Tidsskriftet Tidsskrift for Den norske legeforening

A woman in her 40s with an unusual migraine attack

EDUCATIONAL CASE REPORT

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Background

Migrainous infarction is a rare complication of migraine with aura, accounting for 0.5–1.5 % of all ischaemic strokes. Migraine with aura increases the risk of ischaemic stroke, particularly in women using oral contraceptives. Migrainous infarction is defined as an ischaemic stroke that occurs during a typical migraine with aura attack and is associated with a neuroimaging-confirmed ischaemic brain lesion in the relevant area.

Case presentation

This case describes a woman in her forties with a history of migraine with visual aura who developed transient neurological symptoms – including vertigo, visual disturbances in the left eye and left-sided numbness and weakness – followed by a severe fronto-occipital headache. MRI revealed two small acute infarcts in the right occipital and posterior parietal lobes, consistent with migrainous infarction. No embolic source or other cause was identified in extensive cardiovascular, haematological and autoimmune investigations. The patient was treated with dual antiplatelet therapy and a statin for secondary prevention and made a full recovery with no recurrent migraine attacks.

Interpretation

This case highlights the importance of thorough anamnesis in patients with migraine. New or atypical aura symptoms, prolonged focal deficits, or changes in headache pattern should prompt urgent neurological evaluation and imaging to rule out ischaemic stroke. Early recognition is critical to ensure appropriate management and prevent long-term sequelae.

A woman in her forties was admitted as an emergency with a migraine attack that was unusually severe for her. This was accompanied by new, sequential aura symptoms, which proved to be a rare complication of migraine. This case illustrates the diagnostic challenges posed by atypical presentations and the importance of recognising them.

A woman in her forties with a history of hypertension and migraine with aura was admitted to the emergency department with suspected transient ischaemic attack (TIA). She had initially experienced sudden dizziness and left-sided symptoms, including blurred vision and facial numbness that gradually spread. After a few seconds, she developed weakness/paralysis of the left arm and leg, progressing from proximal to distal. The symptoms lasted for approximately 30 minutes in total. As they began to resolve, she developed a fronto-occipital headache of moderate to severe intensity (Numeric Rating Scale (NRS) 8). Because the headache was more severe than usual, she contacted the out-of-hours primary care service two hours after symptom onset. The transient motor deficit raised suspicion of a diagnosis other than migraine, and she was referred to the emergency department of her local hospital. Since the birth of her first child, she had experienced migraine attacks with headache and visual aura (stars in the visual field, blurred vision and flashes of light) once or twice a year. Her regular medication comprised ethinylestradiol/drospirenone 0.02 mg/3 mg tablets and candesartan 8 mg once daily for hypertension. She also smoked three to five cigarettes a day.

On admission, the patient's headache was improving. Clinical examination showed a blood pressure of 192/73 mmHg, a pulse of 62 beats/min, a respiratory rate of 18 breaths/min and an oxygen saturation (SpO_2) of 100 % on room air. Her blood pressure returned to normal spontaneously over the course of approximately 20 minutes. She was afebrile, and routine blood tests were unremarkable. ECG showed normal sinus rhythm. Neurological examination was unremarkable apart from

allodynia on light touch in the left V1–V3 branches of the trigeminal nerve and the left upper limb. Non-contrast CT of the head, performed due to suspected stroke, showed no abnormalities. The differential diagnosis included migraine with haemiplegic aura and transient ischaemic attack.

Migraine is one of the most common neurological disorders, with a prevalence of 10–15 % among adults (1), and is more common in women than in men. According to the International Headache Society, the risk of ischaemic stroke is three times higher in women with migraine, and six times higher in those who have migraine with aura (2, 3), compared to women without migraine. The risk of ischaemic stroke is estimated to be five to 17 times higher in women with migraine (with or without aura) who use oral contraceptives (2, 3). Smoking and hypertension are well-established risk factors for stroke.

As transient ischaemic attack was clinically suspected and the patient had an ABCD2 score of 4 (1 point for blood pressure > 140/90 mmHg, 2 points for unilateral weakness and 1 point for symptom duration), she received loading doses of clopidogrel 300 mg and acetylsalicylic acid 75 mg orally. The patient declined analgesia. She was admitted to the stroke unit. MRI of the head performed the following day showed two small acute infarcts in the right hemisphere, located in the occipital lobe and the posterior parietal lobe (Figure 1a–d).

