D93 Deaths Due to Excited Delirium in Psychiatric Patients and Unassociated With Stimulants

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After attending this presentation, attendees will know that deaths due to excited delirium can occur in psychiatric patients unassociated with stimulants such as cocaine. The mechanism of death, a cardiac arrhythmia induced by abnormally high levels of catecholamines due to the mental illness itself and to the struggle, will be discussed as will the inability to resuscitate these individuals due to the nature of the arrhythmia.

This presentation will impact the forensic science community by showing that deaths from excited delirium do not have to be associated with the use of stimulants, have the same presentation as cases due to stimulants, and therefore can be prevented by the same treatment methods used with stimulant cases.

Delirium involves an acute (minutes to hours), transient disturbance in consciousness and cognition. There is disorientation; disorganized and inconsistent thought processes; inability to distinguish reality from hallucinations; disturbances in speech; disorientation to time and place; and misidentification of individuals. When the delirium involves combative and/or violent behavior, it is termed Excited Delirium (ED). Excited Delirium Syndrome (EDS) involves the sudden death of an individual, during or following an episode of excited delirium, in which an autopsy fails to reveal evidence of sufficient trauma or natural disease to explain the death. Individuals in ED are often resistant to the effects of pepper spray or application of an electro-muscular disruption devise (e.g., TASER®).

Deaths due to EDS were originally described in individuals with intrinsic mental illness, specifically schizophrenia and/or bipolar disease. In our present society, they are more commonly associated with abuse of illegal stimulants such as cocaine or methamphetamine. Because of this, many individuals mistakenly believe that EDS only occurs in association with drugs. In fact, a significant number of individuals dying of EDS are individuals with intrinsic mental disease with drugs playing no role in the death.

The mechanism of death in cases of EDS appears to be a cardiac arrhythmia induced by abnormally high levels of catecholamines (epinephrine and norepinephrine) due to the intrinsic mental disease or illegal stimulants combined with elevated levels due to the struggle. In the case of individuals using illegal stimulants, the elevated levels of catecholamines are associated with the physiological action of these drugs. In addition, these drugs also have a direct cardio-toxic action on cardiac muscle.

Individuals with intrinsic mental disease, such as schizophrenia, quite frequently experience episodes of acute psychosis/excited delirium. This can be due to failure to take medications, use of drugs of abuse, perceptions of a hostile environment, or a perceived provocation. This tendency to develop excited delirium in schizophrenics is aggravated by the fact that in schizophrenia, there is a disturbance in the metabolism of norepinephrine (NE) both in the brain and peripherally. Numerous studies in both medicated and unmedicated patients with schizophrenia have found elevated NE concentrations in samples obtained from plasma, cerebrospinal fluid, and postmortem brain tissue. In addition, stress in individuals with schizophrenia results in reactive blood levels of NE higher than reactive levels in normal individuals.

Individuals with EDS going into cardiac arrest are rarely resuscitated successfully. The most likely explanation for this is the nature of the arrhythmia. Fatal arrhythmias fall into two general categories: tachy-arrhythmias such as ventricular fibrillation and brady-arrhythmia-asystole. The latter goes from bradycardia to Pulseless Electrical Activity (PEA) to asystole. While successful resuscitation rates as high as 59% have been reported with tachy-arrhythmias due to cardiac disease, with brady-arrhythmia-asystole, successful resuscitation is measured in the low single digits at best. Thus, in a prospective study of 148 subjects who went into cardiac arrest in casinos, 105 subjects had an initial cardiac rhythm of ventricular fibrillation, 17 had PEA, and 26 had asystole. Of the subjects whose initial cardiac rhythm was not ventricular fibrillation, none survived to be discharged from the hospital. Automated external defibrillators were used on the 105 patients with ventricular fibrillation. In 90 instances where the collapse was observed, 53 (59%) survived to be discharged from the hospital. Three of the 15 whose collapse was not observed survived.

This study presents 11 cases of EDS, 7 due to psychosis, where EMTs were present at the scene of a cardiac arrest and immediately documented the arrhythmia. Of the 11 cases: six showed asystole; one showed PEA; two had bradycardia; one was a progression from a normal sinus rhythm to bradycardia, to PEA, and then asystole; and, one case where an EKG showed borderline bradycardia with a second EKG a few minutes later showing asystole. This research suggests that this last case should be classified as a

brady-arrhythmia progressing to asystole. The nature of the arrhythmias observed in these cases of EDS may explain the virtual absence of successful resuscitation from this entity.

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