CASE REPORT

Nonaneurysmal sulcal subarachnoid hemorrhage in a patient with atherosclerotic intracranial stenosis

Ana Gouveia¹, Ana Inês Martins¹, João Sargento-Freitas¹-³, Luciano Almendra¹, Fernando Silva¹-², Bruno Rodrigues¹, Cristina Machado¹, Gustavo Cordeiro¹, and Luís Cunha¹-³

¹Neurology Department, Coimbra University and Hospital Centre, Coimbra, Portugal
²Neurosonology Laboratory, Neurology Department, Coimbra University and Hospital Centre, Coimbra, Portugal
³Faculty of Medicine of the University of Coimbra, Coimbra, Portugal

Correspondence: Ana Gouveia
Department of Neurology, Centro Hospitalar e Universitário de Coimbra
Praceta Mota Pinto 3000-075 Coimbra, Portugal
Email: anargouveia86@gmail.com

Abstract

Background: Nonaneurysmal sulcal subarachnoid hemorrhage (sSAH) is a rare cause of cerebrovascular disease and represents a small proportion of nontraumatic SAH.

Case report: We report the case of a 54-year-old male patient, who presented with severe abrupt headache, followed two days later by right visual field defect and dysphasia. Head CT revealed left peri-rolandic sSAH and two hypodense lesions (left cortical parieto-occipital and left subcortical parietal). Additional investigation found a severe MCA atherosclerotic stenosis.

Discussion: The case reported represents a rare cause of sSAH. We discuss possible pathophysiological mechanisms.

Keywords: Sulcal subarachnoid hemorrhage, Atherosclerotic disease, Intracranial stenosis
Nonaneurysmal sulcal SAH due to atherosclerotic intracranial stenosis

Introduction

Nonaneurysmal spontaneous sulcal subarachnoid hemorrhage (sSAH) is defined as an hemorrhage located in the convexity of the brain, without involvement of adjacent parenchyma or extension into interhemispheric fissure, sylvian fissure, basal cisterns or ventricles. It is a rare cause of cerebrovascular disease and represents 5% of SAH [1]. We report the case of a patient with an exceptional cause of sSAH.

Case Report

A 54-year-old male patient presented with severe abrupt holocranial headache. Two days later, he noticed right visual field defect and fluctuating speech difficulties. Neurological examination revealed right homonymous hemianopia, dysphasia, alexia and dyscalculia. The patient’s medical history included active smoking, hypertension and dyslipidaemia.

The initial head CT revealed left peri-rolandic sSAH and, at the fourth day, two new hypodense lesions (left cortical parieto-occipital and left subcortical parietal). Brain MRI performed in the 7th day of symptoms confirmed the presence of two subacute ischemic lesions in the aforementioned locations (Figure 1).

Transcranial color coded Doppler (TCCD) evaluations were performed repeatedly during the first 10 days and found a persistent focal acceleration in left distal M1 segment of middle cerebral artery (MCA) [Lindegaard Index 4.5] with downstream flow attenuation. The digital subtraction cerebral angiography excluded the presence of aneurysms and arteriovenous malformations and showed a severe focal stenosis in left distal M1 segment of MCA, with a typical atherosclerotic morphology (Figure 2).

The patient was treated with high-dose statin and, after head CT excluding rebleeding, dual antiplatelet therapy.

Six months later, the neurological examination reveals right inferior homonymous quadrantanopia and dyscalculia. There has been complete SAH reabsorption in the CT. In TCCD evaluation there is still evidence of severe left MCA stenosis.

Figure 1. Head CT (a, b, c): left peri-rolandic sSAH (a), left cortical parieto-occipital (b) and left subcortical parietal (c) lesions. Brain MRI (d – T2*, E – DWI, F – FLAIR) – sSAH (d) and subacute ischemic lesion (e and f).
Discussion

There are multiple known etiologies for sSAH, including vascular causes, namely, reversible cerebral vasocostriction syndrome (RCVS) [2], cerebral amyloid angiopathy (CAA) [3], cerebral venous thrombosis [4], vascular malformations [5] and Moyamoya disease or syndrome [6]; and nonvascular causes as brain tumors [7], abscesses [8] and coagulopathy [9]. The largest cohorts reported in literature suggest a difference in etiologies according to the age group. In patients younger than 60 years, RCVS is found more frequently, while in older patients CAA is more prevalent [10, 11].

Few cases of sSAH in patients with severe extracranial atherosclerotic disease have been reported, some of them in association with intracranial atherosclerotic disease [5]. Possible pathophysiological mechanisms are similar to that of Moyamoya disease. The severe arterial stenosis may trigger the development of fragile and dilated pial collaterals in watershed zones that, under an acute hemodynamic change, can rupture and cause an sSAH.

The association of sSAH with isolated intracranial atherosclerotic stenosis is exceptional. Our patient had two ischemic lesions in watershed areas and a sSAH ipsilateral to the severe MCA stenosis. We believe the same pathophysiological mechanisms may have been implicated.

sSAH is distinct from most SAH. The etiologic investigation of a sSAH can be challenging due to its multiple possible causes and their consequent therapeutic implications.

Abbreviations

CAA: Cerebral amyloid angiopathy; MCA: Middle cerebral artery; RCVS: Reversible cerebral vasocostriction syndrome; SAH: subarachnoid hemorrhage; sSAH: Sulcal subarachnoid haemorrhage; TCCD: Transcranial color coded Doppler

Competing interests

The authors declare no conflict of interest.

References


http://dx.doi.org/10.1007/s11910-013-0338-3

http://dx.doi.org/10.1212/WNL.0b013e3181d55efa