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REVIEW

The syndrome of excited delirium

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Abstract The excited delirium syndrome (EDS) is a life-threatening condition caused by a variety of factors including drug intoxication and psychiatric illness. Fatal instances of excited delirium frequently come to the attention of the medical examiner/coroner due to the circumstances and potential causes. Excited delirium may include paranoid, aggressive, and incoherent behavior which may lead to an encounter with law enforcement. In some instances, the person may die while in the presence of law enforcement. This circumstance further broadens the potential causes of death particularly as EDS has no pathognomonic autopsy finding. Although the syndrome of excited delirium is sufficient to explain death, other intervening causes need to be considered. These include chest or neck compression during restraint, blunt trauma, and underlying natural disease. Since chest/neck compression, natural disease (e.g., atherosclerosis), blunt trauma, and excited delirium are not mutually exclusive, all may be present in one death. The forensic pathologist's role is to determine what caused and/or contributed to the death. When attempting to determine the proximate cause of death in instances with multiple potential causes, determining the mechanism of death often is useful. As not all causes of death have pathologically-demonstrable mechanisms of death, examination of the circumstances of the death often are diagnostically important. The main goal of the autopsy of deaths suspected to be due to EDS is to identify (or exclude) intervening diseases or injuries sufficient to explain the death in the context of the investigated circumstances.

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Introduction

There has been debate in medicine as to how to characterize the syndrome of excited (or agitated) delirium and if it even exists [1–7]. In 2009, the American College of Emergency Physicians issued a white paper on the excited delirium syndrome (EDS) and the National Institute of Justice convened a workshop panel to examine the issue [1, 7]. Due to the nature of the syndrome, emergency physicians and forensic pathologists are the two medical disciplines that typically encounter these patients.

Excited delirium syndrome is a life-threatening condition initiated by a variety of causes (Tables 1, 2). Presently, the syndrome is largely associated with drug intoxications (cocaine, methamphetamine) and psychiatric illnesses [8–10]. Excited delirium is a type of delirium involving violent behavior; delirium and excited delirium are not synonyms [8, 9, 11–22]. Fatal instances are characterized by sudden death during or following an episode of excited delirium in which an autopsy fails to detect a disease or physical injury that has an extent or severity to explain the death and the circumstances are consistent with the syndrome [9]. It is a clinicopathologic diagnosis based upon the autopsy and toxicologic results evaluated in the context of the history and circumstances.

EDS is characterized by sudden bizarre and/or violent behavior that may be accompanied by combativeness, confusion, hyperactivity, paranoia, incoherent shouting, hallucinations, hyperthermia, and sudden death [11–20]. It is more common when the weather is warm and humid and deaths occur more often in the summer [13, 18]. As these

Table 1 Delirium/psychosis [17, 72, 73]

Delirium is an acute, confusional syndrome with a transient disturbance in consciousness and cognition that has a variety of causes:
Drug intoxications/withdrawals/noncompliance
Acute functional psychosis (schizophrenia, acute mania)
Endocrine/metabolic disease
Hyperthyroidism, hypothyroidism ("myxedema madness")
Hypoglycemia
Pituitary disease with secondary endocrine effects
Postpartum psychosis
Porphyria
Liver and kidney failure (uremia)
Electrolyte disorders
Acid base imbalance,
Nutritional disorders
Thiamin, Vitamin B12 deficiency ("megaloblastic madness")
Infections
Meningitis, encephalitis, sepsis
Neoplasia (brain tumors)
Seizure disorders
Complex partial seizures ("temporal lobe" or "psychomotor" epilepsy), postictal state
Vascular disorders
Hypertensive encephalopathy/cerebral infarcts
Systemic lupus erythematosus
Disseminated intravascular coagulation
Trauma
Concussion
Subdural, epidural, subarachnoid hematoma
Hypothermia/hyperthermia
Hypoxia
Factitious psychosis/malingering

Table 2 Drugs associated with acute psychosis [17]

