Spontaneous internal carotid artery dissection and acute HIV infection: a possible link?

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Background

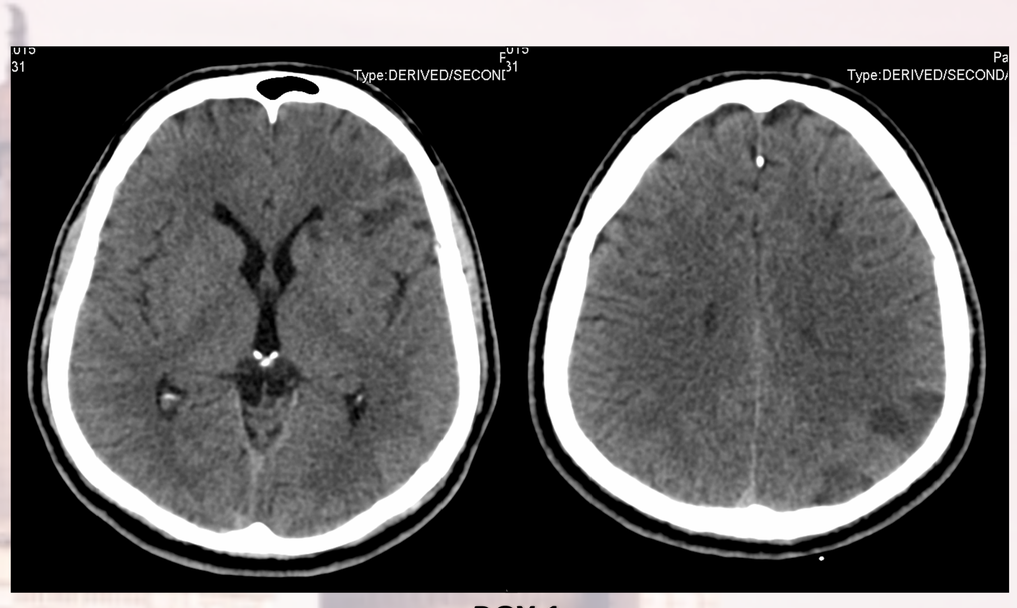
Recent infection is an emerging risk factor and a possible trigger for spontaneous cervical artery dissection (sCAD)[1-2]. HIV infection and related therapy are associated with cerebrovascular diseases[3], but few data exist on their relationship with sCAD.

Case report

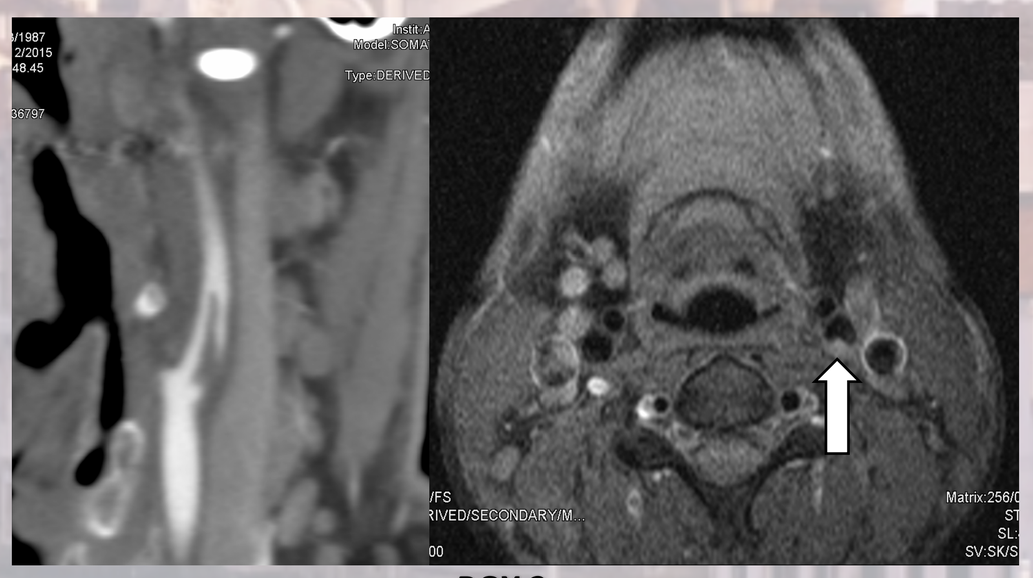
M.A., male, 29 years old, hospitalized for acute onset of severe left lateralized headache and cervical pain with neck stiffness.

