

Thalamic transitory ischemic attacks presenting as Jacksonian sensory march

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Abstract Spreading somatosensory symptoms appearing as Jacksonian sensory march are usually considered to be due to an epileptic seizure. We report on three cases in which these symptoms were caused by thalamic ischemia. Two patients presented with stereotypically recurring hemiparesthesias lasting 2–5 min that gradually spread from the face to the arm and leg on one side. A first cerebral magnetic resonance imaging including DWI was negative in both cases, whereas new thalamic infarctions appeared on repeated imaging when clinical symptoms remained. A third case with a thalamic ischemia did not show recurring events, but also presented with purely sensory spreading symptoms. In all three cases EEG and cardiovascular diagnostics revealed normal results. Pure sensory stroke has previously been described as a result of ischemia of the thalamus or the internal capsule presenting as a sudden onset hemisensory deficit, but spreading symptoms have rarely been reported. According to our observations, thalamic TIAs are an important differential diagnosis of somatosensory epileptic auras presenting with Jacksonian sensory march which require a different clinical management.

Keywords Transitory ischemic attack (TIA) · Thalamic ischemia · Sensory stroke · Jacksonian sensory march · Partial seizures

Introduction

Transient somatosensory symptoms can be the result of different pathophysiological mechanisms: epileptic seizures, migraine auras or transient ischemic attacks (TIA). Clinical characteristics like the onset, development, duration of symptoms, and accompanying features are often the only possibilities to differentiate these diagnoses. Somatosensory epileptic seizures result from a disturbed network activity in the primary or secondary sensory cortex which is often visible by electroencephalographically propagating epileptic discharges in the respective brain regions [1]. The typical Jacksonian sensory march is characterized by a spreading sensation mostly beginning at a distal part of one limb [2]. Pure somatosensory seizures are rare. The sensory symptoms usually last for less than 5 min and are often accompanied by clonic movements or followed by postictal motor impairment [3].

Another differential diagnosis for transient focal neurological deficits is migraine with aura, especially in young patients without cardiovascular risk factors. Sensory auras occur with a frequency of 30–54%. Typically the sensory symptoms develop gradually over 5–15 min and last for 20–60 min until function is regained completely [4].

Case reports

Case 1

A 71-year-old male patient suffered from a repeated numbness starting from the left side of the face spreading to the left arm and leg within seconds and persisting for maximally 5 min before symptoms resolved completely. The symptoms recurred about six times per week in a

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Fig. 1 Diffusion weighed imaging of case 1 (**a, b**) and 2 (**c, d**) before and after the manifestation of thalamic infarction

