CASE REPORT

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Cerebral Tissue Pulmonary Embolization Due to Head Trauma: A Case Report with Immunohistochemical Confirmation

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ABSTRACT: Pulmonary embolization of cerebral tissue as the result of severe head trauma is an uncommon, if not rare, phenomenon, and few cases have been reported in the literature. The authors discuss the case of a 51-year-old male who died six days after suffering extensive head trauma in a motor vehicle collision. At autopsy, white-gray emboli were found in several subsegmental pulmonary arteries. The results of histologic examination with the hematoxylin-eosin stain gave the impression that the emboli were necrotic cerebral tissue; however, routine special stains for neural tissue produced inconclusive results. Immunohistochemical staining of the emboli with monoclonal mouse anti-human neurofilament protein (Dako Corp., Carpinteria, California) confirmed the cerebral nature of the emboli. To the authors' knowledge, this is the first reported case of pulmonary embolization of cerebral tissue confirmed by immunohistochemistry.

KEYWORDS: pathology and biology, embolisms, immunohistochemistry, tissues (biology), cerebral tissue

Case History

A 51-year-old male unrestrained driver received multiple blunt head injuries as the result of a motor vehicle collision in which his car was hit broadside on the passenger side in an intersection by another vehicle traveling at a high rate of speed. In spite of immediate emergency medical care and neurosurgical intervention, the patient died on the sixth hospital day. At the request of the county coroner, an autopsy was performed at the office of the chief medical examiner.

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Autopsy Findings

External examination revealed a superficial occipital scalp laceration, bilateral periorbital hematoma, and multiple contusions and abrasions in various stages of healing on the back, chin, arms, and thighs.

Internal examination showed a right temporo-occipital linear skull fracture, thin layers of residual subarachnoid and subdural extravasated blood, and laceration of the right occipital lobe with contrecoup contusion of the left frontal pole. An intracranial pressure monitor was in place, and marked cerebral edema was evident, with widening and flattening of the gyri, herniation of the cerebellar tonsils, uncal grooving, and secondary (Duret) hemorrhages of the midbrain and pons. Sectioning of the lungs revealed multiple friable thromboemboli, which were remarkable for their white-gray color (Fig. 1).

Microscopy Results

Routine sections of hematoxylin and eosin preparations of lung tissue showed apparent necrotic cerebral tissue rimmed by fibrin within branches of the pulmonary arteries. Customary special stains for neural tissue, such as Luxol fast blue, phosphotungstic acid hematoxylin, and glial fibrillary acidic protein, produced equivocal results (Fig. 2). Immunohistochemical staining of formalin-fixed, paraffin-embedded lung sections with monoclonal mouse anti-human neurofilament protein (Dako Corp., Carpinteria, California), a monoclonal antibody specific for the 70 and 200 kilodalton components of the three major polypeptide subunits present in neurofilaments [*1*], demonstrated the neural origin of the emboli (Fig. 3).

Discussion

Cerebral tissue pulmonary embolization due to trauma in adults is rare. Merkel first described the phenomenon in 1926 in a 47-year-old male who fell 15 ft (4.6 m) from a ladder [2]. The initial appearance of the entity in the English language literature took

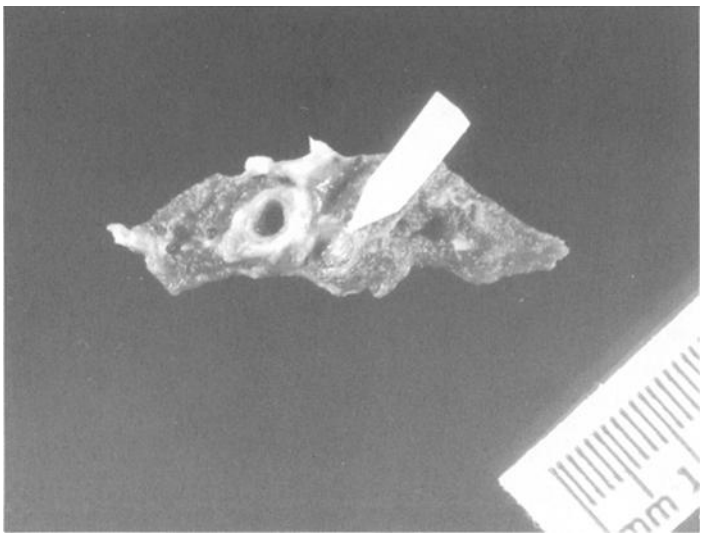

FIG. 1—*Portion of the right lower lobe of the lung showing a white-gray embolus in a branch of the pulmonary artery* (arrow).

