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CASE REPORT

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Walk and Die: An Unusual Presentation of Head Injury

ABSTRACT: We report three deaths in young adult males following closed blunt trauma to the head and face where the affected individuals were able to walk away from the incident, before subsequently collapsing and dying a short distance from the site of the assault. In each case, due to the rapidity of the posttrauma collapse, the pathologist was faced with a diagnostic difficulty at autopsy; the external examination revealed multiple injuries to the head and face, but internal examinations showed limited findings with no structural explanation for the death. We discuss possible mechanisms that could account for this scenario, the implications of alcohol consumption with a concussive head injury, and parallels that can be drawn with the so-called "talk and die," "talk and deteriorate," and "second impact syndrome." Finally, the possible role of so-called "postexercise peril" is discussed in relation to these deaths.

KEYWORDS: forensic science, head injury, walk and die, talk and deteriorate, second impact syndrome, posttraumatic apnea, postexercise peril

Many homicide victims sustain head injuries, and many of these die from the consequences of their head trauma. Invariably, these deaths are associated with the onset of unconsciousness during the assault: most never recover consciousness, but some may do so, only then to deteriorate and die after a lucid interval which is at least 20–30 min and more usually several hours. This so-called "talk and die" or "talk and deteriorate" scenario is almost always associated with the development of an intracranial hematoma—i.e., they have an easily identifiable intracranial cause to explain their deterioration and deaths (1–4).

Sudden death is an uncommon but recognized outcome following a blow to the head and frequently occurs in conjunction with alcohol intoxication (5,6). Under these circumstances, unconsciousness appears always to develop during the assault, although at autopsy there may be no identifiable structural explanation for death. Acute brainstem dysfunction affecting cardiorespiratory function and mild diffuse axonal injury augmented by the central effect of alcohol have been postulated as possible mechanisms for death (7).

A rarer scenario is reported in association with sport events such as American football, where loss of consciousness and even death can follow a head impact in an individual who has suffered recent, often more significant blunt head trauma event, the so-called "second impact syndrome." Again, findings at autopsy are limited although these deaths may be due to so-called "malignant brain edema" (8–10).

We report three cases where the individuals received closed blunt trauma to the head and remained conscious throughout the assault. In all three cases they walked a short distance from the scene of the attack before collapsing, losing consciousness, and dying, i.e., a lucid period of less than 1 min. We present the limited autopsy findings of these cases and consider the possible mechanisms that could account for these scenarios drawing attention to the possible role of so-called "postexercise peril" in these cases (11).

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Case Reports

Case 1

A 20-year-old previously fit male with no history of recent head trauma was involved in a fracas outside a nightclub. He had been drinking alcohol. He was backed against a wall and struck to the head approximately 10 times by four males. During the assault he did not lose consciousness or fall to the ground. Ultimately, he pulled away from his assailants and walked away independently, holding his head in his hands. After walking approximately 73 m (80 yards), which took approximately 50 sec he responded to a friend's question "Are you alright?" with a mumbled "No" and subsequently collapsed onto a vehicle prior to going to the ground. There was no damage to the vehicle. He had lost consciousness and had labored breathing. He never regained consciousness despite resuscitation attempts. He was pronounced dead shortly after arrival at the local hospital.

At autopsy bruising was present to the superficial cervical muscles, the posterior aspect of the parietal bone, the right angle of the jaw, the maxilla area, and immediately behind the left external auditory meatus. Deep bruising was present to the left angle of the jaw associated with a fracture at this site. Bruising was also present to the anterior aspect of the jaw in association with a vertical fracture at the level of the first and second right tooth. The trachea and air passages contained a small quantity of blood and there was blood within the parenchyma of both lungs and within the stomach. The heart, which was examined by a cardiac pathologist, revealed a single petechial hemorrhage on the anterior

external surface of the left ventricle but was otherwise normal. The skull was intact. There were bilateral middle ear hemorrhages, more pronounced on the right than the left. Neuropathological examination revealed congestion of blood vessels within small areas of perivascular hemorrhages, particularly in the posterior part of the corpus callosum. Otherwise there was no evidence of intra-cranial hemorrhage, contusions, or intra-cerebral bleeding. The brain weighed 1688 g, was considered to be swollen, but with no evidence of cerebral edema or flattening of the gyri over the cerebral hemispheres. There was no evidence of brain herniation. There was mild and occasional β amyloid precursor protein (βAPP) deposition in the corpus callosum and cranial nerves consistent with diffuse axonal injury. Toxicological examination identified 184 mg/100 mL of alcohol within the peripheral preserved blood sample, with incidental findings of caffeine and nicotine. The cause of death was attributed to the effects of the head injury.