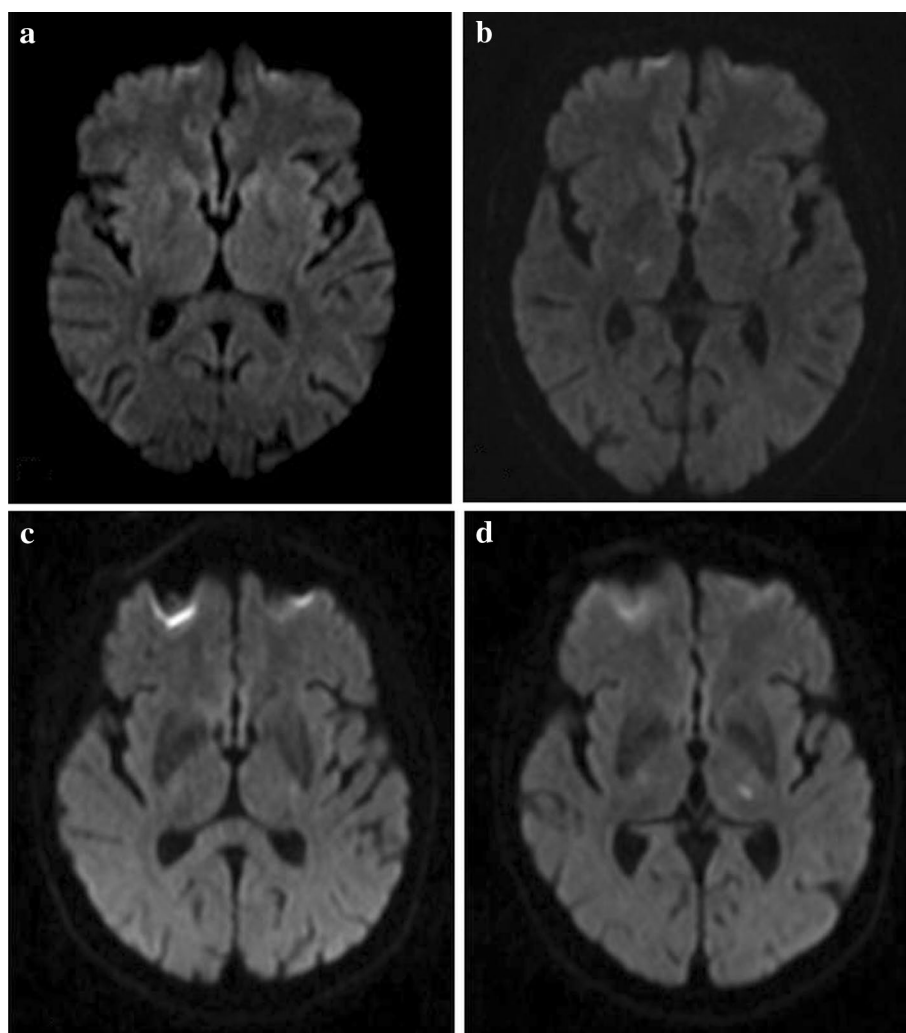
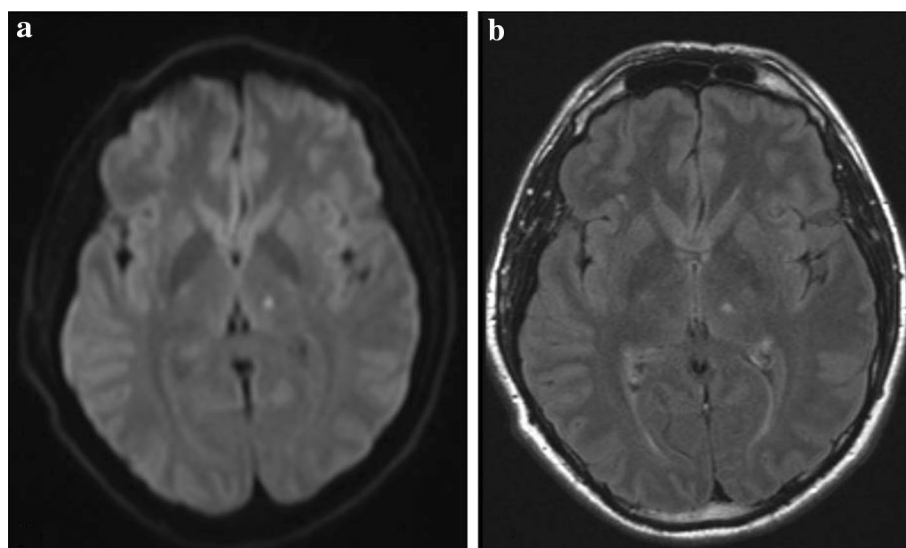


Fig. 2 DWI (**a**) and FLAIR (**b**) imaging of case 3



stereotypical manner. Apart from the recurrent paresthesia he felt completely normal and reported no other deficits, in particular no motor symptoms or problems of speech. He had common cardiovascular risk factors including diabetes and hypertension. Clinical examination between the episodes was normal. Cranial computed tomographic angiography (CT-A) and magnetic resonance imaging (MRI)

showed no acute ischemic lesions or stenosis of cranial arteries, although signs of a mild microangiopathy were observed. ECG, 24 h ECG, extracranial and transcranial sonography as well as an EEG were normal. The patient was dismissed with aspirin and simvastatin as a secondary prevention concerning his microangiopathy and levetiracetam as a new anticonvulsive medication.

Two weeks after the onset of symptoms, the patient presented with a persisting left-sided hemiparesis. A second cMRI showed a new lacunar infarction in the right thalamus (Figs. 1, 3).

