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CASE REPORT

Uta Jenny-Möbius · Elisabeth Bruder
Thomas Stallmach

Recognition and significance of pulmonary bone embolism

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Abstract Embolism of bone marrow to the lungs is a quite frequent finding after trauma but transport and deposition of solid bone is rarely seen, which may simply be because pulmonary calcifications are not recognized as bone fragments. We report on three patients with embolism of bone spicules to small lung arteries of about 0.5 mm in diameter which were plentiful in two of the patients on postmortem examination. However, the true nature of the emboli was only recognizable after decalcification of lung tissues.

It appears that trauma occurring in a septic bone lesion has the greatest chance to provoke bone embolism. The bone spicules do not usually occlude vessel lumina and thus do not severely disturb the blood circulation in the lungs. The bone fragments become covered by endothelium and can remain recognizable for months or even years.

Key words Trauma · Calcification · Bone · Embolism · Autopsy

Introduction

Lung capillaries function as a sieve in the blood circulation and various materials are trapped there. The most common finding are thrombi which have been formed somewhere else and transported with the bloodstream (Dickens et al. 1997). Air bubbles (Bajanowski et al. 1998) and fat droplets (Brinkmann et al. 1976) are frequently seen after trauma and after lymphography, oily contrast medium has been detected in lung capillaries (Brunner et al. 1974). After disaster accidents, embolisms with skin (Andrew 1976), cartilage (Lau 1995), liver (Nunes 1971; Michalodimitrakis and Tsatsakis 1998), or even buckshot (Choi et al. 1994) have been observed. Bone marrow embolism is quite common after trauma, reanimation procedures and bone surgery, but embolism of bone is a very rare event and only four reports could be found in the medical literature (Zichner 1970; Bras and Veraart 1980; Abrahams and Catchatourian 1983; Nagy 1983). During post-mortem examinations, we recently identified bone spicules disseminated within the vasculature of the lungs in three cases. The underlying causes for the bone embolism in these cases and in the cases from the literature are presented, and the significance of this finding is assessed.

U. Jenny-Möbius · E. Bruder · T. Stallmach (✉)
Department of Pathology, University Hospital Zürich,
Schmelzbergstrasse 12, CH-8091 Zürich, Switzerland
e-mail: thomas.stallmach@pty.usz.ch
Tel. +41-1-255 25 16; Fax. +41-1-255 44 16

Case reports

Case 1

A 76-year-old man was admitted to hospital because of poor general condition with dyspnoea and complained of pain in the thoracolumbar region. X-ray examination revealed severe osteoporosis of the vertebral column and an osteolytic lesion at L1 (Fig. 1). Laboratory parameters indicated sepsis and *staphylococcus aureus* was identified in blood cultures. Despite antibiotic treatment, the patient died 5 days later. On postmortem examination, a purulent spondylodiscitis was found with nearly complete destruction of the vertebral body L1 (Fig. 2) with abscess formation in the epidural space and along the M. psoas. Histological examination of the lungs revealed multiple intravascular "calcium deposits". After brief decalcification, the morphology was such that all calcium deposits could be identified as small fragments of lamellar bone.

Case 2

8-month-old child with cyanotic cardiac malformation was operated on twice. A few days after birth, a Blalock-Taussig shunt was performed to maintain blood circulation to the lungs. At the age of 5 months, a Hemifontan operation was carried out to improve oxygenation of the blood. The thorax had to be opened for a third time because of sternum osteomyelitis. The child had several episodes of sepsis and furthermore, a massive intracerebral hemorrhage occurred. Because of the poor neurological performance, only minimal supportive therapy was administered and the child died 3 months after the second operation. On postmortem examination, the lungs were found to be atelectatic due to chylothorax on both