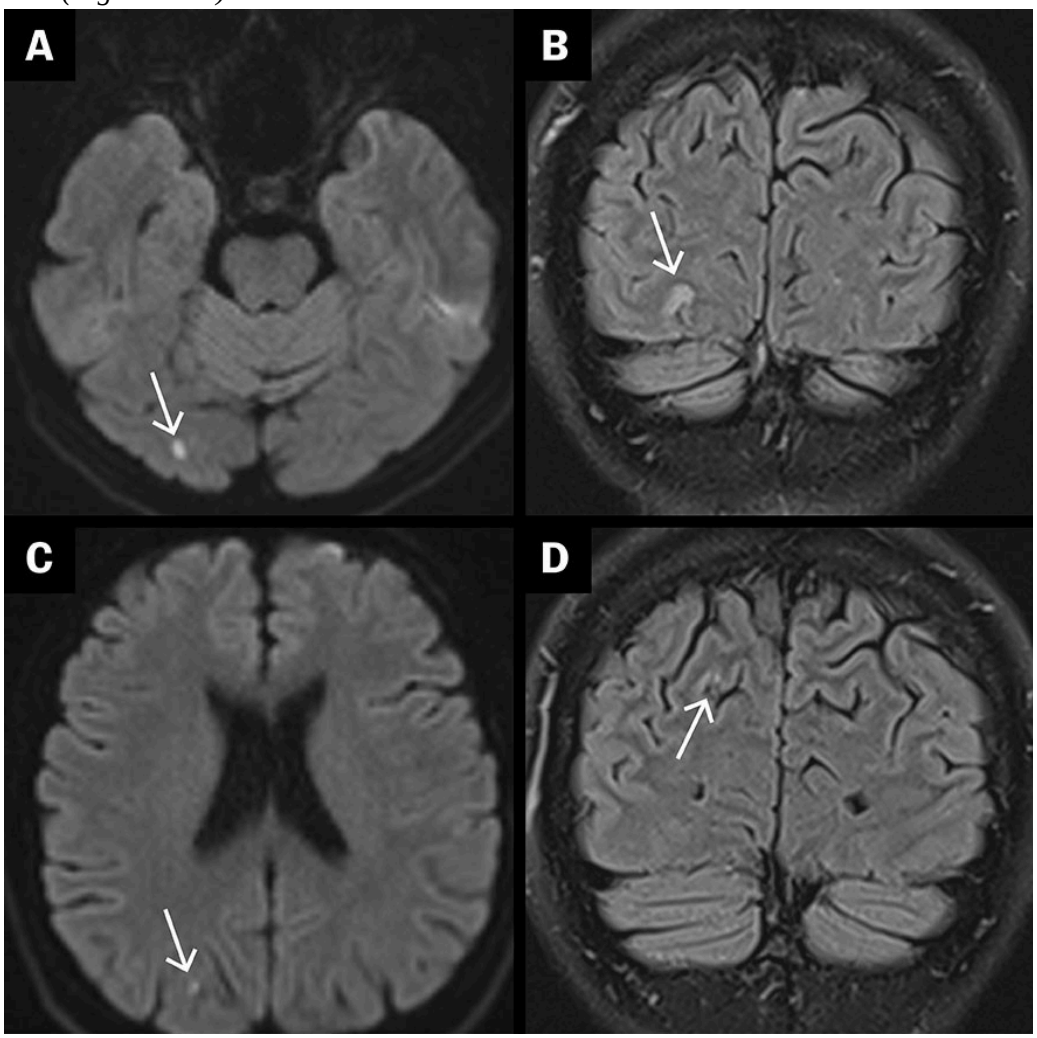

Figure 1 MRI of the brain on day 2. A) Diffusion-weighted imaging (b1000) showed a lesion with high signal in the right occipital lobe, with corresponding low values on the ADC map. These findings indicate diffusion restriction, consistent with cytotoxic oedema as seen in acute ischaemia. B) FLAIR MRI (coronal section) showed high signal corresponding to the findings

on the diffusion-weighted sequence in A. C) Diffusion-weighted imaging (b1000) showed a lesion with high signal in the right parietal lobe, corresponding to the findings in A. D) FLAIR MRI (coronal section) showed high parietal signal consistent with the findings on the diffusion-weighted sequence in C.

Migrainous cerebral infarctions are typically located in the posterior circulation (4). They can be multifocal, but usually affect only one vascular territory. Younger women are particularly at risk (4).

On the same day, investigations were initiated to identify a possible embolic source: CT angiography of the head and neck showed normal vessels. Telemetry monitoring revealed no abnormalities. Transthoracic echocardiography (day 4) and transoesophageal echocardiography (day 5) were also unremarkable. Blood tests (protein C, protein S, antithrombin III, factor Xa, lupus anticoagulant) were negative for inherited thrombophilia and for rheumatological or immunological disorders associated with thrombosis. LDL cholesterol was 2.4 mmol/L (reference range 1.4–4.7). HbA1c was 32 mmol/mol (27–41) and serum glucose was normal.
Due to a suspected visual field defect, the patient was assessed by an ophthalmologist, who found no abnormalities. Cognitive function, evaluated using the Montreal Cognitive Assessment screening tool and assessments performed by physiotherapists and occupational therapists in the stroke unit, were also unremarkable.
Migrainous infarction is a rare complication of migraine with aura and accounts for approximately 0.5–1.5 % of all ischaemic strokes (5). The patient experiences a typical migraine attack with one or more familiar aura symptoms, and imaging confirms an ischaemic infarct in a relevant vascular territory. Furthermore, at least one aura symptom must persist for 60 minutes or longer, and the presentation must not be better accounted for by another diagnosis within the International Classification of Headache Disorders, 3rd edition (ICHD-3) (4). Other symptoms related to the infarct may also occur.

