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A Retrospective and Prospective Study of Cerebral Tissue Pulmonary Embolism in Severe Head Trauma

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ABSTRACT: In an attempt to determine the incidence of pulmonary embolization of cerebral tissue as the result of severe head trauma with and without dural penetration, we have collected the autopsy reports at our institution of all head injury victims over the past 3.5 years. The retrospective and prospective histologic examination of these cases revealed a total of 10% to have emboli of cerebral tissue within the pulmonary vasculature. Immunohistochemical staining of the emboli confirmed the neural origin of the tissue. The dura mater proved to be intact in 70% of the cases of cerebral embolization. We feel this provides evidence that embolization of cerebral tissue may occur without rupture of the dura and/or large venous sinuses. This observation is of clinical as well as forensic importance, as cerebral tissue emboli are a rich source of thromboplastin in the systemic and pulmonary vasculature and may contribute to the morbidity and mortality of head injuries.

KEYWORDS: pathology and biology, cerebral tissue pulmonary embolism, trauma, head trauma, injury

Pulmonary embolization of cerebral tissue following severe head trauma is uncommonly reported at autopsy. McMillan, for example, has reported an incidence of 2% [*1*]. The first reported case, by Krakower in 1936, was in a 23-month-old boy who feel 12 feet onto a concrete floor [*2*]. In these reported cases, laceration of the dura was considered to be the mechanism through which the brain tissue gains access to the venous vasculature. Authors have also reported brain tissue embolization in newborn infants secondary to a traumatic delivery or postnatal trauma [*3–5*]. Oppenheimer and McMillan have documented cases in adults with head injury [*1,6*]. Other than these reports, however, no one to our knowledge has retrospectively and prospectively reviewed autopsies with severe head trauma to determine the incidence of brain tissue embolism to the pulmonary vasculature. We provide such a review in this paper. In addition, we provide immunohistochemical confirmation and determination of the presence or absence of dural laceration.

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Materials and Methods

Beginning in March, 1992, we collected the autopsy reports of all severe head trauma victims from January, 1989 through June, 1992 at the Bowman Gray School of Medicine/ North Carolina Baptist Hospital and stained the histologic sections of lung with hematoxylin and eosin. For the purpose of this study, *severe head trauma* is defined as head trauma sufficient to cause death. In keeping with our usual autopsy procedure, all lungs were inflated with a 10% solution of neutral buffered formalin via cannulization of the trachea immediately above the carina. The lungs were allowed to fix for 30 min, serially sectioned in the parasagittal plane, and at least one 1.0 × 1.0 × 0.3 cm section submitted for paraffin embedding from each lobe, for a total of five lung sections examined histologically. We found ten out of 102 cases (10%) to have microscopic brain tissue emboli within the pulmonary vasculature (Fig. 1). Retrospective and prospective immunohistochemical staining of the histologic sections (neurofilament protein [Accurate Chemical and Scientific Corporation, Westbury, NY], S100 protein [Biomeda Corporation, Foster City, CA], and glial fibrillary acidic protein (GFAP) [Dako Corporation, Carpinteria, CA] confirmed the neural origin of the tissue (Fig. 2). Neurofilament antibody stains the 70 kilodalton and 200 kilodalton polypeptides of neurofilament; GFAP stains the glial fibers; and, S100 stains the neural antigen S100 protein.

Results

The ages of the ten victims who were positive ranged from 3 to 58 years; six were male, and four were female. The injury to death time interval ranged from instantaneous (that is, less than five minutes) to 21 and a half days. The causes of death included two cases of gunshot wounds, five of motor vehicle collisions, and three other blunt force injuries (Table 1). We subsequently searched the autopsy reports to determine whether

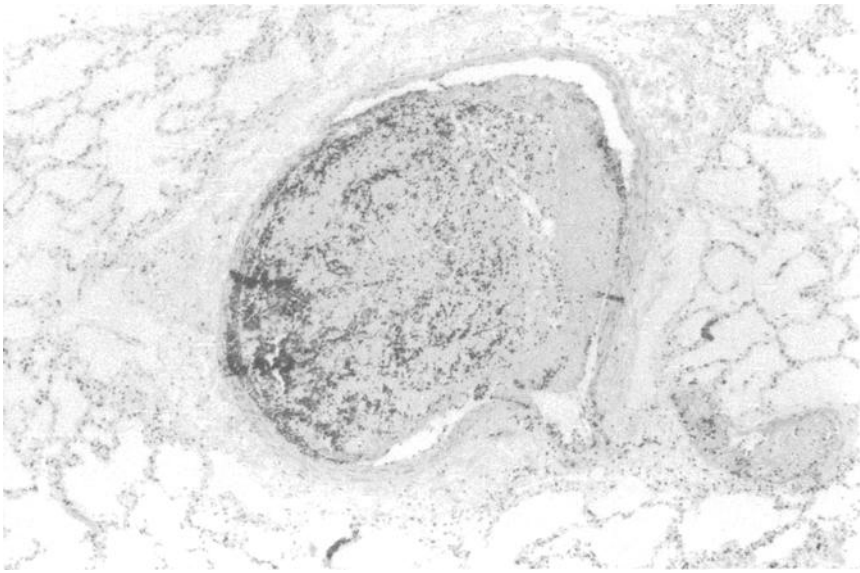

FIG. 1—*Histologic section of lung stained with hematoxylin and eosin shows brain tissue emboli within the pulmonary vasculature (Original magnification = 200x).*