Case 2

A 19-year-old male with no history of recent head trauma was involved in an incident where he was repeatedly kicked in the head and body while sitting on a low wall. His girlfriend helped him to walk away but after 3–3.6 m (10–12 feet), 20–30 sec later, he collapsed. When first examined by paramedics he was in cardio-respiratory arrest, the cardiac rhythm varying between electro-mechanical dissociation and ventricular fibrillation. Despite resuscitation attempts he remained in electro-mechanical dissociation and death was certified approximately 1 h later. It is not known if he had been drinking alcohol.

At autopsy the external examination revealed bruises over the vertex of the scalp, left side of the forehead, left upper and lower eyelids, left upper arm and forearm, right hand, and on the front of the middle third of the chest on the left side. Abrasions were noted over the left side of the forehead, vertex of the scalp, and middle phalanx of the left ring finger. On internal examination the scalp showed minimal deep bruising in association with the aforementioned external injuries. The skull was intact and there were no intra-cranial hemorrhages. The brain weighed 1634 g and was diffusely swollen with no external or internal injuries present. There was no evidence of brain herniation. The heart was examined by a cardiac pathologist and found to be macroscopically and microscopically normal. No other abnormalities were identified.

It was subsequently discovered that the deceased had a family history of cardiac problems. The deceased's father had a history of atypical angina diagnosed at the age of 40 due to coronary artery spasm, and 7 years later he developed "seizures" associated with exercise. The deceased's brother was investigated at the age of 15 for chest pain and ECG revealed supraventricular tachycardia on exercise. The cause of death was certified as "unascertained," with a conclusion that the deceased had probably died as a result of a cardiac arrhythmia with the assault playing a significant role in the death.

Case 3

A 20-year-old male with no history of recent head trauma was involved in an unwitnessed incident during which he was assaulted. He had been drinking heavily and the assault involved punching, kicking and stamping. The man who admitted the assault stated that the deceased got up from the ground after the assault, staggered a distance of approximately 1.5–3 m (5–10 yards), and collapsed. The assailant then ran away.

At autopsy (second examination) the external examination revealed a large area of abraded bruising to almost the whole left side of the face and both eyelids, and towards the lower end the abrasion was vaguely patterned and linear. Bruising was also present to the entire lower lip, left upper lip, and back of the right hand. Abrasions were noted to the right forehead, upper right cheek, middle third of the neck, middle phalanx of the right middle finger, and on the left lower leg. There was also a superficial laceration in the chin to the left of the midline. On internal examination there was some deep scalp bruising, but no skull or facial fractures, no meningeal hemorrhages, and no brain injury. The brain weight is no longer available to the authors. There was no evidence of brain herniation. The heart and all other organs were normal. At the first autopsy, gastric contents were found in the major and intrapulmonary airways, but there was no vomit present at the scene or on the deceased's clothing. Blood and urine alcohol concentrations in specimens from the first autopsy were 226/100 mL and 304/100 mL, respectively. The cause of death was attributed (first pathologist) to inhalation of gastric contents due to alcohol intoxication and blunt force trauma.

Discussion

In each case described in this report, the autopsies presented the investigative pathologist with a diagnostic difficulty. Although there was a clear history of closed blunt head trauma with alcohol consumption in two cases, the subsequent autopsy examination failed to demonstrate sufficient structural damage to explain the death. Cerebral swelling was observed but there was no evidence of brainstem herniation. The fact that separates these deaths from other head injury-related deaths and which added to the diagnostic problem was the ability in each case of the victim to remain conscious during the attack, to walk away, and in one case talk, before collapsing and dying a short distance from the scene of the assault. Our cases thus differ from those previously reported in the rapidity of the onset of unconsciousness prior to collapse. To assist others who may be faced with similar case presentations we have identified and discuss a number of mechanisms proposed in the literature which could explain these atypical presentations; traumatic apnea, malignant brain edema, the neurometabolic consequences of concussion, and cardiac dysfunction.

