correspondence to Dr Chaudhri, Department of Cardiothoracic Surgery, Royal Infirmary of Edinburgh, Little France Cr, Edinburgh, EH16 4SA, United Kingdom; e-mail: bchaudhri@mac.com.

© 2009 by The Society of Thoracic Surgeons
Published by Elsevier Inc