Intracerebral microbleeds in sepsis: susceptibility-weighted MR imaging findings

Micro-hemorragias intracerebrais na sepse: achados de imagem na RM ponderada em susceptibilidade magnética

Diogo Goulart Corrêa[1], Luiz Celso Hygino Cruz Júnior[2], Paulo Roberto Valle Bahia[3], Emerson Leandro Gasparetto[2,3]

[1]MD; Medical Resident of the Department of Radiology, Universidade Federal do Rio de Janeiro (UFRJ), Rio de Janeiro RJ, Brazil;

[2]MD; Neuroradiologist at Clínica de Diagnóstico por Imagem/Diagnósticos da América S/A (CDPI/DASA), Rio de Janeiro RJ, Brazil;

[3]MD, Ph.D; Assistant Professor of Radiology (Neuroradiology), UFRJ, Rio de Janeiro RJ, Brazil.

Correspondence: Diogo Goulart Corrêa; Rua Rodolpho Paulo Rocco 255 / Cidade Universitária / Ilha do Fundão; 21941-913 Rio de Janeiro RJ - Brasil; E-mail: diogogoulartcorrea@yahoo.com.br

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We present two patients with sepsis and intracerebral microbleeds. The first case is a nine years old girl who presented visual hallucinations, tremors in the limbs, and an episode of generalized tonic-clonic seizure in the 12th day of an otherwise successfully treatment of a pulmonary sepsis. Brain magnetic resonance imaging (MRI) showed numerous small rounded foci of decreased signal intensity on susceptibility-weighted imaging (SWI) spread throughout the brain, predominantly in the corpus callosum (Fig 1), which had high signal intensity on the phase map of SWI, suggesting blood deposits. The remaining conventional MRI sequences were normal. The patient and her mother denied any history of head trauma. During hospitalization, platelets counts, partial thromboplastin time, prothrombin time, and international normalized ratio were always normal.

The second patient is a 40 years old woman treating a septic shock of urinary origin for three weeks, who presented generalized tonic-clonic seizures. SWI showed linear low signal intensity on the cortex surface, mainly in frontal lobes, and multiple foci of low signal intensity on the subcortical white matter and cerebellum, which had high signal intensity on the phase images of SWI, suggesting areas of subarachnoid hemorrhages in the frontal lobes and microbleeds into the subcortical white matter and cerebellum (Fig 2). During hospitalization, D-dimer was normal. Although she had some altered values in platelets count (100,000/mm^3, was the lower value), prothrombin time (worst INR value was 2.3), and partial thromboplastin time, due to sepsis, she did not developed disseminated intravascular coagulation.

The typical imaging features of intracerebral microbleeds are small foci of decreased signal intensity on gradient-recalled echo T2* and/or SWI on MRI, usually without correspondence on others sequences[1]. Generally, microbleeds are related with hemorrhagic transformation of an ischemic stroke, recurrence of spontaneous intracerebral bleeding, cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy, cerebral amyloid angiopathy and trauma[1]. There are few studies correlating intracerebral microbleeds with infective endocarditis[2], but none with other causes of sepsis.

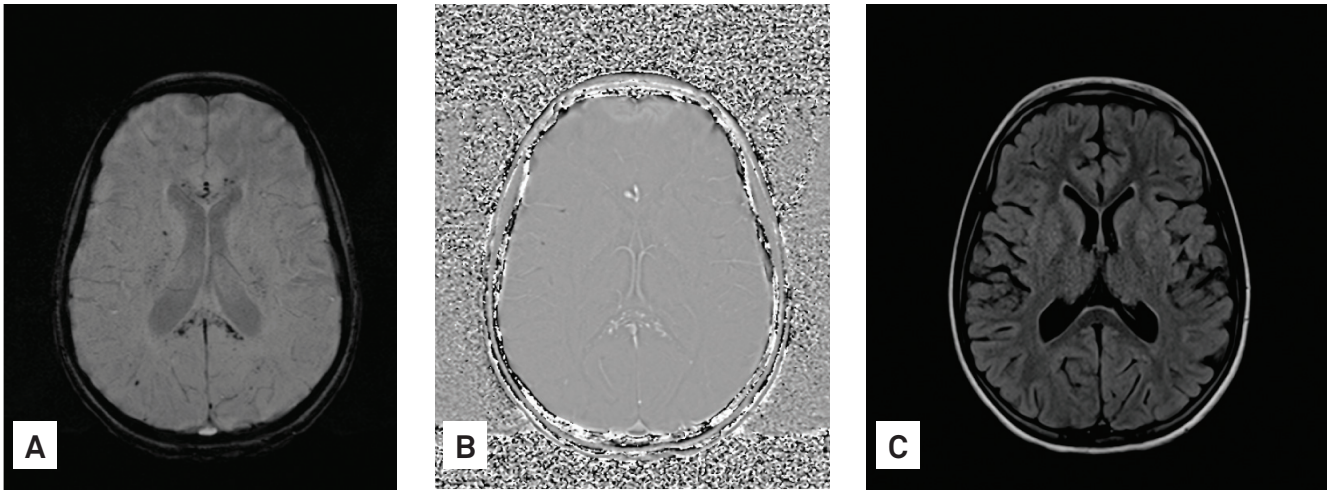

Fig 1. Susceptibility-weighted imaging (A) and phase images of susceptibility-weighted imaging (B) show multiple foci of low signal intensity on genu and splenium of the corpus callosum and subcortical white matter on susceptibility-weighted imaging, with high signal intensity on the phase images of susceptibility-weighted imaging, suggesting blood deposits. FLAIR image (C) on the same position shows no abnormalities.