Concussion can be considered the mildest form of brain injury, which, when severe can manifest as diffuse axonal injury. Cerebral concussion can be defined as "a transient interruption of brain functions caused by mechanical force. Memory, consciousness, motor control or brain stem functions may be temporarily interrupted or impaired during this phenomenon. The mechanical deformation of the brain tissue in concussive injury is sufficient to interfere with functions of the polarized neuronal membranes and synapses and to render numerous cerebral neurons temporarily dysfunctional" (5). A concussion is usually not sufficient to cause structural damage, but can result in abnormal cerebral metabolism for weeks after the initial injury (12). Temporary neuronal dysfunction gives the symptoms associated with concussion, but usually resolves. Giza and Hovda (12) documented potential "periods of vulnerability" after a concussive injury, during which excessive external stimulation or a second injury can result in cell death and abnormalities in neurotransmission. This vulnerability is thought to be due to altered cerebral glucose metabolism, calcium influx, and *N*-methyl-D-aspartic acid receptor abnormalities. However, we are of the opinion that concussion on its own does not explain these case presentations as there was no evidence of loss of consciousness during the assault or a second injury or excessive stimulation following the assault.

Diffuse brain swelling is a common consequence of head trauma, but the cause is unknown. It is recognized that an apparently minor head injury can cause diffuse cerebral swelling with serious consequences. McCrory describes two pathological mechanisms thought to be responsible, the first of which is cerebral hyperemia and increased blood volume as a result of disordered cerebrovascular autoregulation. This is often termed "malignant brain edema." The second is due to true cerebral edema (9,10). The distinction between cerebral swelling and cerebral edema was made by Klazko, who observed that cerebral edema could be cytotoxic or vasogenic and that both could occur after head trauma (13). The mechanism of death would therefore be transtentorial brainstem herniation as a result of raised intracranial pressure, which would affect brainstem cardiorespiratory centers. However, this does not easily explain the observations in our cases as although there was evidence of brain swelling, there was no evidence of brainstem herniation. As "malignant brain edema" associated with death is reported to take 20 min or longer to develop and true cerebral edema takes hours to days then it may be that our cases died with developing cerebral edema rather than from the effects of cerebral edema.

It has been reported that diffuse brain swelling with delayed deterioration occurs after repeated concussive injuries in sport. This phenomenon has been termed the "second impact syndrome," although there is considerable debate as to whether or not this syndrome actually exists. Second impact syndrome has been defined as occurring "when an athlete who has sustained an initial head injury, most often a concussion, sustains a second head injury before the symptoms of the first have fully cleared" (8). Cantu et al. suggested that second impact syndrome is the result of vascular engorgement within the cranium, raising intracranial pressure and causing herniation of cerebella tonsils and temporal lobes, compromising the brainstem (8). The evidence in support of such a syndrome has been reviewed and, whilst not compelling, deaths continue to occur in similar and often unexplained circumstances in sport. McCrory reviewed published reports of second impact syndrome, and found that in 11 of the 17 cases described there was no evidence of a second injury occurring. He found 13 cases of sports-related catastrophic brain injury associated with unexplained cerebral swelling, many of which did not involve a second impact. Some of these cases collapsed during sport participation but he records three who walked away and then collapsed. Two of these individuals were found to have subdural hematomas and cerebral edema, and the other had cerebral edema, brainstem herniation, and midbrain necrosis (10). Thus, although our cases appear to share similarities in the timing between the trauma and the collapse, the absence of documented previous head trauma and identifiable intracranial pathology does not allow us to present our cases as examples of "second impact syndrome," assuming that the syndrome is universally accepted to exist.

Delayed deterioration after head injury has also been documented in pediatric medicine. Bruce describes a syndrome in which children over the age of 1 year who sustain a minor head injury have a delayed progression to coma, pupillary changes, and apnea, with postmortem evidence of diffuse cerebral swelling and congestion of small blood vessels in the white matter (14). He postulates that cerebral metabolism and changes in cerebral blood flow and volume, in association with cerebral edema may be possible causative mechanisms.

The role of alcohol in these cases also needs to be considered. The blood alcohol levels in the two cases are not sufficient alone to cause cardiorespiratory depression, which would normally be seen in concentrations over 350 mg/100 mL, although it is possible that alcohol has still played a role in the mechanism of death. Zink and Feustel demonstrated via animal experiments that moderate alcohol intoxication combined with concussive brain injury can cause significant respiratory depression and an impaired ventilatory response—so-called "posttraumatic apnea" (7). Ethanol is a central nervous system depressant and it is possible that it depresses neurones to such an extent that they become more vulnerable to injury, particularly in the brainstem, increasing the risk of fatal arrhythmias and apnea. Ethanol enhances the inhibitory effect of gamma-aminobutyric acid and suppresses the excitatory actions of L-glutamate. The net effect of this on medullary neurones is inhibition of sympathetic nerve activity, which could impair cardiorespiratory center response with resulting apnea and fatal dysrhythmias (6).