The location of the ischaemic lesions on MRI corresponded to the symptoms the patient had experienced at home, including her familiar aura symptom of blurred vision in the left eye, which may have represented homonymous hemianopia. In addition, the headache on the day of admission was more intense than usual, and she experienced new sensory and motor aura symptoms (dizziness, numbness, allodynia and paresis). The patient therefore met the following criteria for migrainous infarction: a history of migraine with aura, relevant MRI findings, and no better alternative explanation (4). However, she did not meet the criteria of symptom duration ≥ 60 minutes or a typical attack.

As secondary prevention, the patient was started on dual antiplatelet therapy with acetylsalicylic acid 75 mg once daily and clopidogrel 75 mg once daily for 21 days, followed by clopidogrel 75 mg once daily long term (6,7). She started with atorvastatin 10 mg once daily, with a treatment target for stroke prevention of LDL cholesterol < 1.8 mmol/L. While in hospital, her blood pressure ranged from 101/57 to 110/71 mmHg, and her daily dose of candesartan was reduced from 8 mg to 4 mg.
The pathophysiological mechanisms underlying migraine with aura and migrainous infarction are not fully understood; however, cortical spreading depression is known to be the pathophysiological substrate of migraine with aura and the trigger for headache (8). During a migraine attack, cerebral perfusion is reduced, typically propagating from the occipital lobe to the parietal and temporal lobes at a rate of approximately 2–

3 mm/min. More pronounced reductions in cerebral perfusion (approximately 20–40 %) can lead to ischaemia. Various factors, including ischaemia, can trigger cortical spreading depression (9). Furthermore, repeated episodes of cortical spreading depression during migraine attacks can reduce neuronal resilience and lead to cerebral ischaemia (10).

Candesartan has a prophylactic effect in migraine (11,12) and may have contributed to the patient's initially infrequent migraine attacks. The patient was discharged in good condition after five days. A 7-day ambulatory cardiac event monitor (performed after discharge) was normal, with no evidence of arrhythmia. At a three-month follow-up in the stroke outpatient clinic, she had no sequelae and a modified Rankin Scale (mRS) score of 0. She had not experienced further migraine attacks and had returned to full-time work. She had reduced smoking to one cigarette per week and was no longer using oral contraceptives.

Discussion

A detailed clinical history is essential for distinguishing between primary and secondary headache disorders. In patients with a history of migraine, it is important to identify changes that may indicate more serious disease, including new aura symptoms and new-onset neurological deficits (13). Within a short timeframe, our patient developed transient new neurological symptoms, including dizziness, sequential sensory and motor deficits with a spreading pattern, and an unusually severe headache. Such a clinical presentation can be consistent with migraine with aura or haemiplegic migraine, both of which were considered as differential diagnoses. The episode could therefore have been misinterpreted as purely a migraine. Haemiplegic migraine is rare, with a prevalence of approximately 0.01 % (14). The patient's new symptoms, together with a headache that was atypical for her, prompted the need for emergency evaluation.

Were the patient's infarcts migraine-induced, or was the headache ischemia-induced? Patients who experience migraine with aura have an increased risk of cryptogenic stroke, while attack-associated reductions in cerebral perfusion can lead to migrainous infarction (15,16). Conversely, reduced cerebral perfusion related to occlusion or stenosis of the carotid arteries may cause migraine aura (15,17). This patient had no evidence of atherosclerotic disease or hyperlipidaemia. Following extensive investigation, no peripheral or central embolic source was identified. The patient's symptoms were consistent with convincing MRI findings, and no better aetiological explanation was identified. It was therefore concluded that she had experienced a migrainous infarction, despite not fulfilling the criteria for a typical attack or aura duration.

Key historical features when assessing patients with known migraine with aura include the type of aura, its duration, the severity of the headache, whether symptoms occurred simultaneously or sequentially, whether the aura symptoms are typical or new, and whether there is any loss of function suggestive of a possible TIA or stroke. Headache and gradual development of neurological symptoms with positive phenomena (sensory disturbances, visual phenomena) are typical of migraine with aura; however, if focal

symptoms persist for more than 60 minutes, the patient should be referred urgently for neurological assessment and MRI with diffusion-weighted imaging to enable early detection of possible cerebral infarction (18).

The patient has consented to publication of the article.

The article has been peer-reviewed.

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Publisert: 2. July 2026. Tidsskr Nor Legeforen. DOI: 10.4045/tidsskr.26.0097
Received 3.2.2026, first revision submitted 11.4.2026, accepted 30.4.2026.