

## CASE REPORT

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# Motor-Vehicle Collision-Related Death Due to Delayed-Onset Subarachnoid Hemorrhage Associated with Anticoagulant Therapy\*

**ABSTRACT:** Delayed deaths following injury are not rare. Various mechanisms may be responsible for such deaths, including pulmonary thromboembolism, fat embolism, infection, systemic inflammatory response syndrome, and delayed hemorrhage. In the present case, we describe a death due to delayed subarachnoid hemorrhage following a motor vehicle collision, wherein the hemorrhage occurred ten days after the incident, while the patient remained hospitalized for skeletal trauma. At no time prior to the hemorrhage did the victim show any symptoms of brain injury. Autopsy revealed basilar subarachnoid hemorrhage arising from a cerebral cortical contusion. There was no evidence of aneurysm or arteriovenous malformation. A significant underlying contributing factor in the delayed hemorrhage was the victim's chronic anticoagulant therapy, which was required because of a mechanical heart valve.

**KEYWORDS:** forensic science, complications of therapy, subarachnoid hemorrhage, anticoagulant therapy, delayed death

Death due to craniocerebral trauma sustained in vehicular collisions remains a common cause of unnatural death in the United States. In many cases seen by forensic pathologists, death occurs at or shortly after the injuries are sustained. In some individuals, death occurs following hours, days, or weeks of medical intervention. Others die as a result of complications of their injuries months or even years following the initial trauma. Various medical interventions may be considered contributing factors in these and other deaths. In this report, we present a death due to delayed-onset subarachnoid hemorrhage associated with anticoagulant therapy which was administered to the victim of a motor vehicle collision because of an underlying prosthetic aortic valve.

### Case Report

On a sunny Texas afternoon, a restrained, 40-year-old woman was driving a late-1980s model automobile on a dry, two-lane, asphalt, rural highway, when an oncoming pick-up truck turned in front of her, resulting in a head-on collision. The woman was conscious, but required prolonged extrication, as she was pinned in her vehicle. She was transported to the nearest hospital, where she was

found to have fractures of the right tibia, fibula, and calcaneus, as well as a laceration of the chin and multiple cutaneous contusions of her trunk. Past medical history was significant for a remote aortic valve replacement (at age 16 years for rheumatic heart disease), for which she was receiving chronic anticoagulant therapy. During her entire hospitalization, she remained on anticoagulant therapy (Coumadin, 2.5 mg daily) for her underlying prosthetic heart valve. A cardiology consultation with echocardiography revealed no acute problems. She underwent surgical fixation of the tibia and fibula fractures at the local hospital prior to being transported to a metropolitan trauma referral center, where she was to undergo repair of the calcaneus fracture. Ten days after the accident, the victim developed altered mental status and agonal respirations, requiring emergency intubation. A computed tomography scan of the head and brain revealed acute basilar subarachnoid hemorrhage. Cerebral arteriography was not performed. As the patient had demonstrated no symptoms of brain injury prior to this event, the clinical impression was that she had suffered a possible ruptured cerebral artery aneurysm. Since brainstem function was absent, "do not resuscitate" status was established, and the victim died 11 days after the automobile collision. Because of the nature of the incident, the case was referred to the medical examiner's office, where an autopsy was performed.

Autopsy revealed the presence of surgically-fixed tibia and fibula fractures, cardiomegaly (450 g), a prosthetic ball-and-cage aortic valve, multiple cutaneous contusions, and a healing laceration of the chin. Head examination was significant for the presence of approximately 15 mL of patchy subdural hemorrhage over the cerebral convexities and in the posterior fossa, as well as moderate to extensive subarachnoid hemorrhage, most prominent adjacent to the cerebellum and inferior right temporal lobe. No cerebral

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FIG. 1—Gross photograph of the formalin-fixed brain, showing temporal lobe contusion with overlying subarachnoid hemorrhage.

artery aneurysms or vascular malformations, skull fractures or subscalpular hemorrhage were identified. Sections of the formalin-fixed brain revealed a hemorrhagic discoloration of the right medial temporal cortex (Fig. 1), which, on microscopic exam, proved to be a cerebral contusion. The subarachnoid hemorrhage was most voluminous immediately adjacent to the contusion. Histologic exam revealed no evidence of fat or bone marrow embolism. The cause of death was ruled, “complications of blunt force injuries.” Based on the autopsy findings and the clinical history, the final mechanism of death in this case was related to the subarachnoid hemorrhage. Since no vascular abnormality could be identified, the hemorrhage was most likely related to the underlying brain contusion with anticoagulant therapy contributing to the hemorrhage.

## Discussion

Deaths related to complications of traumatic injury are not rare. Such deaths can occur decades after the initial trauma, as exemplified by the classic scenario of the spinal cord gunshot wound victim with resultant paraplegia, who dies years after sustaining the injury, via sepsis related to a urinary tract infection. The causal relationship between the initial trauma and death may or may not be readily evident in such cases. When death does not occur immediately or in the critical, high-risk period of time (which varies from minutes to weeks) following the trauma, the death (or the decompensation that leads to death) may be considered “sudden and unexpected.” When such deaths occur, clinicians and others may initially attribute the death to an unrelated natural disease process, as occurred in the present case.

While delayed deaths following trauma may certainly be related to the trauma, it is also true that not every death that occurs following an injury is related to trauma. As such, it is prudent to perform complete medicolegal autopsies whenever a death occurs following trauma and the relationship between the trauma and the death is in question. In certain cases, a detailed reconstruction of the events leading to death, along with careful review of medical records, is in order. In some cases, even complete autopsy and extensive review of the circumstances may not provide a clear answer to the question of whether or not the trauma contributed to death.

Sudden, unexpected, trauma-related decompensation and/or death occurring hours to weeks following trauma may involve a variety of underlying mechanisms, including delayed hemorrhage

(1–4), epidural hemorrhage (1), pulmonary thromboembolism (5,6), fat embolism (7,8), infection (9,10), and multiorgan failure related to systemic inflammatory response (11). Underlying natural diseases or conditions, including pharmacologically or genetically induced predispositions, can be contributory in such deaths. For example, a person dying from pulmonary thromboembolism related to deep venous thrombosis due to stasis and immobility following significant trauma can have additional underlying contributing risk factors for developing venous thrombosis, such as obesity or various genetic predispositions. In a similar fashion, persons requiring anticoagulant therapy, whether chronically or short-term, are at risk for delayed hemorrhage following trauma (12). The current case represents such a case, in a woman requiring chronic anticoagulant therapy for a prosthetic heart valve. The clinical history of sudden neurological decompensation, the gross and histologic identification of a cerebral contusion with associated subarachnoid hemorrhage, the absence of any other identifiable cause of the hemorrhage, and the history of blunt force head trauma (chin laceration) are all factors which establish the brain injury sustained in the motor vehicle collision as the underlying cause of death. The chronic anticoagulant therapy is considered a contributing factor in this death, in that the delayed hemorrhage might not have occurred if it were not for the bleeding tendency imparted by the anticoagulant therapy.

The present case serves to remind clinicians and pathologists that any death following a traumatic injury must be considered a possible injury-related death. It also serves to remind physicians of the potentially deleterious effects of anticoagulant therapy, particularly in persons who have been injured.

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