Ethanol
Ethanol intoxication
Ethanol withdrawal
Sympathomimetic drugs
Cocaine
Amphetamines
Tricyclic antidepressants
Monoamine oxidase inhibitors
Methylphenidates
Sedative drugs
Sedative drug withdrawal
Hallucinogens
Cathinones [45]
Lysergic acid diethylamide (LSD)
Mescaline
Phencyclidine (PCP)
Psilocybin
Anticholinergic drugs
Includes drugs with anticholinergic side-effects, antipsychotic agents, antihistamine, etc.
Corticosteroids

individuals often come to the attention of law enforcement personnel, they are frequently restrained. A violent struggle often precedes the restraint. Sometime after the struggle, the individual is noticed to be unresponsive and in cardiopulmonary arrest [12, 13, 19].

If death occurs during or shortly following struggle and/or restraint, the differential diagnosis of the cause of death expands to include possible physical trauma including neck or chest compression (from choke holds or restraint), blunt impact injury, electrical injury from the use of electronic control devices, or chemical injury from pepper sprays [23–26]. The investigation and certification of these deaths can become controversial for the forensic pathologist [8, 9, 27–29] who may face challenges from the family, the press, and other legal agencies who are concerned that the police caused the death.

Pathogenesis and pathophysiology

Historically, excited delirium has been described in psychiatric patients as acute exhaustive mania (Bell's mania) [8, 9]. Much attention has been given to the role of restraint and struggle [14, 16, 30–37]. In addition to asphyxial mechanisms, neurochemical abnormalities involving dopamine, elevated potassium concentrations, lactic acidosis, autonomic dysfunction, and catecholamine cardiac effects have been studied [9, 19, 38–40]. Cocaine and amphetamine may cause the syndrome. As cocaine affects the dopamine and catecholamine systems of the body, some have likened excited delirium to the neuroleptic malignant syndrome (NMS) [13]. One theory is that abnormalities in the number and type of dopamine receptors result in the excited delirium syndrome. Elevated environmental temperature creates a further risk for morbidity and mortality particularly in conjunction with cocaine abuse [41].

Autopsy

At autopsy, these deaths are best treated as "in custody" deaths which includes full body photographs demonstrating all positive and negative findings. There is no pathognomonic autopsy finding and minor injuries (abrasions, contusions, cuts) are typical in deaths due to EDS. There are

case reports of people in an excited delirium with more serious injuries from slamming the heads against a wall and jumping down stairs or through windows [12, 17, 42, 43]. Serious craniocerebral trauma has been reported in deaths due to excited delirium [43]. These head injuries must be carefully evaluated in the context of the circumstances and mechanisms of death to determine if they are causative of death or incidental. An injury must mechanistically and circumstantially fit with causing death in order for it to be invoked as the cause of death. Nonlethal blunt injuries sustained in the process of subduing an individual can be inappropriately over-interpreted [13]. Without a mechanistic link (e.g., lacerated liver with a large hemoperitoneum) some external injuries may be incidental.

The presence or absence of internal neck injury and petechia (conjunctival, facial, and oral mucosa) are documented. Postmortem radiographs and subcutaneous dissection for occult contusions also may reveal injuries that would not be identified on a routine autopsy. EDS deaths may have a prolonged survival interval if the patients are successfully resuscitated. They may later develop rhabdomyolysis, acute renal failure, and disseminated intravascular coagulation. Some EDS patients are successfully resuscitated and survive [44].

Toxicologic analysis is needed to discern the underlying cause for the syndrome if it is due to intoxication. Common and uncommon drugs of abuse (cocaine, methamphetamine, PCP, and other hallucinogens) as well as various psychiatric medications and anticholinergic medications should be included in the screen. A recent report described bath salts (cathinones) causing non-fatal episodes of the excited delirium syndrome [45]. If there is a prolonged survival interval in the hospital, retrieval and testing of a hospital admission blood sample may be valuable.