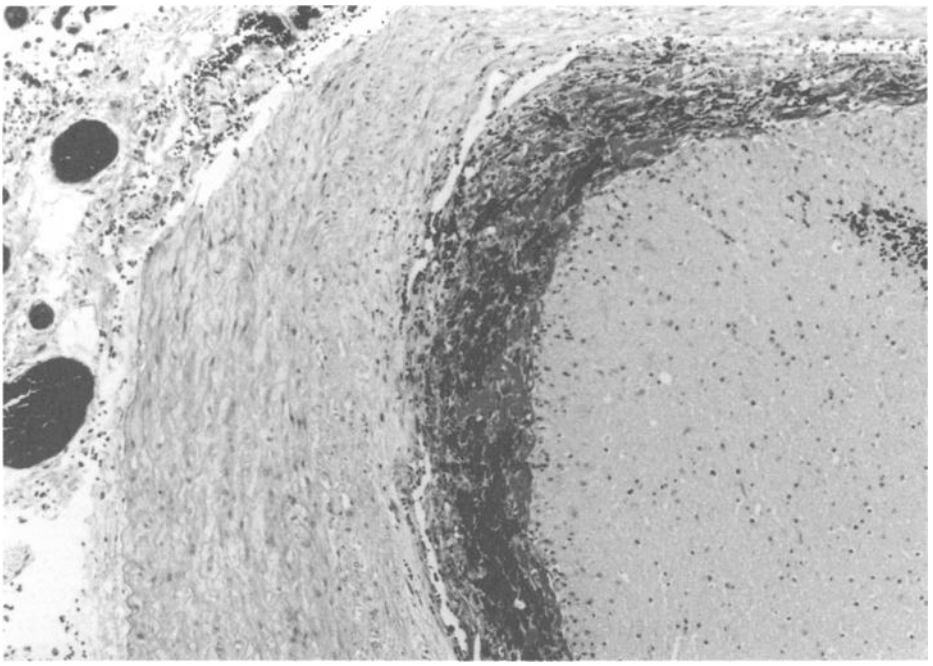

FIG. 2—*Section of the pulmonary artery filled with apparent necrotic brain tissue* (right). *The pulmonary arterial wall is at center (stain, Luxol fast blue; magnification, ×100).*