Histopathological analysis of these microbleeds shows focal hemosiderin deposition, which can be an evidence of microangiopathy[1]. Furthermore, there is evidence of microcirculation dysfunction, including the cerebral, due to sepsis[3]. Then, we hypothesize that endothelium dysfunction, generated by sepsis of any origin, and may be a cause of intracerebral microbleeds.

Gradient-recalled echo T2* and SWI are especially sensitive for detection of these kind of hemorrhages. These sequences have been recently incorporated into the daily practice, but they are not always done in a sepsis scenario, because the majority of these patients are clinically unstable and needs a fast exam. Thus, the presence of intracerebral microbleeds in patients with sepsis may be more common than we currently suppose. Probably, this type of bleeding is not being detected on these patients because the more sensitive sequences are not being performed.

SWI is more sensitive than gradient-recalled echo T2* in detecting size, number, volume, and distribution of hemorrhagic lesions[4]. Then, if SWI is available, this sequence should be performed, even in detriment of gradient-recalled echo T2* in order to save time.

Furthermore, intracerebral microbleeds are a frequent finding in brain MRI, even in healthy people. Widespread use of gradient-recalled echo T2* and/or SWI has increased their detection in several diseases, in which generate great concern for clinical management, such as in ischemic stroke[5], spontaneous intracerebral bleeding, cerebral amyloid angiopathy and trauma[1]. In this meaning, the importance of intracerebral microbleeds needs more investigation.

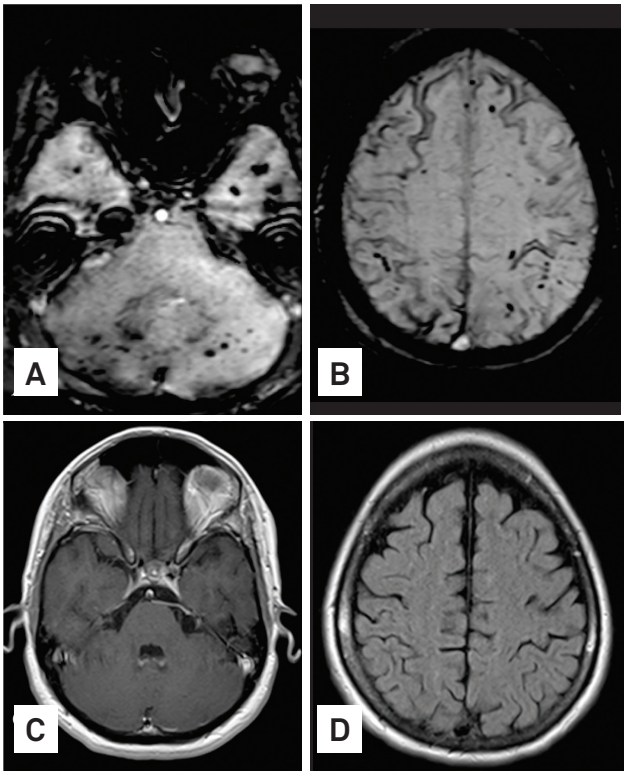

Fig 2. Susceptibility-weighted imaging showing the posterior fossa (A) and at the skull convexity (B) demonstrate multiple foci of low signal intensity on the subcortical white matter and cerebellum, suggesting microbleeds, and linear low signal intensity on the surface of the frontal cortex, compatible with subarachnoid hemorrhage. Post-contrast-T1 weighted image on the same position as A (C) and FLAIR image on the same position as B (D) show no abnormalities. The rest of the exam did not show any sign of aneurysms or meningitis.

References

1. Koennecke HC. Cerebral microbleeds on MRI Prevalence, associations, and potential clinical implications. Neurology 2006;66:165-171.
2. Klein I, Iung B, Labreuche J, et al. Cerebral microbleeds are frequent in infective endocarditis: a case-control study. Stroke 2009;40:3461-3465.
3. Lundy DJ, Trzeciak S. Microcirculatory dysfunction in sepsis. Crit Care Clin 2009;25:721-731.
4. Mittal S, Wu Z, Neelavalli J, Haacke EM. Susceptibility-weighted imaging: technical aspects and clinical applications, part 2. AJNR Am J Neuroradiol 2009;30:232-252.
5. Conforto AB, Lucato LT, Leite CC, Evaristo EF, Yamamoto FI, Scaff M. Cerebral microbleeds and intravenous thrombolysis: case report. Arq Neuropsiquiatr 2006;64:855-857.