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Ruptured Infective Aneurysm Presenting as an Isolated Subdural Hematoma: A Rare Cause of Death in A Drug Addict

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Abstract

A ruptured infective aneurysm presenting as an isolated subdural hematoma is rare. Subdural hematomas are primarily caused by blunt trauma to the head. In this case of a 25-year-old drug addict who was found unresponsive, taken to the hospital where a Subdural Hematoma (SDH) was discovered, and subsequently expired six days later, the initial presentation was that of a homicidal death. Upon post-mortem examination, a previously undiagnosed infective endocarditis of the mitral valve as well as multiple infarctions of the brain, spleen, and kidneys were identified. The subdural hematoma originated from a superficial cortical hemorrhage which perforated into the subdural space. Microbiological investigation revealed colonization by *Methicillin - resistant Staphylococcus aureus.* The manner of death in this case was natural, which is rare given that the typical manner of death in drug-related deaths is accidental or homicidal. All deaths of drug addicts should be subjected to a complete forensic autopsy since various causes of death can be contributory.

Introduction

- Cause of Death (COD) in drug addicts is predominantly accidental or intentional overdoses [3].
- Research has found that about 20% suffer lethal traumas, and 10% die from natural causes [3].
- Inflammatory diseases such as pneumonia, myocarditis, endocarditis, and hepatitis may play a contributory role in natural COD in drug addicts [3].
- Intravenous drug use can be associated with needle sharing, infective pathogens such as Hepatitis B and C, and Human Immunodeficiency Virus (HIV) [3].
- A majority of drug-related fatalities are due to opiates [3].
- Recent studies show that mixing different psychotropic substances including alcohol is prominent [3].
- Since COD in patients with a history of drug abuse use can be attributed to multiple factors, a complete forensic autopsy including histology, microbiology, and toxicology should be performed in all cases.

Case History

- A 25-year-old female found unresponsive on Aug 18, 2017 face down, partially clothed, and covered in emesis and feces
- Taken to a local emergency room where a subdural hemorrhage was discovered
- Underwent a craniotomy and evacuation of the hematoma and died in the hospital on Aug 24, 2017
- Autopsy confirmed a subdural hematoma, endocarditis, HIV infection, hepatitis C infection, and evidence of intravenous drug abuse.

Medical History

- Human Immunodeficiency Virus (HIV)
- Hepatitis C infection

Hospital In-Patient Laboratory Results

- *Methicillin - resistant Staphylococcus aureus* (MRSA)
- Blood culture

- Gram stain: aerobic and anaerobic bottles gram-positive cocci in groups
- Culture results: aerobic and anaerobic bottles MRSA
- Drug screen, urine - positive for THC (TH- cannabinol) and Cocaine

Autopsy

Gross Description

- Heart - 293 g. The coronary artery distribution is normal with no atherosclerosis or thrombi; the epicardium shows patchy purulent exudate. There is a friable, polypoid, soft 1" vegetation on the anterior mitral valve leaflet. Scattered microabscesses are grossly visible in the epicardium and myocardium to include the papillary muscles.
- Lungs - left - 933 g., right - 1026 g. Marked congestion and edema with no definite purulence on sectioning. Both lower lobes are firm and airless.
- Kidneys - left - 250 g., right - 190 g. Scattered cortical scars with grossly visible microabscesses on the cortical surfaces on both kidneys.

Figure 1: Friable, polypoid, soft 1" vegetation on the anterior mitral valve leaflet.

Photo by medical examiner personnel at Tidewater Office of the Chief Medical Examiner, Virginia.

Brain Examination

- **Weight fixed:** 1184 g
- **External findings:**
- Bilateral cerebellar Subarachnoid Hemorrhage (SAH) R>L
- Diffuse edema R>L
- R. parietal lobe herniating at the dural graft site
- R. uncus soft, suggestive of herniation
- 2.7 cm x 2.5 cm superomedial parieto-occipital SAH with a 0.8 cm rupture into Subdural (SD) space.

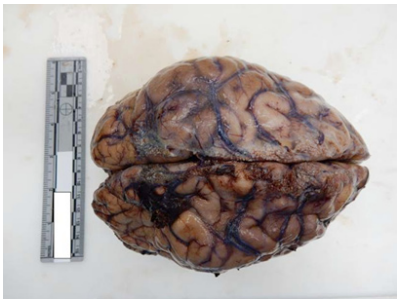

Figure 2a: Superomedial parieto-occipital subarachnoid hemorrhage.

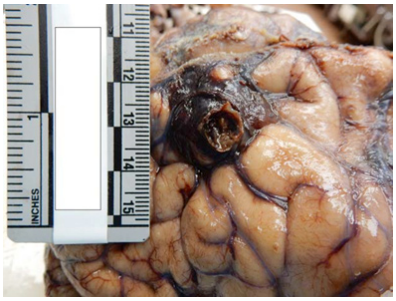

Figure 2b: Rupture of the right superficial cortical vessel**.**

Photo by Michael Hays, MD at Tidewater Office of the Chief Medical Examiner, Virginia.