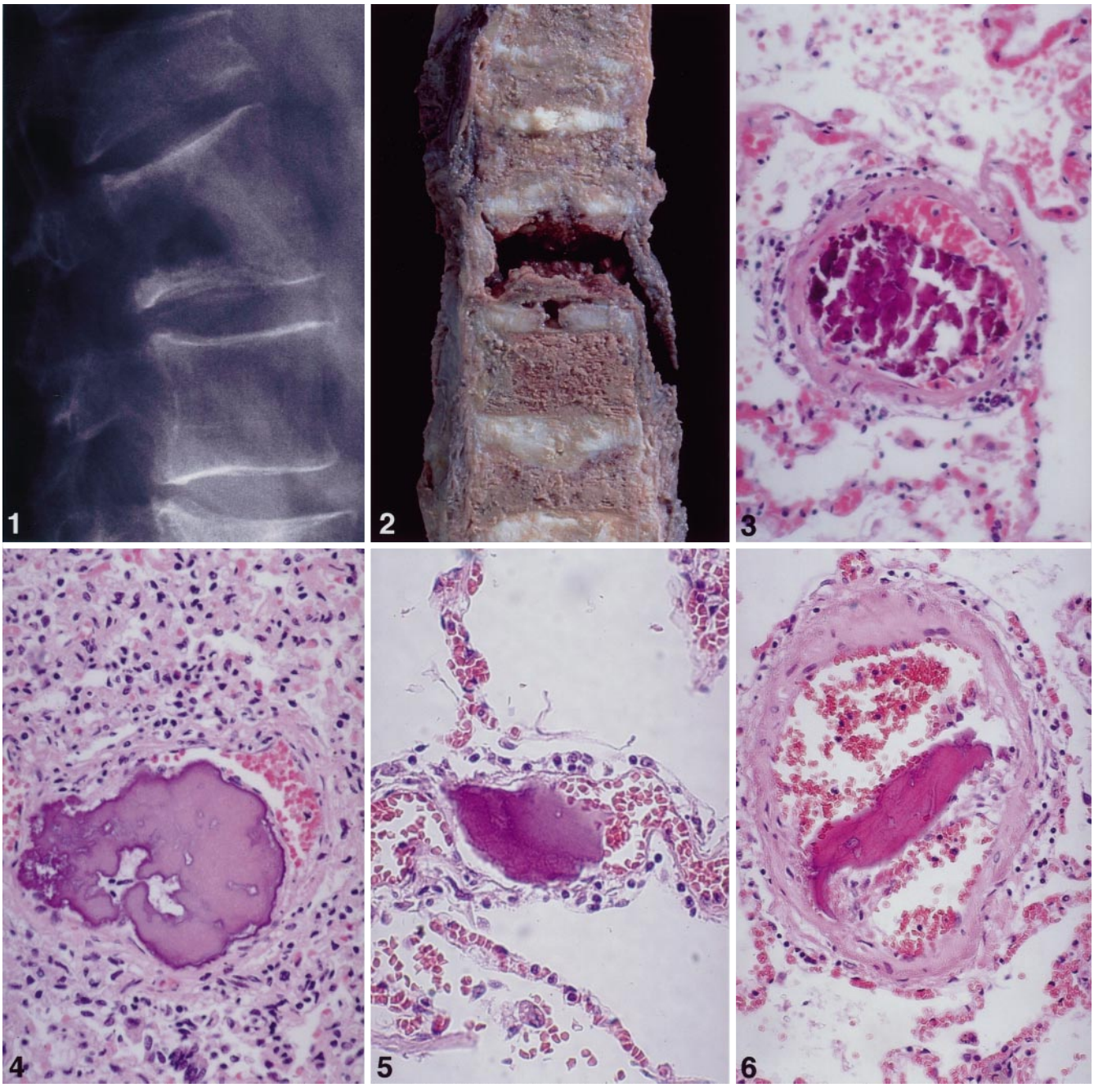

Fig. 1 X-ray of lumbar spine in case1: lateral view of L1 shows osteolytic lesion with sclerosed rim and impression fracture

Fig. 2 Frontally hemisected vertebral column in case 1 with near complete destruction of vertebral body L1 caused by osteomyelitis

Fig. 3 Lung artery with crushed mineralized material. Hematoxylin-eosin × 250

Fig. 4 Intravascular bone is revealed after decalcification because typical osteocyte lacunae become detectable. Hematoxylin-eosin × 250

Fig. 5 Bone fragment within lung capillary, a rare finding due to the usually greater size of bone particles. Hematoxylin-eosin × 400

Fig. 6 Bone spicule that became lodged and has been covered completely by endothelium. Due to this mode of retention some of the vessel lumen remains open and allows the blood circulation to continue. Hematoxylin-eosin × 250

sides. Histological examination of the lungs revealed bone spicules in small sized arteries which were only detectable after decalcification of the tissue.

