# Giant cell arteritis mimicking vertebral dissection: a case report.

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## Introduction

Giant cell arteritis (GCA) is a systemic granulomatous vasculitis of elderly individuals affecting large and medium vessels. The amount of artery elastic tissue dictates the site of GCA involvment; superficial temporal, ophthalmic and posterior ciliary arteries are often affected, causing headache and visual disturbance. Vertebrobasilar ischemia due to vertebral involvement is known. Arteritis usually doesn't spread to intracranial arteries owing to lack of elastic fibers to which inflammation is targeted. Thus, cerebral ischemia is mainly determined by thromboembolism from larger extracranial arteries.(1)

## Case Report

A 72 year-old man – affected with diabetes and hypertension - was admitted to our stroke unit with neck pain, dizziness, nausea and gait ataxia. **Diffusion-weighted magnetic resonance imaging** (MRI) showed ischemia in the left lateral medulla oblongata**; CT angiography** demonstrated: 1)irregularity of the left vertebral artery (VA) wall in the proximal segment, "double lumen" appearance in the extra-intra cranial junction and signs of distal occlusion; 2)intradural right VA narrowing and irregularity. Lupus anticoagulant, anticardiolipin and antiphospholipid antibodies were absent. This had been interpreted as 'bilateral dissection' of the VA and antiaggregants had been started. After one month he developed acute dysarthria, dysphagia and right emiataxia; **MRI** revealed infarction in the right cerebellar pedicle and multiple bilateral brainstem ischemic lesions**. CT angiography** documented a longer narrowing of the right VA, irregularity and severe stenosis of the basilar and left middle cerebral arteries. **Color duplexsonography** of both temporal artery showed hypoechoic concentric wall thickening, characteristic of active-GCA(2). High-dose-prednisolone treatment was started and after three days biopsy of the right temporal artery was performed, showing luminal obstruction, disruption of the internal elastic lamina and hyperplasia of the intimal layer. Although the therapy, patient worsened to coma and died ten days later.

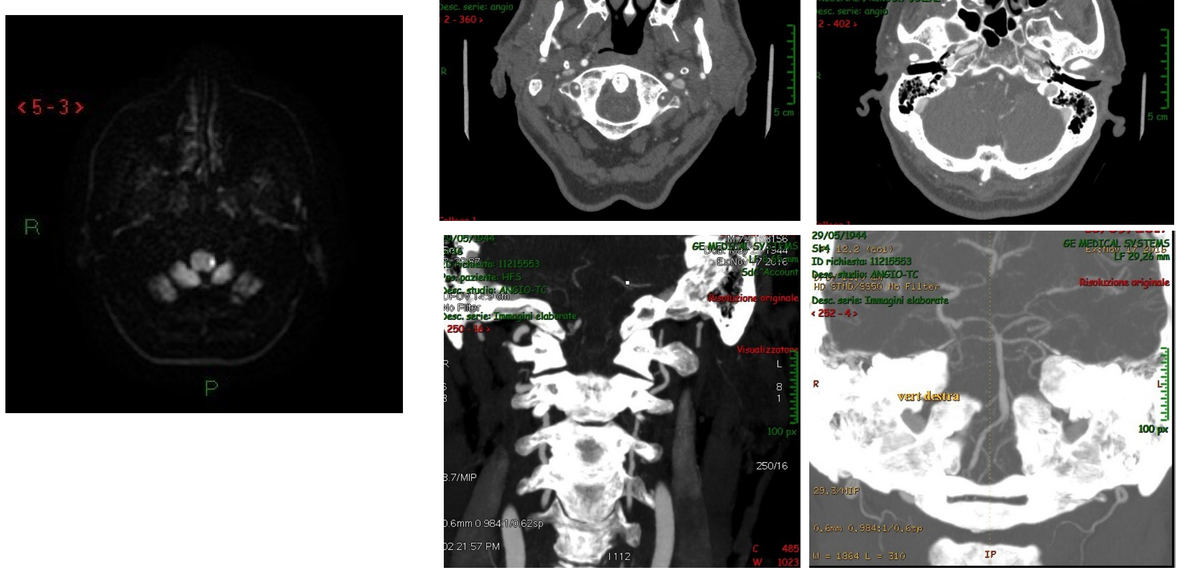
## Discussion

Spontaneous dissection and GCA of the VA might have similar clinical features(2). Occlusion of internal carotid and/or VA are the major causes of strokes in patients with GCA. Intracranial arteritis is rare and represents a subset with fatal course that fails to respond to corticosteroids. Vertebral dissection may link to GCA occurring secondary to chronic arteritis. Autopsy-proven GCA of the distal VA followed by dissection has already been described(3).

