G72 Ruptured Intracerebral Arteriovenous Malformations, Hypertensive Cardiovascular Disease, and Acute Hypertensive Effects of Stimulants: Is There a Direct Link?

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The goal of this presentation is to provide an understanding of the relationship between hypertensive cardiovascular disease and rupture of intracerebral Arteriovenous Malformations (AVMs).

This presentation will impact the forensic science community by improving the understanding of the impact of hypertensive cardiovascular disease on the natural history of intracerebral AVMs. The relationship of AVM rupture with the acute hypertensive effects of drugs will also be explored.

Currently available data regarding natural history of AVMs is conflicting; clinical studies have been limited by small sample size, young age of patients (prior to peak incidence of hypertension), and lack of long term follow-up.[1-5] Accordingly, some have concluded that hypertension has no impact on rupture of AVMs, while other sources include hypertension as a major risk factor for AVM rupture.[4-6] Therefore, the risk of hypertensive cardiovascular disease on intracerebral AVMs is presently unclear. Whether rupture of an AVM may be precipitated by or associated with an acute hypertensive episode caused by substance abuse (e.g., cocaine, methamphetamine, and phencyclidine) has direct relevance for manner of death.

A retrospective cohort study was designed in which all autopsy case files between January 2005 and July 2012 were reviewed to identify adult decedents (age >18 years) in which an AVM was listed as a cause of death. Cases were excluded if the decedent had a remote history of neurosurgical intervention. Other types of intracranial fistulas (i.e., dural arteriovenous malformations) and nonfistulous malformations (i.e., cavernous hemangiomas) were excluded. One case of unruptured AVM was included.

A total of 11 cases were identified that met study criteria. Relevant biographical and historical information was collected. Gross autopsy and histopathological data were collected on each decedent to assess for the presence or absence of cardiomegaly, left ventricular hypertrophy, end organ (kidney) damage, and extent of involvement and chronicity of the AVM. Heart weights were compared to expected heart weights determined from a model described by Gaitskell *et al*. in 2011.[7]

Seven of the 11 cases (64%) were male and 4/11 (36%) were female, with an average age of 44 years (range 29 – 58 years). Average body weight was 177 pounds (range 118 – 248 pounds) and average height was 66.5 inches (range 62 – 70 inches). The average body mass index, characterized as overweight, was 28.2kg/m^2 (range 17.9 – 39.1kg/m^2). The cases comprised of mostly (91%) ruptured AVMs with associated hemorrhage: Subarachnoid (SAH) 8/10 (80%); subdural (SDH) 2/10 (20%), and intraparenchymal 5/10 (50%).

Autopsy heart weights averaged 422g (range 250 – 690g) with an average left ventricular wall thickness of 1.5 cm (range 1.0 – -2.0cm). Four of ten (40%) decedents with ruptured AVMs had both cardiomegaly and left ventricular hypertrophy (left ventricular thickness >1.4cm). Five of the ten (50%) ruptured cases had Left Ventricular Hypertrophy (LVH) and 3/4 (75%) of the cases with cardiomegaly and LVH also had evidence of hypertensive kidney disease (nephrosclerosis and arteriolosclerosis). Ultimately, six out of ten (60%) of the cases with ruptured AVMs had evidence of hypertensive disease as evidenced by one of the following: LVH, cardiomegaly, or hypertensive kidney disease. A clinical history of hypertension was identified in two of 11 (36%) of the cases (each confirmed by histopathology); one case was an unruptured AVM and one was a ruptured AVM. Finally, two of the ten (20%) decedents with ruptured AVMs also had acute cocaine toxicities; one had both hypertensive heart and kidney disease, the other had histopathologic evidence of hypertension. Each was classified as an accident.

Ultimately, it cannot be concluded from these data that hypertensive cardiovascular disease is a direct risk factor for rupture of intracerebral AVMs. However, the low percentage of hypertension in decedents with ruptured AVMs suggests the association is weak at best. This data would support a cautious approach when assigning manner of death to decedents who died from a ruptured AVM and also had stimulants detected during autopsy. Furthermore, whether to include underlying hypertension as a contributory cause of death is not clear. A major limitation of this study is the low number of study subjects.

References:

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Arteriovenous, Malformation (AVM), Hypertension

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