J Neurol (2009) 256:1017–1018
DOI 10.1007/s00415-009-5041-6

LETTER TO THE EDITORS

Sixth cranial nerve palsy and contralateral hemiparesis (Raymond's syndrome) sparing the face

P. Mégevand · B. Pilly · J. Delavelle · N. Tajouri · A. B. Safran · T. Landis · C. Lüscher

Received: 4 August 2008 / Revised: 6 January 2009 / Accepted: 28 January 2009 / Published online: 1 March 2009

The combination of VIth cranial (abducens) nerve palsy and contralateral hemiplegia is termed Raymond's syndrome [1–3]. Whether this rarely reported syndrome [4] necessarily includes central facial paralysis is debated [5].

Case report

A 74-year old man presented with horizontal diplopia of sudden onset, worsened by directing gaze to the left. His past medical history included arterial hypertension treated with an angiotensin conversion enzyme inhibitor, a 60-pack-per-year smoking history and moderate alcohol consumption.

Neurological examination showed moderate left VIth nerve paresis and slight paresis of the right arm and leg. Both spontaneous and voluntary facial movements were normal. Deep tendon reflexes were normal and symmetric. Cutaneous plantar response was flexor on both sides. There was no ataxia or sensory deficit. Gait was unremarkable.

Laboratory examinations were normal except for slightly raised liver enzymes. Magnetic resonance imaging, performed 2 days after the onset of symptoms, showed a lesion in the left caudal ventral paramedian pons (Fig. 1a, b). Magnetic resonance angiography as well as extra- and intracranial ultrasound with Doppler examination did not reveal any hemodynamically significant arterial stenosis. Heart rhythm remained regular during 24-h monitoring in the stroke unit.

The patient was not handicapped by the slight hemiparesis; diplopia resolved partially over several days. Acetylsalicylic acid was introduced.

Discussion

The French neurologist Fulgence Raymond (born 1844, died 1910) described the clinical picture that bears his name in 1895 [1–3]. Landry previously reported a similar patient with a hemisensory deficit in addition to hemiplegia [2, 6]. In both Raymond's and Landry's patients, the hemiplegia included central facial palsy. The lesion (probable syphilis in Raymond's patient, stroke in more recent reports) lies in the medial ventral caudal pons. Other eponymous syndromes of the caudal pons include Millard-Gubler's syndrome (peripheral facial palsy plus contralateral hemiplegia) and Foville's syndrome (peripheral facial palsy and horizontal gaze palsy plus contralateral hemiplegia), both caused by lesions extending more laterally and dorsally [2, 3].

A patient with VIth nerve paresis and contralateral hemiparesis sparing the face, caused by a lesion in the

P. Mégevand (✉) · T. Landis · C. Lüscher
Department of Neurology, Geneva University Hospitals and University of Geneva, Rue Micheli-du-Crest 24, 1211 Geneva 14, Switzerland
e-mail: pierre.megevand@medecine.unige.ch

B. Pilly · N. Tajouri · A. B. Safran
Ophthalmology Clinics, Clinical Neuroscience Department, Geneva University Hospitals and University of Geneva, Rue Micheli-du-Crest 24, 1211 Geneva 14, Switzerland

J. Delavelle
Radiology Department, Geneva University Hospitals and University of Geneva, Rue Micheli-du-Crest 24, 1211 Geneva 14, Switzerland

P. Mégevand · C. Lüscher
Basic Neuroscience Department, Faculty of Medicine, University of Geneva, Rue Michel-Servet 1, 1211 Geneva 4, Switzerland

