

## Stroke Mimic Caused by Acetazolamide

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### ABSTRACT

**Objectives:** To describe a case of a stroke mimic caused by iatrogenic ataxia due to acetazolamide.

**Case description:** An 86-year-old man with a history of gout and glaucoma, presented to the emergency department with progressive confusion, dizziness, disequilibrium and slurred speech, 3 days after he had started acetazolamide following ocular surgery. Physical examination showed he was hypertensive and had dysarthria; it was not possible to observe his gait due to pain in the right foot presumed to be due to a gout crisis. A stroke was thought to be the cause of these neurological deficits so a head CT scan was performed but did not show any alterations. During a stay in the stroke unit the neurological deficits remained unchanged and so, after review of the history, neurological side effects due to acetazolamide were suspected and the drug was suspended. A head MRI was performed to rule out stroke and the patient gradually improved. In the meanwhile, the patient was observed by an ophthalmologist and repeat surgery was proposed because of increased intraocular pressure.

**Conclusions:** Neurological deficits with a normal head CT scan in the emergency department pose many difficulties and require extensive knowledge of brain vascular anatomy and the differential diagnoses for stroke.

### LEARNING POINTS

- Not all focal neurological deficits are strokes.
- The need to diagnose stroke very quickly in order that effective treatment can be started can obscure the actual diagnosis so the differential diagnoses should always be carefully considered.
- Even though the sudden onset of focal neurological deficits suggests a stroke, a careful history should indicate the correct diagnosis.

### KEYWORDS

Acetazolamide, ataxia, stroke mimic

### INTRODUCTION

Acetazolamide is a carbonic anhydrase inhibitor commonly used after glaucoma surgery to decrease intraocular pressure. Some of the neurological side effects include ataxia, confusion, convulsions, depression, dizziness, drowsiness, excitement, fatigue, flaccid paralysis, headache, malaise and paresthesias<sup>[1,2]</sup>.

When a patient presents with acute focal neurological deficits and a normal head CT scan, stroke mimics should be carefully considered, which requires a broad knowledge of these pathologies and of stroke itself.

## CASE DESCRIPTION

An 86-year-old, previously independent man presented to the emergency department with confusion, dizziness, disequilibrium and slurred speech. Three days previously he had undergone a vitrectomy and had started on oral acetazolamide 250 mg id and ranitidine. He was also using eyewashes containing brimonidine, dexamethasone and bromfenac. A previous prescription for colchicine had been suspended due to hyperuricaemia. Physical examination showed the patient was hypertensive, while the neurological examination revealed he had dysarthria, bilateral dysmetria, ataxia and diminished strength in the lower left limb. As a stroke was suspected, a head CT scan was performed but was normal.

During the first few days in the stroke unit the deficits remained unchanged and the patient had an ataxic gait with small and unstable steps. A control head CT scan showed 'hypodensity suspected as being due to subacute ischaemic alteration in the right semioval centre'. This alteration was not compatible with the symptoms and so after the patient's history was revised and acetazolamide was checked for side effects, it was proposed the neurological deficits were caused by the acetazolamide even though the dosage was correct.

Acetazolamide was suspended and an MRI was performed to confirm the head CT scan changes. The patient gradually recovered from the neurological deficits over the following days, while the MRI was normal.

Meanwhile, an ophthalmologist had confirmed an increase in the patient's intraocular pressure so he was referred to a specialized centre for eye surgery.

## DISCUSSION

To our knowledge, stroke mimics caused by acetazolamide have not been previously described in the literature, although other side effects have been reported. Confusion is not a typical finding in patients with stroke and so should suggest an alternative diagnosis.

Some issues in our case made it particularly challenging. First, the patient had stopped colchicine and developed an ataxic gait due to a gout crisis, which made it harder to complete a neurological examination and to ascribe the disturbed gait to a neurological deficit. Second, the head CT scan repeated in the stroke unit showed a hypodensity corresponding to a subacute alteration, which made it a hard decision to suspend therapeutic and rehabilitation measures. Third, this could have been a vertebrobasilar stroke or bilateral synchronous embolism, both of which require optimal medical therapy and rehabilitation and may be followed by stroke recurrence. Fourth, before the results of the head CT scan were compared with the results of the head MRI, it was suggested that the patient required eye surgery, which he might not need if it was a stroke and he could resume acetazolamide.

In the emergency medicine context, the management of patients with focal acute neurological deficits with a normal head CT scan is challenging. The clinician must have a thorough knowledge of the differential diagnoses of stroke and of neuroanatomy and be able to link the imaging alterations to the clinical presentation.

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