Although there is no gross or microscopic finding diagnostic of death caused by excited delirium, postmortem neurochemical examination of the brain (dopamine synaptic markers/receptors in the striatum and hypothalamus) has been reported by some to help diagnose EDS [13, 19, 46, 47]. This testing may help support the diagnosis of EDS but an efficient intervening cause of death (e.g., neck compression) still needs to be considered and excluded. In addition, the underlying cause of the excited delirium also needs to be determined.

The role of the medical examiner/coroner in these deaths is to certify the cause and manner of death. Due to the complex physiologic, chemical, environmental, and traumatic interactions that occur, the circumstances surrounding a death often supply important information needed for proper certification. Primary sources of information (witnesses) are used to create a step-by-step, freeze-frame analysis of the events [29]. Witnesses are asked what they saw and heard. Was there neck or chest compression? If so, for how long? Was the person speaking, yelling, or making noises? For how long, and was it continuous? When did the person become unresponsive? A search for video documentation of the event (patrol car dashboard cameras, security cameras, or by-standers) should be done as the episode is often chaotic for the first responders and retrospective estimations of times and sequences may not be precise.

Restraint, mechanical injury, electronic control devices, and pepper spray

Mechanical and positional asphyxias are potential causes of death that should be considered with deaths associated with excited delirium [32, 42, 48–50]. The classical types of "asphyxia" are defined in Table 3. Although prone restraint with up to 50 lbs of weight on the back can result in a restrictive pulmonary function pattern, hypoxia or hypoventilation has not been demonstrated [51]. At autopsy, the differentiation of death due to intoxication versus asphyxia is challenging as there may by few, if any, definitive diagnostic autopsy findings. Investigative (e.g., witness statements) information is particularly helpful to assess if chest or neck compression caused death. If the person was speaking, yelling, or moaning, it is important to note when it stopped.

Some instances have been recorded on videotape. It is not unusual to hear a person in an altercation with restraint to state "I can't breathe." In order to vocalize one must be able to move air past the vocal cords (i.e., to breathe). If a person has constant compression of the chest that is enough

Table 3 Asphyxia: classic types

I. Compression of neck (usually a cerebral ischemic mechanism due to vascular compression)
II. Obstruction of airway without neck compression:
A. Smothering: external occlusion
B. Choking: internal occlusion
III. Chest compression (traumatic asphyxia)
IV. Positional/postural asphyxia (body position, such as inversion/awkwardly flexed neck, that restricts/interferes with respiration)
V. Exclusion/displacement of environmental oxygen
VI. Combination (e.g., smothering and chest compression)
VII. Other (e.g., cyanide poisoning, so-called cellular asphyxia)
Asphyxia: The physiologic and chemical state in a living organism in which the acute lack of oxygen available for cellular metabolism is associated with inability to eliminate excess carbon dioxide. The critical feature in asphyxia is cellular hypoxia or anoxia. The use of the term usually is restricted to instances with unnatural circumstances.
Suffocation is a general term for asphyxia with the failure of oxygen to reach the blood.

to prevent movement of the chest, could they still yell "I can't breathe"? Intermittent compression may explain this apparent dichotomy. A subjective air hunger related to the exertion, and an inability to adequately hyperventilate may also explain why a person can move enough air to verbalize but feel that they cannot physically breathe. During the restraint, the struggling may stop. The responders may believe the person is starting to calm down or may be pretending to become calm, thus they do not lessen their restraint. Once the person is released from restraint they can be unconscious and in cardiac arrest. This occurrence is very concerning for a compressional asphyxia mechanism of death.