Case 2

A 69-year-old male patient presented with recurrent right-sided paresthesias. He reported a feeling of pins and needles starting on his right forehead further spreading to his right arm and leg within a minute. The sensations were followed by a feeling of numbness. After 2–3 min the symptoms resolved and reappeared with a frequency of 2–3 episodes per day. He was a smoker and suffered from coronary artery disease treated with aspirin, and hypertension treated with a Ca²⁺-antagonist (lercanidipin). He admitted to take his medication irregularly. He had no neurological deficits on interictal clinical examination. A cranial CT-A scan showed no evidence of ischemia or bleeding. There was mild microangiopathy and a 50–60% stenosis (according to NASCET criteria) of the right internal carotid artery (ICA). Transcranial sonography revealed an additional stenosis of the right ACA and MCA. The MRI was normal except for another stenosis of the left ACP that was not observed by the other techniques. The EEG was normal. Further cardiac diagnostics including 24 h ECG and echocardiography showed a mild diastolic dysfunction of the left ventricle, but no other findings that would suggest embolic ischemia. Three days after onset of symptoms the patient complained about a persisting numbness in his right arm. MR imaging was repeated now showing a DWI-hyperintense area in the left lateral thalamus and dorsal internal capsule (Figs. 1, 3).

Case 3

A 48-year-old patient presented with sudden onset of tingling and reduced sensation of the right hand that spread within seconds to the right side of the face where it remained persistently. He reported no symptoms of weakness, jerking movements or speech problems. He was a non-smoker and had no history of hypertension or diabetes. Clinical examination showed no further neurological deficits. A cranial CT scan at the day of onset showed no signs of ischemic lesions, whereas an MRI on the following day showed a small infarction of the left medial thalamus (Figs. 2, 3). No relevant stenosis of intra or extracranial arteries was detected in

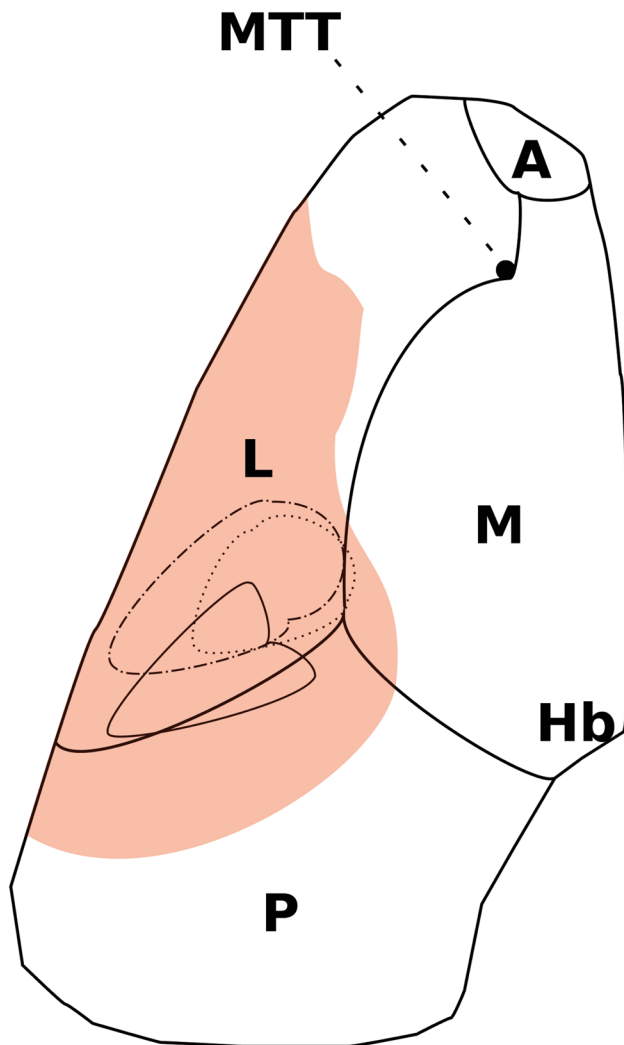


Fig. 3 Localization of the thalamic infarctions in case 1–3. Schematic drawing of the anterior (A), lateral (L), medial (M) and posterior (P) thalamic nuclei groups at the level of the habenulae (Hb) and the vascular territory of the inferior lateral thalamic branches (red shaded area, based on the schematic drawing De Freitas and Bogousslavsky [9]). The FLAIR hyperintense area of the infarction at this level of patient 1 (dot dashed line) as well as the horizontal mirrored FLAIR demarcation at this level of patient 2 (straight line) and 3 (dotted line). All the infarctions are located in a similar location in the dorsal aspects of the lateral nuclei group that consists of the ventrocaudal nucleus (Vc) based on the anatomic definition of Schaltenbrand or ventral posterior lateral (VPL), ventral posterior medial (VPM) and ventral posterior inferior (VPI) nucleus (anatomic definition of Morel)