The terms "talk and deteriorate" and "talk and die" have been applied in medical literature to describe patients who suffer from mild to moderate head injury, are able to articulate recognizable words, and then deteriorate within 48 h of the injury (1–4). In a review of cases over a 10-year period, Goldschlager et al. documented six such cases in which the predominant pathology was cerebral contusions, peri-contusional edema, progressive generalized swelling, and cisternal effacement, but none in which there was isolated diffuse brain swelling (3). The remaining nine cases were found to have intracranial mass lesions, the most common of which was an acute subdural hematoma. However, the majority of their patients in this review were elderly and the mechanism of injury was most commonly a fall. Similarly, Ratanalert et al. found surgical mass lesions in 90% of the patients considered in their study (2). Reilly et al. found that a quarter of the patients they encountered did not have significant intracranial hematomas (1). This is of significance in emergency medicine, because patients who are lucid after an apparently minor head injury who present to the emergency department may be discharged after an intracranial hematoma has been excluded, and subsequently deteriorate. However, the attribution of the terms "talk and deteriorate" and "talk and die" are not appropriate to our cases again due to the rapidity of the events and the lack of intracranial pathology. Hence, the choice of our wording "walk and die."

With regard to cardiac dysfunction, on postmortem examination and histology in each of the presented cases, no morphological cardiac abnormalities were identified. However, it is well recognized that fatal cardiac arrhythmias can and do develop in morphologically normal hearts. A family history of heart disease becomes important in such cases although of our three cases only Case 2 recorded a family history of cardiac dysfunction. Deaths have been reported in the literature related to psychological stress and pain, both in animals and humans, although these are associated with microscopic cardiac changes (15,16). Although a morphological abnormality was not identified to provide a mechanism for the cause of death, an arrhythmogenic cause, possibly regulated by cerebral cardiorespiratory dysfunction or systemic metabolic alterations is considered to be the most likely explanation for the deaths in our cases. Dimsdale et al. described the phenomenon of so-called "postexercise peril" (11). In their observations of 10 healthy men who underwent exercise testing they report the rise in catecholamine levels, not only as a response to the exercise during the exercise period but also the level continued to rise in the immediate postexercise period. A 10-fold increase over baseline of norepinephrine could have profound cardiac effects especially in those with pre-existing cardiac disease. Although Dimsdale et al.'s original paper discusses this in relation to pre-existing coronary artery disease it could be postulated that in those with undiagnosed cardiac arrhythmogenic syndromes, as have become recognized in recent years as causes of sudden unexpected adult death, this

natural physiological response could place these individuals at increased risk of death under the circumstances described in our cases. In addition to this, blood potassium concentrations also alter during exercise, first increasing and then rapidly decreasing in the postexercise period. The combination of raised catecholamines and deceasing potassium levels in the presence of pre-existing cardiac disease may result in fatal cardiac arrythmias. A case of a 16-year-old female with occult Carvajal syndrome who was verbally abused and had ''minor'' direct trauma prior to running up a 100 m incline and then collapsing and dying has recently been reported. The authors of this case report discuss the possible role of ''postexercise peril'' in relation to her death (17).

In summary, we present three cases of young adult males with closed blunt head trauma all presenting with an extremely short lucid period following the assault prior to collapse and death. Autopsy examinations in all three cases provided no satisfactory explanation for the death, leaving the pathologist with difficulty in explaining on morphological grounds alone how the individuals had come by their death. The known presence of alcohol in two of the three cases must be considered a significant contributing factor due to the recognized additive effect of alcohol on closed head injury although it does not explain the death on its own. The cases share similarities to the previously reported ''talk and die/deteriorate'' syndromes and some cases reported in series discussing the controversy of ''second impact syndrome.'' However, our cases differ from these in the lack of previous documented head trauma, the timing of the lucid period prior to collapse, and the lack of observable intra-cranial pathology, particularly brainstem herniation. Thus, we postulate that rather than a single mechanism or macroscopic finding a combination of malignant brain swelling without herniation, cellular neuronal and cerebral cardiorespiratory center dysfunction with resulting posttraumatic apnea and fatal cardiac arrhythmia acted together to result in the atypical presentations in these cases. Of these mechanisms we favor that the mechanisms underlying so-called ''postexercise peril'' played a significant contribution in these deaths.

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