CASE REPORT

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Concentration of Alcohol in Delayed Subdural Hematoma

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ABSTRACT: The medicolegal implications of a delayed subdural hemorrhage are described, with particular reference to the significance of the alcohol content in the hematoma versus that in the peripheral blood at the time of injury.

KEYWORDS: pathology and biology, hematoma, alcohol

A hematoma found during the autopsy of a person injured shortly before death often lends itself to the determination of alcohol and drugs. However, certain limitations in interpreting the results of such analyses must be considered.

The following is a case report where extended consumption of alcoholic beverages preceded a traffic accident, yet alcohol was negative in a subdural blood clot evacuated at autopsy after 22 h of coma.

Case Report

A young woman was admitted comatose to the hospital minutes after she was struck by an automobile. She had been at a bar with her parents for about 4 h to celebrate her nineteenth birthday. While there she consumed alcohol at a relatively constant rate until approximately 30 min before the accident. Evaluation in the hospital emergency room included a blood alcohol concentration (drawn 1 h following the accident) of 0.12%. Skull X-rays showed linear left parietal and temporal fractures. Bilateral carotid angiography demonstrated no midline shift. She was treated conservatively with intravenous dexamethasone and mannitol to counteract brain swelling. Some gradual improvement of her condition became manifest during the following 15 h. Thereafter, signs of responsiveness and body temperature decreased, and the pupils became dilated and sluggish to light stimulation. Twenty-one hours following admission there was an episode of respiratory distress with subsequent cardiac arrest, unresponsive to resuscitation attempts.

At autopsy there was diffuse asymmetrical subdural hemorrhage, in addition to the left-

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sided skull fractures and bilateral cortical contusions with associated subarachnoid hemorrhage. Tests for alcohol in peripheral and subdural blood were negative.

Discussion

Craniocerebral injuries often result in intracranial hemorrhage, either subdural or intracerebral or combined. Epidural hemorrhage in adults is rare. In cases of prolonged survival, evidence for the presence of alcohol or drugs at the time of injury may be determined by analysis of a collection of blood produced and isolated at the time of injury [*1,2*]. Although some of the alcohol in the hematoma may be lost by diffusion, the blood concentration at the time of injury may at least be approximated. Subdural and intramuscular hematomas are frequently ideal depositories of such evidence, but certain reservations must be considered. In the case under discussion, the consumption of alcohol before the accident is well established by both clinical history and measurement of blood alcohol shortly after hospital admission. The subdural hematoma at autopsy, however, failed to reveal alcohol. The logical answer to this apparent discrepancy is that the subdural hemorrhage did not develop until the alcohol was eliminated from the peripheral blood.

Alcohol is dissipated by oxidation, diffusion, and excretion. The rate of decrease of blood alcohol may be 0.015% per hour or under some circumstances considerably faster [*3*]. It is reasonable to expect in this case, therefore, that the alcohol would have been eliminated from the blood within 15 h of hospitalization, during which time there was brain swelling without subdural hemorrhage. The clinical record indicates a worsening of the patient's condition from approximately 15 to 16 h after hospital admission. The subdural hematoma evacuated at autopsy does not contradict the laboratory test for alcohol but rather confirms the absence of subdural hemorrhage during the time that the blood alcohol was positive and supports the conclusion that subdural bleeding followed treatment and was delayed until the last hours of hospitalization. Accordingly, had the blood alcohol been 0.30% upon hospital admission, the subdural hematoma would probably have reflected the diminished blood concentration at the time of hemorrhage.

In this and similar cases, the rapid development of severe cerebral edema following closed head trauma may produce a tamponade effect so as to impede subdural bleeding. This effect is especially understandable when it is considered that most subdural hematomas are of venous rather than arterial origin. Upon supportive treatment of this brain swelling, the tamponade effect is reduced and delayed subdural hemorrhage supervenes. The clinical record in this case certainly supports this sequence. Blood which becomes isolated from the circulation in this way and is subsequently recovered at autopsy reflects the approximate blood alcohol concentration at the time of hemorrhage.

Reports in the neurosurgical literature of delayed intracranial hematoma following head injury indicate that this phenomenon is more common than usually recognized and is associated with a poor prognosis. Lipper et al, in a prospective study of patients with severe head injury, found that most delayed intracerebral hematomas occurred within 48 h and that most extracerebral hematomas developed within two weeks following trauma [*4*]. An understanding and appreciation of this phenomenon is important in medicolegal autopsies, so as not to put improper interpretation on the results of toxicologic analysis of autopsy specimens for the presence of alcohol or drugs at the time of injury.

The occurrence of delayed intracranial hemorrhage is not a new discovery, but there has been no published discussion of its medicolegal implications. Tests for alcohol and drugs on subdural hematomas discovered at autopsy may be useful in its recognition or confirmation. In cases of prolonged survival following injury, initially positive concentrations of alcohol and drugs may become negative, which is frequently the case in our experience. Finally, it is suggested that a note be made at the conclusion of the autopsy report in such cases explain-

ing the apparent discrepancy between the alcohol concentrations in the peripheral blood versus that in the subdural clot.

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