Case 3

A 68-year-old man was admitted to hospital because of severe abdominal pain. He was in a neglected condition and reported to be an alcoholic. During the preliminary diagnostic procedures, the patient had an epileptic attack and after that remained in a coma. An aspiration pneumonia was suspected and despite antibiotic treatment the patient died 2 days later. On postmortem examination, a severe alcoholic liver damage was found plus a massive aspiration pneumonia. Histological examination revealed a few lamellar bone particles within small arteries of the lungs.

Materials and methods

In our routine, each lobe of the lung is examined using one block of about 2.5 × 2 cm. In case 1 (spondylodiscitis), bone emboli were seen within each block and within each low power field on microscopic examination. In case 2 (baby after cardiac surgery), bone emboli were seen in three blocks and in about every 10th. field under low power microscopy. In case 3 (liver cirrhosis and neglect), there were only very few bone emboli which were difficult to find.

In all these cases, the routine histological slides stained with hematoxylin-eosin only contained amphophilic fragmented material (Fig. 3) which was clearly intravascular and sometimes surrounded by endothelium. Initially, the findings were interpreted as calcified remnants of old thrombemboli. The morphology was such that lamellar structure and osteocyte lacunae could only be recognized after short decalcification (Figs. 4–6). For the purpose of decalcification, formalin-fixed tissue or the paraffin blocks, which had already been used to cut the undecalcified slides, were put into a solution of 10% EDTA for 24 h. Nearly all of the bone particles were situated within small arteries with a diameter of less than 0.5 mm and had rarely reached the capillaries (Fig. 5). None of the small arteries appeared to be completely occluded by the splinters. As a typical feature, bone spicules seemed to have penetrated the endothelium and became lodged in the vessel wall. Inflammation or fibrosis was hardly ever seen in the surrounding tissues (Fig. 6).

Discussion

Embolism of bone marrow has been reported in 6% of patients with trauma (Rappaport et al. 1950) and it was even seen in 7% of 620 unselected autopsies (Kemona et al. 1989). In about 12% of the patients with marrow embolism, bone fragments have additionally been observed (Zichner 1970). Embolism of lamellar bone chips has been documented in two patients after implantation of a hip endoprothesis who died 3 months and 2 years after the operation respectively, and the bone particles were found at autopsy (Bras and Veraart 1980). In a series of 9000 autopsies only one case was seen (Nagy 1983) and 12 cases have been reported after bone marrow transplantation (Abrahams and Catchaturian 1983).

From the clinical history of cases 1 and 2, it appears that bone embolism is the consequence of an inflammatory disease within bones. The old man suffered from an acute suppurative osteomyelitis. In the infant, osteomyelitis was chronic at the time of autopsy but obviously had been suppurative 3 months before. However, in both cases, trauma cannot be excluded as the primary cause for the embolism of bone fragments. X-ray examination had shown a fracture line within the vertebral osteolysis of the first patient and the infant had a repeated sternotomy. All patients reported in the literature were traumatized and received the injuries from 1 day up to 2 years before death. Thus, inflammation is not a necessary prerequisite for bone embolism, however, it might promote it by breaking up the bone structure. In the third case in our series septicaemia was present in the terminal phase of the illness but no inflammatory lesions could be detected in the skeleton. From the previous history it is quite likely that trauma had occurred before and even fractures. Neither in our patients nor in the cases reported in the literature was bone embolism considered to be an element contributory to death. However, it is quite clear that bone embolism is a forensically important sign of vitality. It has, furthermore, been pointed out (Schinella 1973) that bone embolism should draw the attention to trauma, which has gone undetected before. Attention should be paid to the fact that bone embolism remains a recognizable feature for a long period of time. It seems that the blood circulation in the lungs is not seriously disturbed by this type of embolism, maybe, because bone chips hardly ever occlude the vessel lumen. Without decalcification, intravascular bone is crushed to such an extent that in most cases it will not be recognized as bone. Short decalcification will reveal the true nature of the finding.

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