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## Pontine infarction due to basilar artery stenosis presenting as pathological laughter

Received: 9 April 1996  
Accepted: 1 July 1996

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**Abstract** A 69-year-old woman presented right hemiparesis accompanied by pathological laughter (“fou rire prodromique”). The right hemiparesis progressed over several days and MRI revealed a left pontine infarct caused by basilar artery stenosis which was demonstrated by MRA.

**Key words** Pontine infarct · Basilar artery stenosis · Pathological laughter · Fou rire prodromique

### Introduction

Pathological laughter generally occurs following bilateral or diffuse lesions of the brain and usually takes the form of pathological laughing and crying [1]. Recently, there have been a number of case reports describing pathological laughter caused by isolated brain lesions, without pathological crying [2–4]. We describe a patient with a left pontine infarct presenting with progressive right hemiparesis accompanied by pathological laughter. Magnetic resonance angiography (MRA) revealed severe stenosis of the basilar artery.

### Case report

A 69-year-old housewife was admitted to hospital complaining of right-sided weakness occurring on the previous day. She was fully alert, well-oriented and cooperative. There was a moderate degree of right-sided facio-brachio-crural hemiparesis with mild dysarthria and a right extensor plantar response. In addition to these symptoms and signs, she started to experience occasional episodes of inappropriate and involuntary laughter triggered by various stimuli, most frequently when talking with someone. She did not feel happy, either during or after the laughing attack, and was puzzled by it, but she could not control the paroxysms. These episodes usually persisted for about 15 to 30 s, without pathological crying. Cranial CT and EEG performed on the day of admission were normal. The hemiparesis deteriorated progressively, and intravenous heparin (10,000 IU/24 h) was commenced the following

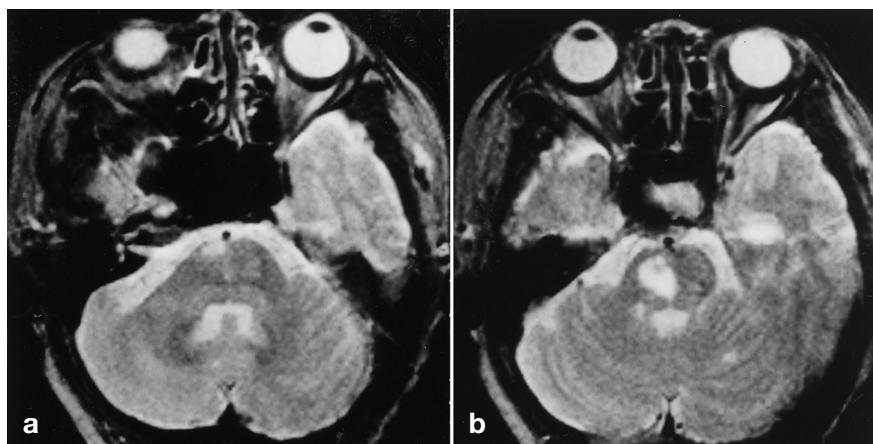
day. A repeat Cranial CT, 2 days after the first revealed a low-density lesion on the left side of the pons. MRI (Fig. 1) performed 10 days later demonstrated a ventromedial pontine infarct, and MRA (Fig. 2) revealed severe stenosis in the mid-portion of the basilar artery. Her right-sided hemiparesis had deteriorated for several days after admission and then gradually improved. Pathological laughter occurred over a period of about 3 weeks and then gradually became less frequent. Three months later, she had residual right-sided hemiparesis but was able to walk alone and no longer experienced pathological laughter.

### Discussion

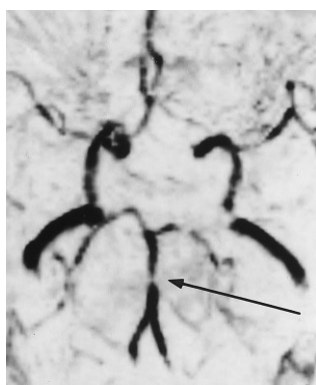
Laughter is termed pathological when it is inappropriate and continuous. It is not associated with expected mood changes and there is an absence of voluntary control over the extent and duration of the facial expressions [1, 4]. Our patient fulfilled the definition of pathological laughter.

Recently, there have been a number of case reports describing pathological laughter caused by isolated brain lesions without any other manifestations of pseudobulbar palsy or pathological crying [1–4]. Pathological laughter which occurs at the onset of cerebrovascular disease was described as “fou rire prodromique” in 1903, but has rarely been reported [4]. To our knowledge, there have been only two radiologically proven cases. Ceccaldi and Milandre [3] recently re-

**Fig 1** Axial T2-weighted MRI shows a left ventromedial pontine infarct extending to the basal surface



**Fig 2** Axial MRA shows severe stenosis in the mid-portion of the basilar artery (arrow)



ported one with left capsular-thalamic infarcts. Wali [4] reported a patient with “fou rire prodromique” caused by a brainstem infarct leading to a “locked-in” state. The lesion location in this patient was similar to ours. The exact mechanism of pathological laughing and crying are produced by the interruption of a control system, presumably lying at the base of the

brainstem, which can be involved at one of several levels extending from the medulla oblongata to the thalamus [1]. Poeck [1] speculated that there is tonic and phasic innervation of the movements of facial expression. In the “fou rire prodromique” there is loss of the tonic control of laughter, while in spasmodic pathological laughter there is loss of the phasic control. In our patient, it is possible that the ventromedial pontine lesion disrupted the tonic innervation of the faciorespiratory centre in the lower brainstem [4].

Patients with pontine infarcts extending to the basal surface, as in our case, often have basilar artery stenosis and a worse prognosis than those with deep pontine infarcts without surface extension [5, 6]. Additionally, it has been suggested that patients with vertebrobasilar territory infarcts who show progressive worsening have poor collateral circulation and a poor prognosis [7, 8]. Our patient presented with a progressive course with severe basilar artery stenosis demonstrated by MRA, but she was able to recover independence.

**Acknowledgement** We thank Dr. Hirohiko Murakami for his clinical assistance.

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