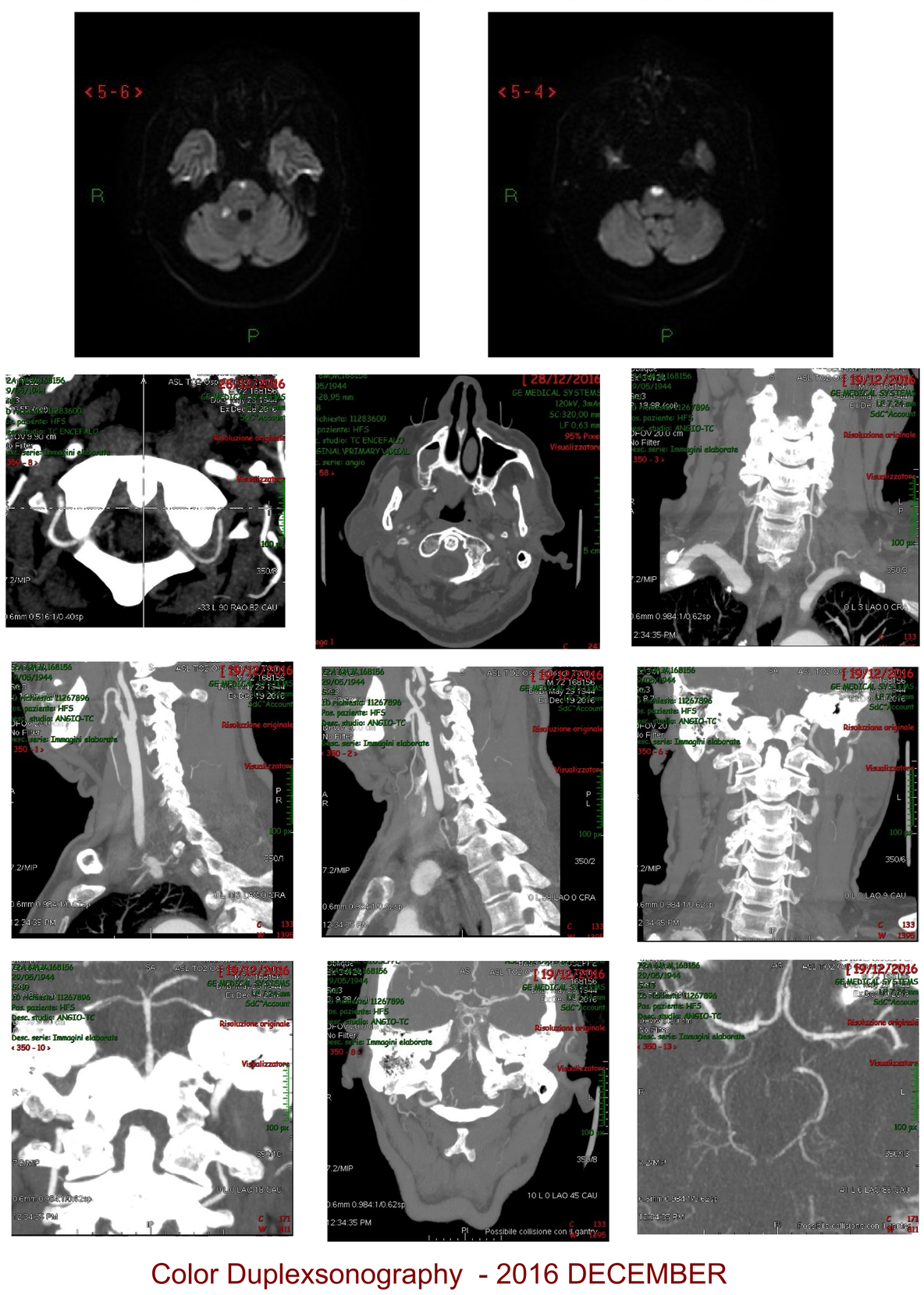
## Conclusion

In patients over 50 years presenting with vertebrobasilar ischemia due to stenosis, irregular thickening or dissection of VA, GCA should be considered. Diagnosis may be difficult without headache and in presence of atherosclerotic risk factors, but prognosis can be worsened by the delay of immunosuppressive treatment.

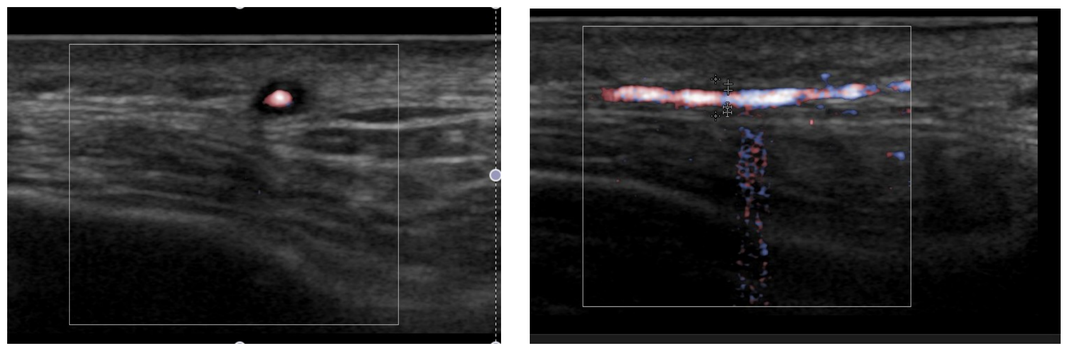
MRI AND CT ANGIOGRAPHY – 2016 NOVEMBER



MRI AND CT ANGIOGRAPHY – 2016 DECEMBER



Color Duplexsonography - 2016 DECEMBER



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