- **Leptomeninges:**
- Dura partly removed; clotted blood
- R. dural graft, 12.5 cm x 10.0 cm
- R. Subdural Hematoma (SDH) supra/infra tentorial

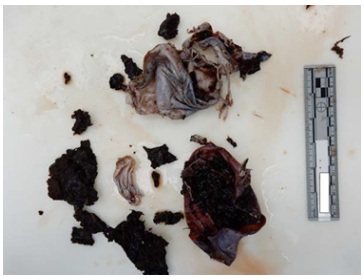

Figure 3: Partly removed dura and clotted blood.

Photo by Michael Hays, MD at Tidewater Office of the Chief Medical Examiner, Virginia.

- **Coronal section findings:**
- R. uncal necrosis/herniation
- Compressed R. ventricle
- 0.5 cm ruptured aneurysm

- Additional embolic sites with microabscesses in the right Middle Cerebral Artery (MCA) distribution at grey/white matter junctions, 0.2-0.3 cm

Figure 4: Microabscesses in right middle cerebral artery distribution at grey/white matter junctions.

Photo by Michael Hays, MD at Tidewater Office of the Chief Medical Examiner, Virginia

Histological Examinations

- **Heart:** multifocal parenchymal abscesses. The epicardium shows a variable, predominantly mononuclear inflammatory response with variable numbers of hemosiderin-laden macrophages.
- **Mitral valve:** valve leaflet with acute inflammation, abscess formation, and polypoid masses of fibrin admixed with confluent geographic bacterial colonies.
- **Spleen:** geographic areas of infarction with bacterial overgrowth. The edges of infarction show variability in acute inflammatory response.
- **Lung:** congestion, anthracosis, edema, terminal changes. Patchy bronchopneumonia with acute inflammation variably present within the bronchial lumina and alveolar spaces.
- **Kidney:** patchy interstitial mixed inflammation. There are confluent, geographic areas of infarction and abscess formation.
- **Liver:** slight fatty change, patchy portal triaditis with an increase in portal fibrous tissues without overt cirrhosis.

Micrographs of Cerebral Tissue

H&E Stain

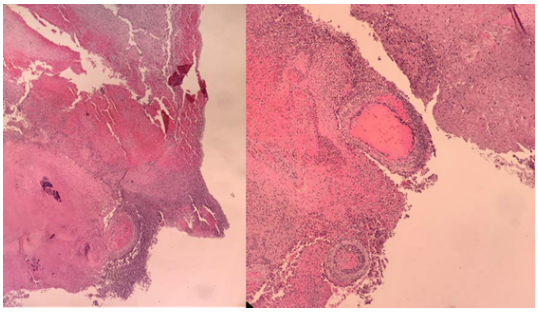

Figure 5a: Clot with inflammatory response.

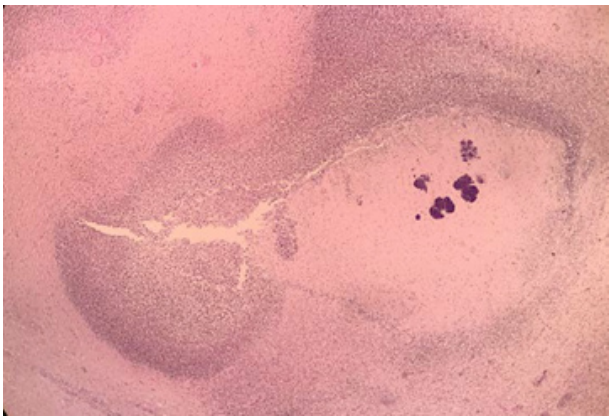

Figure 5b: Microabscess.

Slide images by Michael Hays, MD at Tidewater Office of the Chief Medical Examiner, Virginia

Grocott's Methenamine Silver (GMS) Stain

Figure 6: Negative for fungal infection.

Slide images by Michael Hays, MD at Tidewater Office of the Chief Medical Examiner, Virginia

Gram Stain

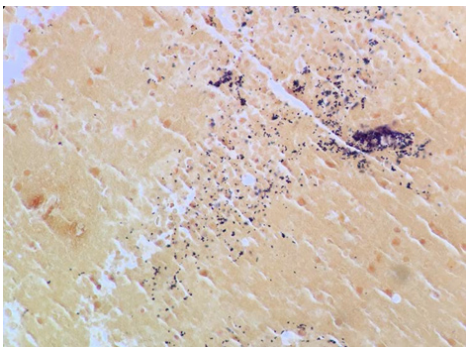

Figure 7: Gram-positive cocci.

Slide images by Michael Hays, MD at Tidewater Office of the Chief Medical Examiner, Virginia

Elastin Stain

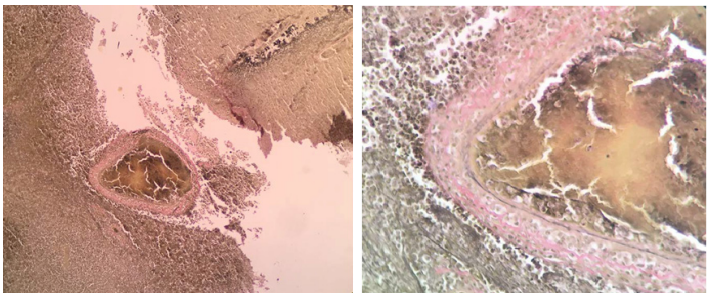

Figure 8: Vessel wall damage from the inflammatory response.