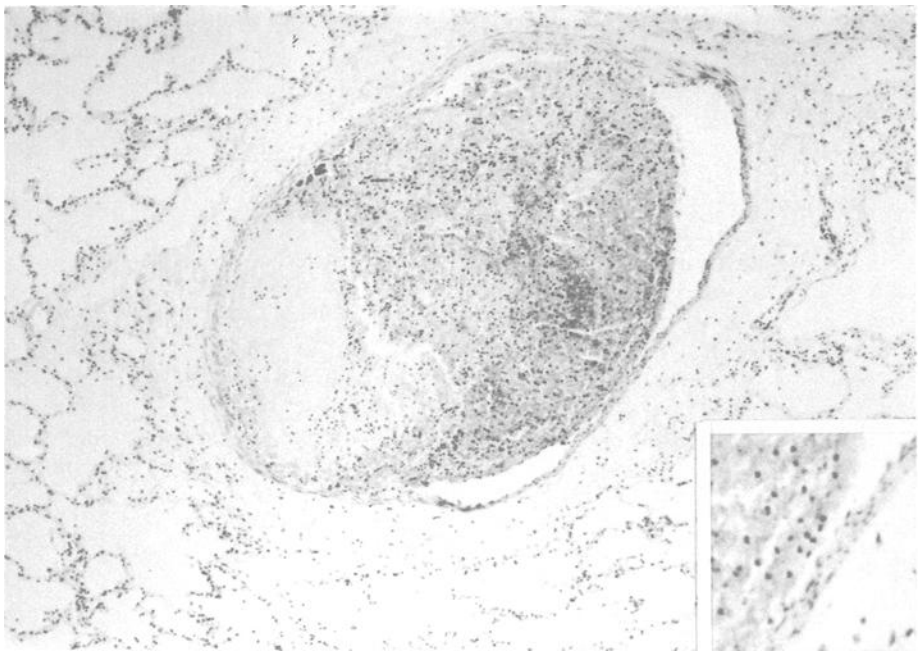

FIG. 2—*Neurofilament protein immunohistochemical staining the same section of lung confirms the neural origin of the tissue embolus (Original magnification = 200x). Inset shows higher power view of embolus (Original magnification = 400x).*

or not the dura was intact. In seven (70%) of the pulmonary embolization cases, the dura was intact (Tables 1 and 2).

Discussion

Upon review of the literature, most researchers estimate an incidence of brain tissue emboli to the pulmonary vasculature in cases of severe head trauma at 2% [*1*]. In contrast, we found the incidence in such cases to be 10%. One possible explanation for not finding an even higher incidence could be sampling error. Typically, brain tissue emboli were found in some but not all of the pulmonary lobes in the positive cases. The mechanism of entry of brain tissue into the venous system with subsequent distribution to the lungs is unclear [*7,8*]. Some believe that brain tissue embolization is possible only if the dura or a large venous sinus has been ruptured [*5*]. In our review and the reports of others, embolization may also result with an intact dura [*4,7,8*]. We believe that brain tissue may gain access to the systemic circulation via small central nervous system veins without laceration of the dura as a prerequisite.

Brain tissue embolization may have a significant impact on the premortem clinical management of the head trauma patient. Thromboplastin, a species specific plasma membrane glycoprotein and a primary physiologic initiator of coagulation, is present in most tissues and is particularly concentrated in cerebral tissue [*9*]. Furthermore, brain tissue is well known to cause plasma coagulation, shock, and consumptive coagulopathy upon direct contact with the blood stream [*5*]. Cerebral brain tissue emboli are, therefore, more than a postmortem curiosity and are of significant potential clinical importance.

TABLE 1—

Age/race/sex	Cause of death/ Type of head trauma	Condition of dura	Injury to death time interval
34WM	GSW head (x1)	Lacerated	Instantaneous
34WF	GSW head (x1)	Lacerated	Less than 12 hours
18WM	Blunt trauma (MVA)	Intact	Approximately 12 hours
18WF	Blunt trauma (MVA)	Intact	12.5 hours
29WF	Blunt trauma (MVA)	Intact	Instantaneous
15WF	Blunt trauma (MVA)	Intact	1 hour
3BM	Blunt trauma (pedestrian hit by car)	Intact	Less than 12 hours
58WM	Blunt trauma (fell off horse)	Intact	Approximately 12 hours
45WM	Blunt trauma (MVA)	Lacerated	2.3 hours
31BM	Blunt trauma (hit by brick)	Intact	21.5 days

Based upon our 10% incidence of brain tissue embolization secondary to severe head trauma, these hematologic events have the potential to play a significant role in the morbidity and mortality of head trauma patients. From a statistical and public health perspective, cerebral tissue pulmonary emboli should be sought in all autopsied cases of death due to head injury.

Conclusion

We have demonstrated a 10% incidence of cerebral tissue pulmonary embolization due to head trauma, previously thought to have an incidence of only 2%. We have also shown that the dura need not be lacerated in order for embolization to occur. Reporting this data, they alert the forensic community to this phenomenon of considerable clinical and pathologic importance.

TABLE 2—*Head trauma fatalities.*

Total number of cases (1989–1992)	102
Number of cases with brain tissue emboli	10
Number of cases with brain tissue emboli and intact dura	7

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