- Clinical history: **fever** and widespread **rash** 10 days before
- Medical examination: no neurological deficits; **cervical lymphadenopathy**
- First-level diagnostic work-up: laboratory tests (**abnormal liver function**), brain CT (box 1), CSF analysis (**13 leukocytes** with normal proteins and glucose values), Doppler ultrasonography (**parietal hypoechoic lesion in the left internal carotid artery**)
- CT and MR Angiography (box 2)
- Diffusion weighted MRI (box 3) demonstrated the **acute ischemic nature of the cerebral lesions**
- No history of cervical trauma
- Patient was treated with broad spectrum antibiotic and antiviral therapy and anticoagulant therapy
- Whole body FDG-PET and CT: **enlarged liver and spleen, diffuse lymphadenopathy with reactive aspects and lung consolidation**

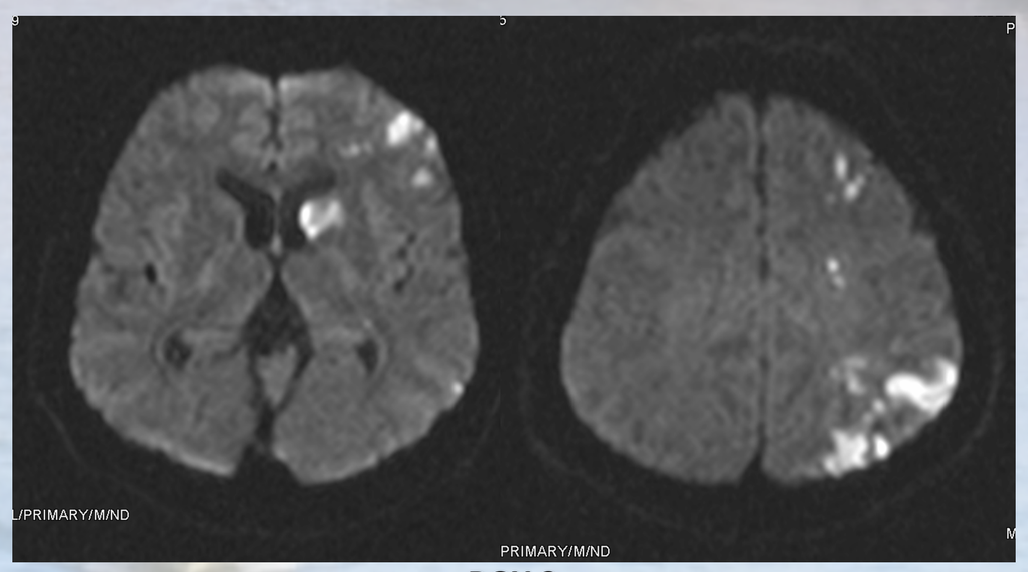
- **Serological tests: HIV-1 INFECTION with 188893 HIV-1 RNA copies/ml and 301 CD4+ T-cell count/ml, suggestive for acute HIV infection**



BOX 1
Multiple hypodense areas in the left frontal and parietal lobes and in the left caudate nucleus



BOX 2
Internal carotid artery dissection with free-floating luminal thrombus



BOX 3
Acute ischemic lesions in the territory supplied by the left internal carotid artery

DISCUSSION

To the best of our knowledge, there are only anecdotal reports of sCAD and chronic HIV infection, and this is the first description of sCAD occurred during an acute HIV infection. Some data suggest that recent infections may predispose to sCAD through: 1) indirect inflammatory and immunological host response with cytokines and proteases activation and excessive extracellular matrix degradation[1]; 2) endothelial damage and prothrombotic mechanisms[2]. These hypotheses are supported by the finding in sCAD patients of increased levels of inflammatory markers, leucocytosis, peri-arterial edema on cervical high-resolution MRI and seasonal incidence pattern[2].

References:
[1] Guillon B. et al, "Infection and the Risk of Spontaneous Cervical Artery Dissection: A Case-Control Study", Stroke. 2003 Jul; 34 (7): e79-81
[2] Debette S., "Pathophysiology and risk factors of cervical artery dissection: what have we learnt from large hospital-based cohorts?", Curr Opin Neurol 2014, 27:20-28
[3]Benjamin LA et al, "HIV infection and stroke: current perspectives and future directions", Lancet Neurol. 2012 Oct; 11 (10): 878-90.

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