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Segmental arterial mediolysis: A clinical-pathologic review, its role in fibromuscular dysplasia and description and differential diagnosis of the masquerader-muscular artery cystic necrosis

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ABSTRACT

Segmental arterial mediolysis (SAM) is an uncommon non-inflammatory iatrogenic arteriopathy caused by alpha-1 adrenergic agonists or Beta-2 agonists able to release norepinephrine from the peripheral nervous system. Causative agents include adrenergic agonists used to control blood pressure, B-2 tocolytic agonists, and ractopamine used as a repartitioning agent in animal husbandry. The liberated norepinephrine both injures and stimulates a robust reparative response in the muscular arteries in the abdomen, brain base, and coronary arteries. This response may be augmented by endothelin-1 formed in the arterial adventitia. Three types of arterial lesions develop in the injurious stage: 1) apoptotic induced mediolysis, 2) separation of the outer media from the adventitia and 3) the formation of arterial gaps. The latter enlarge, particularly in elderly patients, to form gap-aneurysms complicated by dissections and dissecting aneurysms that when ruptured cause the calamitous hemorrhages that clinically announce SAM. The other types of injury remain clinically silent but with repair develop sequelae and can metamorphose into fibromuscular dysplasia. The sequelae are mainly asymptomatic but may cause arterial stenosis and ischemic lesions. The definitive diagnosis of SAM requires histological conformation but misinterpretation of smooth muscle vacuolar change has caused diagnostic errors. Muscular artery cystic necrosis a newly named non-inflammatory muscular artery arteriopathy may be confused with SAM both clinically and pathologically. This arteriopathy represents the muscular artery equivalent of cystic media necrosis of the elastic arteries since it exhibits similar morphologic features and can occur concomitantly with this entity. Adrenergic agents to counter hemorrhagic shock in SAM are contraindicated since they may intensify injury and create new lesions. The use of norepinephrine antagonists introduces a new, but as yet untested, treatment option for SAM.

KEYWORDS

Segmental Arterial Mediolysis; Norepinephrine; Adrenergic Agonists; Ractopamine; Arterial Apoptosis; Aneurysms; Dissecting Hematomas; Arterial Fibromuscular Dysplasia; Muscular Artery Cystic Necrosis

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