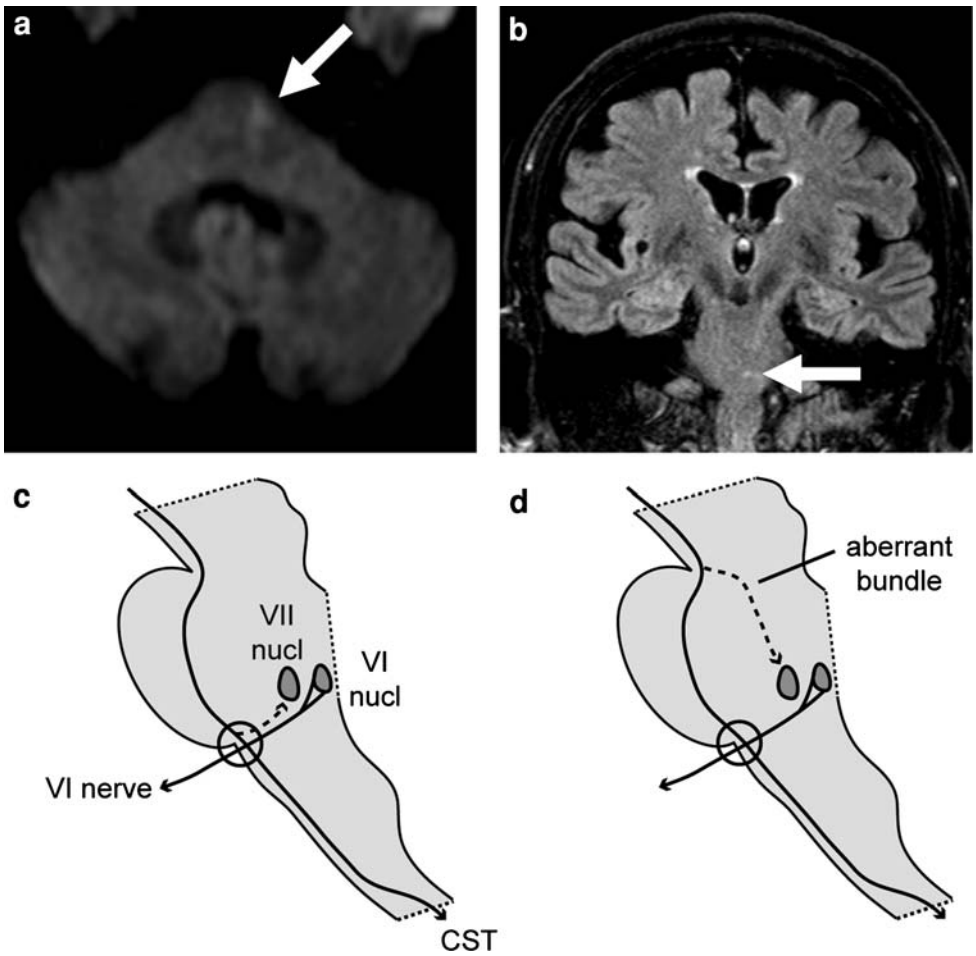

Fig. 1 Axial diffusion-weighted (**a**) and coronal FLAIR brainstem MRI (**b**) showing an ischemic lesion in the left caudal ventral paramedian pons (*arrows*). When corticofacial fibers (*dashed arrow*) travel with the corticospinal tract in the ventral pons, a lesion in the medial caudal ventral pons (*circle*) damages the corticospinal, corticofacial and VIth cranial nerve fibers together (**c**). If corticofacial fibers travel in an aberrant bundle in the pontine tegmentum, the medial caudal ventral pontine lesion damages only corticospinal fibers and the VIth cranial nerve, sparing the corticofacial projection (**d**). CST: corticospinal tract; VI nucl: abducens nucleus; VII nucl: facial motor nucleus

pontomedullary junction, was recently diagnosed with Raymond's syndrome [7]. Others suggested using the eponym only when central facial palsy is present [5]. This controversy likely reflects uncertainty about the anatomical course of corticofacial fibers in the brainstem. Corticofacial fibers can take one of several routes through the pons, including an "aberrant bundle" located close to the medial lemniscus in the pontine tegmentum [8]. This anatomical variability might reflect the phylogenetically recent appearance of the corticospinal and corticobulbar tracts [8]. Corticofacial fibers traveling dorsally in the aberrant bundle would not be involved by a ventral lesion of the caudal pons, producing Raymond's syndrome sparing the face (Fig. 1c, d). Alternatively, a very small pontine lesion may only partially damage the corticospinal and corticobulbar tracts. Regardless of the presence or absence of central facial palsy, the combination of VIth nerve palsy and contralateral hemiplegia should direct the clinician's attention towards the medial ventral caudal pons.

Acknowledgements P.M. is supported by grant #323600-111505 from the Swiss National Science Foundation.

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