## G21 Cocaine Induced Intracerebral Hemorrhage in a Patient With Cerebral Amyloid Angiopathy: A New Risk Factor for Stroke in Cocaine Users

*Daniel G.K. Phillips, MD*, University of Maryland, Baltimore, Department of Pathology, 22 South Greene Street, Baltimore, MD 21201; Joyce L. deJong, DO, and Michael A. Markey, MD, Sparrow Forensic Services, Sparrow Hospital, 1215 East Michigan Avenue, Lansing, MI 48909-7980; and Rudy J. Castellani, MD, University of Maryland, Department of Pathology, 22 South Greene Street, Baltimore, MD 21201*

The goals of this presentation are to: (1) understand the association between intracerebral lesions and cocaine use, (2) recognize amyloid angiopathy, it's relationship with stroke as well as age, and it's immunohistochemical detection, and (3) recognize amyloid angiopathy as a possible contributing factor for hemorrhage in cocaine use.

Because this case represents the first reported association between cocaine-induced hemorrhage and cerebral amyloid angiopathy, this presentation will impact the forensic science community by bringing to light that amyloid angiopathy may be an under-recognized but significant risk factor for hemorrhage in older cocaine users.

Hemorrhagic stroke is a common complication of recreational cocaine use. The precise mechanism of hemorrhage in such patients is unclear, although vasospasm, ischemia, vascular thombi/thromboemboli, elevated blood pressure, and vasculitis have all been implicated. Systemic hypertension and saccular aneurysms are generally accepted as predisposing factors for cocaine-induced stroke. The authors report the case of a 62-year- old woman who suffered left parieto-occipital intracerebral hemorrhage with herniation and death, following a cocaine binge. In addition to the gross neuropathological findings, microscopic examination showed marked cerebral amyloid angiopathy in the vicinity of the hemorrhage as well as cortical areas. To explore the issue of chronic cocaine use as a risk factor for cerebral amyloid angiopathy per se, we additionally studied brain tissue in eight patients between the ages of 60 and 80 who were positive for cocaine metabolites at autopsy, with the presumption being that patients in this age group with evidence of cocaine use at autopsy were most likely chronic cocaine users. None of these additional subjects had vascular deposits of amyloid-beta by immunohistochemistry.

In conclusion, to the best of our knowledge, this report represents the first case of cerebral amyloid angiopathy-associated intracerebral hemorrhage precipitated by cocaine. It is suspected that other cases occur but go under-reported, on the one hand because cocaine-induced stroke is widely recognized and additional predisposing factors (e.g., amyloid angiopathy) may not be specifically sought, and on the other because cocaine may not be suspected in the advanced age at which amyloid angiopathy typically presents. It is further suggested that cerebral amyloid angiopathy occurs independently of the effects of cocaine, as no vascular labeling was found for amyloid-beta in eight older subjects who were cocaine users.

**Cocaine, Amyloid Angiopathy, Intracerebral Hemorrahge**

 ** Presenting Author*