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DISSEMINATED MICROINFARCTIONS WITH CEREBRAL MICROBLEEDS

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Abstract

Disseminated microinfarctions are uncommonly encountered in clinical practice. Here we describe a patient with longstanding cerebral microbleeds who developed acute cognitive decline in the setting of acute hypotension. MR imaging showed acute disseminated microinfarctions, with no change in microbleeds. This case emphasizes the important relationship between ischemic and hemorrhagic microvascular disease of the brain, especially in the setting of acute blood pressure changes compounding preexisting microvascular injury.

Cerebral microinfarcts manifest as acute lesions seen on diffusion-weighted imaging (DWI) sequences of brain MRI, and then either disappear or become incorporated into confluent white matter lesions (1,2). Disseminated microinfarctions are uncommon, and when present are usually associated with bacterial endocarditis, other cardiac disorders, carotid artery disease, aortic arch disease, and fat embolism (3–5). We describe herein a patient who developed disseminated microinfarctions in the setting of extensive pre-existing microvascular disease, including prominent cerebral microbleeds.

Case Report

A 78 year old African-American male was admitted for shortness of breath and altered mental status. The patient had a history of multiple strokes, hypertension, adult-onset diabetes, hyperlipidemia, smoking, and chronic kidney disease. MRI the previous year showed small deep infarcts, severe white matter disease, and extensive cerebral microbleeds, mostly deep hemispheric (Figure). MRA of the head and neck showed no occlusive lesions. The presentation included temperature 97.5F, blood pressure 111/65, and O2 saturation 99%

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on supplemental oxygen. The patient was alert with limited verbal output and oriented to name and place. CT chest suggested aspiration pneumonia, antibiotics were started, and mental status improved. However, following an emesis, he became unresponsive as blood pressure dropped to 92/57, he required up to 14mcg/min norepinephrine to maintain his blood pressure, and developed acute renal failure. O2 saturation was 89% with supplemental oxygen, drainage was obtained on suction, and the patient was intubated for airway protection and started on broad spectrum antibiotics. Blood cultures were repeatedly negative, and there was no evidence of microangiopathic hemolytic anemia. MRI brain revealed innumerable scattered punctuate foci of restricted diffusion (Figure). Transesophageal echocardiogram with bubble study showed no embolic source. Follow-up MRI showed mild improvement of the DWI lesions, and no change in white matter disease. The patient continued to decline, transitioned to comfort care, and expired.

Discussion

This patient developed disseminated microinfarctions in the setting of an acute hypotensive episode. The patient had longstanding microvascular disease, with extensive cerebral microbleeds and white matter disease. The distribution of microbleeds suggested a likely etiology of hypertension and chronic kidney disease, but cerebral amyloid angiopathy could not be entirely ruled-out.

Cerebral microbleeds and white matter disease have a strong association (6). This has suggested a common etiology based on arteriolar injury and dysfunctional regulation of cerebral blood flow, with hypotension and hypertension having pathophysiologic importance for microinfarctions and microbleeds, respectively (7). We suggest that this patient's disseminated microinfarctions were precipitated by acute hypotension in the setting of chronic profound preexisting microvascular disease, consistent with prior reports (8,9).

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Figure.

A-E Gradient echo images through the cerebrum and basal ganglia demonstrate innumerable susceptibility artifact indicative of microbleeds, established on prior imaging (A,B) in 2016; images from 2017 shown in C-E.

F: T2-FLAIR image demonstrates confluent subcortical and periventricular leukoaraiosis seen throughout deep and subcortical white matter.

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G-L: Diffusion-weighted images display innumerable disseminated foci of restricted diffusion consistent with microinfarctions throughout the entire brain

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