Slide images by Michael Hays, MD at Tidewater Office of the Chief Medical Examiner, Virginia

Toxicology

Hospital blood positive for benzoylecgomine, 0.027 mg/L

Pathological Diagnosis

- R. subdural hemorrhage - status post right craniotomy with evacuation of the subdural hematoma and dural repair
- Bacterial endocarditis of the mitral valve due to MRSA
- Purulent pericardial fluid
- Grossly visible abscesses of the myocardium, spleen, and kidney
- Bronchopneumonia
- History of Human Immunodeficiency Virus (HIV) and hepatitis C infection
- Pulmonary anthracosis and bullous emphysema.

Cause of Death

Subdural hematoma from ruptured infective cerebral aneurysm due to MRSA endocarditis as a complication of IV drug use

Discussion

- With a history of drug abuse and being found at a known drug house, the patient was initially thought to have succumbed to a drug overdose.
- When the SDH was discovered at the hospital, COD was possibly trauma.
- SDH is rare and can be caused by various etiologies: cortical artery bleeding, vascular lesions, coagulopathy, neoplasms, spontaneous intracranial hypertension, cocaine, and arachnoid cyst [1].
- From autopsy, confirmation of bacterial infection suggested an infection-related COD.
- The etiology of infective aneurysms (IAs) is not clearly defined, since they may be difficult to identify by neuroimaging. IA-related hemorrhage may be subarachnoid (~20% of patients), intraparenchymal (~25%) or even intraventricular (~5%). Treatment is largely medical (antibiotics) rather than surgical [1,5].
- IAs account for 5-6% of intracerebral aneurysms [1].
- IAs arise from microbe-carrying emboli, usually originating from an infected heart valve or pulmonary vein [2].
- Most frequently IAs occur in the distal branches of the cerebral arteries, particularly where due to bacterial infection, with particular predilection for the MCA. Bacterial colonization may be facilitated by the absence of vasa vasorum in branches of these vessels. Inflammation and destruction of the artery appear to proceed from adventitia inwards making the subarachnoid space locally disappear causing the blood to flow directly to the subdural space or the brain parenchyma [2, 4].
- May present with SAH, brain hemorrhage, infarction, or headache [1].
- IAs have a mortality of approximately 30% if bacterial and approximately 90% if fungal [1].
- Infection-induced acute/chronic inflammation weakens the vessel wall, resulting in ectasia leading to formation of an IA [1-13].
- 65% of patients with IAs have underlying endocarditis [5].
- Other common sources of infection: intravenous drug abuse (6.3%), bacterial meningitis (5.2%), poor dental hygiene (4.2%) and cavernous sinus thrombosis (2.8%) [5].
- ~3% of patients with infective endocarditis have been claimed to develop such aneurysms [2].
- Male to female ratio of Infective Endocarditis (IE) is 1:2 [3].
- Important risk groups for IE are intravenous drug abusers (IVDAs), individuals with degenerative valve sclerosis or prosthetic valves and patients exposed to nosocomial infections [3].
- Predisposing role of rheumatic valvulopathy in IE has decreased [3].
- According to a cohort study of 125 intravenous drug abusers (IVDAs) with native valve IE, the right and left sides of the heart are affected with approximately equal frequency: the tricuspid valve was involved 46%, the mitral valve in 25.6%, the aortic valve in 19% and the pulmonary valve in 0.8%. Among the causative microorganisms, Staphylococci (65.6%) and Streptococci (25.6%) predominated. The mean age of the diseased IVDAs was 37 years in Mathew's study material [3].

- Due to clinical symptoms of IE being subtle (fever, night sweats, arthralgia, anorexia), it is often not diagnosed during life [3].
- If neurological symptoms are present as a consequence of CNS embolization (apathy, drowsiness, confusion or a dazed state) these can be confused with symptoms of a drug overdose [3].
- Infectious emboli originating from vegetations of left-sided bacterial endocarditis are disseminated to distant organs (brain, spleen, kidneys) [3].
- In summary, the occurrence of an acute SDH is extremely rare in the context of IE; with regard to IAs only 10 cases have been reported [3, 5].

Conclusion

- Undiagnosed infective endocarditis is a possible cause of death in drug addicts.
- In rare cases, SDH may not be a sequela of blunt trauma.
- A primarily intracerebral hemorrhage may perforate through the arachnoid forming an acute subdural hematoma.
- Although COD in drug addicts is predominantly accidental or an intentional overdose, this case illustrates that natural death can be a presentation.
- Procedures for conducting a complete autopsy vs. an external only autopsy (aka "view") in cases involving drug related deaths varies at each medical examiner's office. Frequently young decedents with a drug abuse history or death scene indicating drug abuse receive only a view and toxicology.
- These findings indicate why it is important to conduct a comprehensive autopsy of deaths related to drug abuse.

Acknowledgements

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