CT-A and ultrasonography. 24 h ECG and transesophageal echocardiography were not suggestive for an embolic stroke. Further laboratory analyses (CSF, laboratory screening for vasculitis and thrombophilia) were normal. The patient was dismissed with a new medication of aspirin and simvastatin.

Discussion

In three similar cases, we here report clearly spreading sensory symptoms mimicking a Jacksonian sensory march, which were, however, caused by thalamic ischemia as proven on MRI. The symptoms were never followed by headache, appeared in high frequency and dissolved after minutes, which would be unusually short for a migraine aura. The stereotypical presentation and duration of symptoms made epileptic seizures the most probable diagnosis. The first patient, therefore, received an antiepileptic medication, which, however, did not change the nature and frequency of symptoms. In the third case, the symptoms persisted after the first event of a spreading sensation which made an epileptic seizure unlikely.

TIAs mimicking epileptic seizures are a rare phenomenon. Among those, sensory as well as motor symptoms have been described. For example, ischemic attacks due to an occlusion of the internal carotid artery can lead to temporary limb-shaking or jerking movements that should not be confounded with focal epilepsy. Transitory sensory symptoms caused by ischemia localized in the internal capsule have previously been described as ‘capsular warning syndrome’ [5]. However, this mostly involves recurring weakness of the arm and leg lasting for several minutes. In the context of capsular warning syndrome only one case has been described so far initially presenting with an isolated Jacksonian sensory march [6]. Pure sensory stroke is known to be caused by thalamic infarction. However, the symptoms usually have a sudden onset or occur in a stepwise fashion with sudden onset each [7]. Isolated hemisensory deficits are highly suggestive of the inferolateral region of the thalamus (ventromedial and ventrolateral nuclei) that is penetrated by small thalamogeniculate arteries arising from the posterior cerebral artery (P2) [8, 9]. Further though rarer localizations for pure sensory stroke include the pontine tegmentum and cortical areas of the parietal lobe [10].

Transient sensory symptoms are a diagnostic challenge, because one must rely solely on the patient’s report. Further diagnostics including cranial imaging, EEG and cardiovascular diagnostics are often negative. As a result, the treating physician is easily driven to assume and treat the diagnosis that seems most likely based on the clinical presentation. We showed two cases, in which the assumptions of epileptic seizures turned out to be wrong and ischemic attacks were not treated as such in the first place. It can hence be misleading to regard the spreading of sensory symptoms as a diagnostic tool.

Additionally, the third case suggests that it is more reliable to focus on the duration of symptoms than on the spreading character.

Thalamic ischemia might be more often the reason for spreading transient sensory loss than it is generally known, especially in patients with cardiovascular risk factors. We, therefore, intend to bring transient ischemic attacks of the thalamus to awareness as an important differential diagnosis in patients presenting with Jacksonian sensory march and to suggest to treat such patients accordingly—at least transiently—with a dual strategy to avoid persistent ischemia as best as possible.

Author contributions VS, study concept and design, acquisition of data. SL, acquisition of data. BB, imaging data and analysis. HL, study concept and design, critical revision of manuscript for intellectual content.

Compliance with ethical standards

Conflicts of interest Victoria Schubert—reports no disclosures. Dr. Lauxmann reports non-financial support from Eisai, outside the submitted work. Dr. Bender reports non-financial support from Bayer Vital, personal fees from Mevis, outside the submitted work. Dr. Lerche reports grants from German Federal Ministry of Education and Research, grants from Deutsche Forschungsgemeinschaft, during the conduct of the study; personal fees and other from UCB, from Desitin, personal fees and other from BioMarin, personal fees and other from Telethon, personal fees and other from Eisai, grants, personal fees and other from Bial, outside the submitted work.

Ethical standards All procedures have been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

Informed consent Informed consent was obtained from all three patients.

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