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Case Report

Internal carotid artery dissection associated with acute dengue virus infection: a case report

Annelise Akemi Higa Lee, Ana Carolina Montouro Stoarri, Renan Domingues

Neurology Department, Santa Casa de Misericórdia de São Paulo, São Paulo, Brazil

Annelise Akemi Higa Lee
a.higalee@gmail.com

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Abstract

Cervical artery dissections (CAD) can occur spontaneously or as a direct result of significant trauma. Viral infections, such as SARS-CoV2, influenza, and Epstein Barr, are risk factors for spontaneous CAD.
Dengue virus infections have dramatically increased in recent decades, and Brazil is one of the endemic areas.
The dengue virus can cause headache and neurological complications such as encephalitis, myelitis, Guillain-Barré syndrome, and myositis. No report has yet been found in the literature of dissection of the internal carotid artery secondary to dengue infection.
Our objective is to report the case of a patient with dissection of the internal carotid artery associated with acute dengue virus infection.

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Introduction

Cervical arteries dissection (CAD) is one of the main causes of ischemic stroke in the young population. The incidence of this disease is generally low, reaching approximately 2.6 to 3.0 per 100,000 people per year (1).

The clinical presentation includes an ipsilateral headache. The pain is usually severe and can mimic a migraine attack. Other common manifestations are ipsilateral Horner's syndrome, ischemic and hemorrhagic stroke. The diagnosis is confirmed by cranial and vascular neuroimaging showing arterial occlusion or stenosis, dissecting aneurysm, double lumen, or intramural hematoma. Most dissections result from cervical region trauma. Forty one percent of the CAD are spontaneous (2). These cases are often associated with risk factors such as connective tissue disease, arteriopathies, pregnancy, and acute infections.

The association between recent infection and cervical artery dissection has been reported in association with different viruses such as COVID-19, influenza, and Epstein Barr infection (3-6).

Dengue virus infection is a serious public health problemin Brazil and its incidence has grown (1). Acute dengue is associated with neurological manifestations, however, the neuropathogenesis of thesemanifestationsis still unclear. It is believed that viral factors, host immunologic system, and metabolic factors may be involved in the neurological manifestationsof this disease (7).

Case report

45 years old, male, born and living in São Paulo, admitted to the emergency room complaining of a headache refractory to analgesia, associated with myalgia and prostration, beginning 6 days before admission. He also reported fever (39°C) and diffuse skin rash. The headache was in the left frontal region with irradiation to the ipsilateral neck, throbbing, with nausea, and without photo- or phonophobia. No previous comorbidities were reported.

On physical examination, he was hydrated, normotensive, eupneic, with no pain on abdominal palpation. On neurological examination, no meningeal signs, no focal deficits, with normal fundoscopy. Laboratory tests showed no thrombocytopenia and no liver dysfunction. A positive NS1 antigen for dengue virus was positive.

Cranial computed tomography (CT) showed normal brain parenchyma, however, an asymmetry of the juxtacranial cervical internal carotid arteries was reported. CT and magnetic resonance (MRI) angiographyshowed a 0.9 cm pseudoaneurysm in thepetrous segment of the internal carotid artery. No other risk factors than acute dengue infection for CAD were found (Figures 1 and 2).

The patient was discharged a few days later and acetylsalicylic acid was introduced after acute dengue recovery.

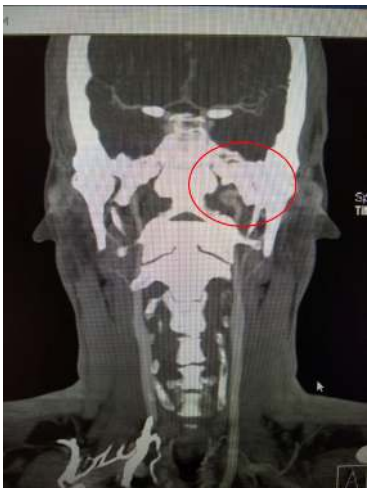

Figure 1. CT angiography showing left carotid artery pseudoaneurysm.

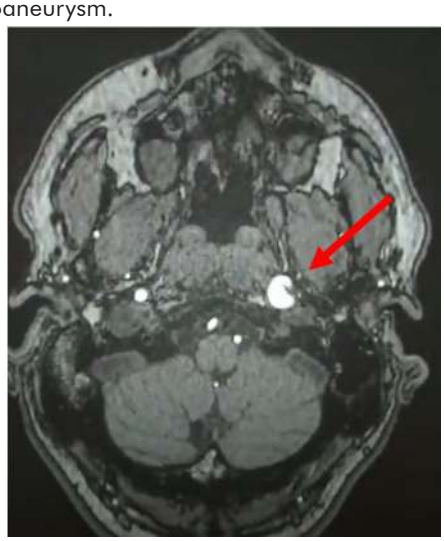

Figure 2. The left carotid artery pseudoaneurysm demonstrated by MRI angiography (arrow).

Discussion

As far as we know, this is the first report of CAD and headache associated with acute dengue. It could be argued that the headache reported by the patient was associated with acute dengue infection and not due to CAD. However, differently from what was reported by this patient, dengue headache is almost always bilateral, and photo- and phonofobia are usually reported (8).
The association of acute dengue with neurological manifestations, including stroke, has been reported.

Dengue associated stroke is usually a hemorrhagic stroke due to coagulation abnormalities. Other central nervous system (CNS) complications are strongly related with brain parenchymal abnormalities. However, cerebral arterial disease has not been directly implicated in CNS manifestations (9, 10).

In the present case, the acute dengue could have contributed to CAD through an indirect inflammatory response anda prothrombotic mechanism (2). In fact, dengue causes endothelial dysfunction and leads increased vascular permeability. These phenomena can determine hypovolemic shock and encephalopathy (11). At this point, it is not possible to rule out that an inflammatory vascular abnormality was involved in the arterial dissection detected in our patient. Future studies addressing cervical and cranial vessels in patients with dengue, especially when CNS manifestations are present, should clarify the relationship between this infection and cerebral and cervical arteries.

Conclusion

This case first describes the association between CAD and acute dengue infection. More studies are necessary to better understand the potential pathophysiological association between these two conditions.

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Annelise Akemi Higa Lee
https://orcid.org/0000-0002-0796-7156
Ana Carolina Montouro Stoarri
https://orcid.org/0009-0007-9460-206X
Renan Domingues
https://orcid.org/0000-0002-6058-7937

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