Electronic control devices (ECDs) are used on people with EDS [25, 52]. ECDs are battery powered devices used to temporarily incapacitate a person using a short duration, high voltage, and low amperage electric current. The current will find low resistance routes within the body (e.g., blood vessel and nerve pathways) which can physically incapacitate the body [53]. Several studies and more than 100,000 ECD applications on volunteers have occurred without reported cardiac arrests or deaths [28, 39, 54]. Electricity causes death by two likely mechanisms. One is a contraction of muscles (including respiratory muscles) that results in asphyxia if the electrical current application is continuous and prolonged. The second mechanism is electrical current through a vital organ that is susceptible to disruption by the flow of electricity (e.g., the brain or heart) [28, 53, 55–58] Tissue thickness and electrode location on the body may be a factor in cardiac disruption. A study of sheep in which the skin and subcutaneous tissues were reflected was able to trigger ventricular fibrillation if the dart was implanted within 2.3 cm of the heart [59]. A man with an implanted pacemaker, who survived an ECD application, had ventricular myocardial capture at a high rate corresponding to the exact time of the barb application as determined by subsequent interrogation of the pacemaker [60].

A study of deaths following electro-muscular disruption devices, initiated by the National Institute of Justice, concluded that there was no conclusive medical evidence that indicated a high risk of serious injury or death to humans from the direct or indirect cardiovascular or metabolic effects of short-term ECD exposure in healthy, normal, non-stressed, non-intoxicated persons. The risk of death in an ECD-related use of-force incident was less than 0.25 %. It concluded that ECDs do not cause or contribute to death in the large majority of those cases [61]. Witness statements may clarify the role of ECDs in a death. If unresponsiveness occurred minutes after the device application ceased then the expected pathophysiologic mechanism of an electrocution is unlikely [62].

Pepper spray is meant to cause discomfort and irritation but rarely causes death. It may cause bronchospasm as evidenced by florid bronchiolitis and the rapid onset of dyspnea following exposure to the spray [23].

Stress and disease

Stress can play a role in causing death (e.g., homicide by heart attack) [63, 64]. Stress, from restraint or struggle, may also occur in deaths due to EDS [65]. Veterinary studies have reported unexpected death of big game animals following their chase and capture due to "capture myopathy" (or "exertional myopathy") [14, 66]. Clinical studies have demonstrated the effects of stress ("myocardial stunning") on the heart such as takotsubo cardiomyopathy [67, 68]. Reports of healthy marathon runners who collapse near the finish line may share some type of post-exertional mechanism with EDS. One study of ultramarathons (56 km) showed that the collapse of 85 % of runners with exercise-associated collapse occurred after they had completed the race [69]. Underlying advanced heart disease combined with stress and extreme physical exertion is a well-known recipe for a sudden cardiac death. Another potential factor in these EDS deaths is an unrecognized channelopathy. Arrhythmias in some channelopathies may be triggered by exertion (e.g., catecholaminergic polymorphic ventricular tachycardia) and a prolonged QT interval was noted in one survivor of EDS [70]. This also may explain why only a few of the thousands of emotionally disturbed individuals who are restrained, die suddenly [71].

Disease may result in delirium. Patients with marked hypoxia, diabetic ketoacidosis or hypoglycemia, heat stroke, thyrotoxicosis, serotonin and neuroleptic malignant syndromes, and intracerebral hemorrhage, may have mental status changes (acute confusional states) and some may become agitated [72–74]. Psychiatric issues may mimic excited delirium syndrome and may be associated with psychotropic drug withdrawal or non-compliance [7].

A death due to excited delirium usually is certified as "pending further studies" on the day of the autopsy. Further investigation of the circumstances, toxicology testing, and other ancillary studies, can take days or weeks to complete. Once the investigation is concluded, clear communication of the cause and manner of death to the family and appropriate law enforcement agencies is done.

Key points

1. In deaths due to EDS, the proximate cause of death is the disease or intoxication that caused the EDS.
2. In order to diagnose EDS, an autopsy and detailed investigation of the circumstances of death are needed to determine the etiology and exclude other efficient intervening causes.

3. Although electronic control devices may be involved with instances of EDS, they do not cause or contribute to death in the vast majority.
4. Cocaine is a common intoxicant with EDS but other drugs (including bath salts) can result in EDS.

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