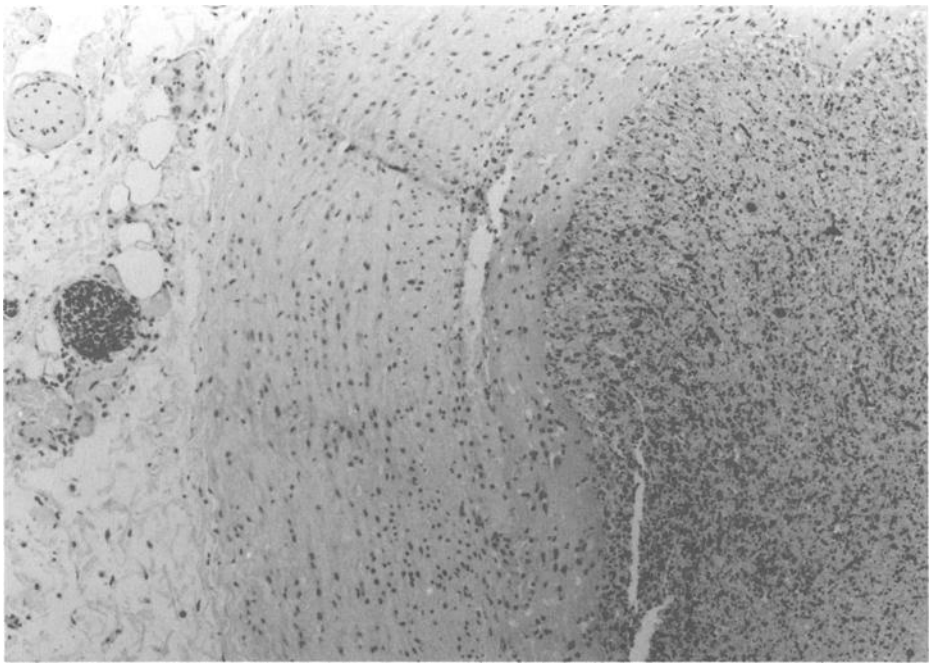

FIG. 3—*Section of pulmonary artery stained with monoclonal neurofilament protein stain, demonstrating the cerebral origin of the embolus. The cross section of a peripheral nerve* (left) *serves as a positive stain control (stain, monoclonal neurofilament protein; magnification, ×100).*

place in 1936, when Krakower reported brain tissue surrounded by a layer of dense, laminated fibrin in a 23-month-old boy who had fallen 12 ft (3.7 m) onto a concrete floor, rupturing the superior sagittal sinus [*3*]. While Tedeschi and Hechtman [*4*] found 15 cases reported up through 1967, the current authors have found only 3 cases reported since then that are not associated with birth injury [*5–7*]. Pulmonary embolization of brain tissue as a result of birth trauma, usually complicated by breech presentation, has been reported in 12 cases [*8,9*]. In one case, "paradoxical" brain emboli in a coronary artery were noted as having been due to a patent foramen ovale [*8*].

Oppenheimer [*10*] retrospectively reviewed 277 autopsies, performed over a 25-year period, on patients who died after intracranial surgery. No brain tissue was found in the pulmonary vasculature in any of these cases, even in the subset of 30 cases in which decompression operations were performed after head injury. In the same period, a review of 42 autopsies of fatalities due to head injuries without surgical intervention uncovered no case of pulmonary embolism of cerebral tissue [*10*]. In another 18-year review of 213 autopsies on patients with head injuries, only 4 cases showed cerebral tissue emboli, all in individuals who had suffered skull fracture and brain laceration [*11*]. Only 2 of these 4 had had intracranial surgery. No embolization of brain tissue was seen in 24 additional cases over three years of patients dying after intracranial surgery for nontraumatic disease [*11*].

The mechanism of entry of cerebral tissue into the venous system with subsequent distribution to the lungs is unclear. Bohm et al. clearly state that "brain tissue embolism is only possible if a large venous sinus has been ruptured" [*8*]. The experience of Tackett [*12*], Valdes-Dapena and Arey [*13*], and the current authors would indicate otherwise, for, in their combined four cases, dural sinus tears were not identified. The authors believe that access to the venous circulation was obtained through smaller veins.

The authors' initial frustration in being unable to prove unequivocally the cerebral nature of the pulmonary emboli was shared by McMillan [*11*]. In his four cases, special neural stains, such as iron hematoxylin, myelin sheath, Bodian's, and glial fiber, produced negative results, and the Holzer stain produced only faint positivity in two of the four cases. These inconclusive results were attributed to the emboli's small size and the post-mortem changes within the emboli [*11*]. In the present case, necrosis of embolic brain tissue in the six-day interval between the trauma and death may have contributed to the ineffectiveness of the initial stains.

The basis for the fibrin deposition about the cerebral tissue emboli observed by Krakower [*3*] and the current authors is clearer. Brain tissue is rich in tissue factor (thromboplastin), a glycoprotein which is a primary physiologic initiator of coagulation [*14*].

Conclusions

Pulmonary embolization of cerebral tissue as the result of severe head injury is rare, with an estimated incidence of 2% [*9*]. Assiduous search with careful serial sectioning of lungs may increase this percentage, and special procedures may be necessary to prove the cerebral nature of the emboli. To the authors' knowledge, this is the first reported case of pulmonary embolization of cerebral tissue confirmed by immunohistochemistry.

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