

CASE REPORT

A deceitful stroke, a hidden tumour

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SUMMARY

A 41-year-old woman with malignant left middle cerebral artery infarction underwent an immediate decompressive craniectomy, obtaining good recovery, both clinically and radiologically. However, approximately 6 months after a successful discharge, the patient returned with gradual memory deterioration, dysarthria and progressively worsening headaches. Subsequent CT and MRI of the brain revealed a solid malignancy located in the same region as the extensive infarction. Following this finding, she underwent surgical debulking of the tumour and follow-up radiotherapy, with good progression of symptoms and overall good outcome.

BACKGROUND

This report demonstrates the need for timely and appropriate imaging in young patients with extensive ischaemic strokes. It reinforces the fact that decompressive craniectomy is an important consideration in selected patients with malignant middle cerebral artery (MCA) infarction. Finally, the report strongly suggests that, in young patients with extensive middle cerebral infarctions, underlying malignancies must be considered and appropriate investigations requested in a timely fashion in order to avoid misdiagnosis and delays in treatment.

CASE PRESENTATION

A 41-year-old woman with no medical history presented to the A&E department after a sudden

onset of moderate headache associated with right-sided weakness. On her way to the A&E department, she became increasingly unwell, with deterioration in state as well as level of consciousness, worsening of her right-sided weakness and slurred speech. On arrival, the patient was noted to have global aphasia with right hemiplegia and psychomotor agitation. There was no loss of consciousness and there were no seizures.

An immediate CT of the head was performed, demonstrating a thrombosed left MCA associated with a large hypodense area of infarction involving more than 1/3 of the corresponding vascular territory (figure 1), together with subfalcine and uncal herniation (figure 2). Five hours later, a contrast CT was performed, showing well-demarcated ischaemic involvement of both left frontal and temporal lobes (figure 3) associated with an area of increased density within the surrounding oedema, centrally in the left temporal lobe, most likely internal haemorrhage (figure 4). Also, a CT angiogram of carotid arteries was performed simultaneously, without any evidence of significant stenosis or dissection.

Following acquisition of these CT images, the patient was transferred to the neurosurgical unit, for ALA-guided decompressive craniectomy. This was performed successfully, with no immediate side effect and good recovery. A repeat CT of the head with contrast was performed, showing an acute haemorrhagic infarction of the anterior branches of the left MCA.

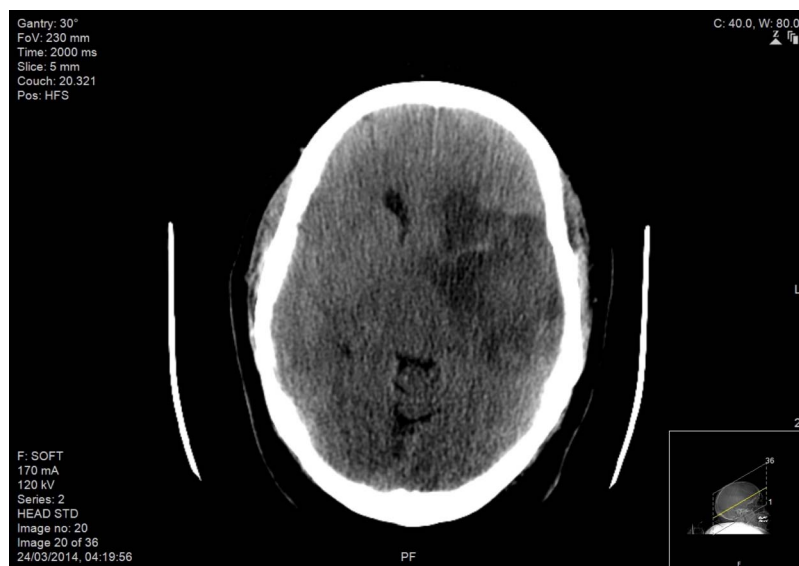
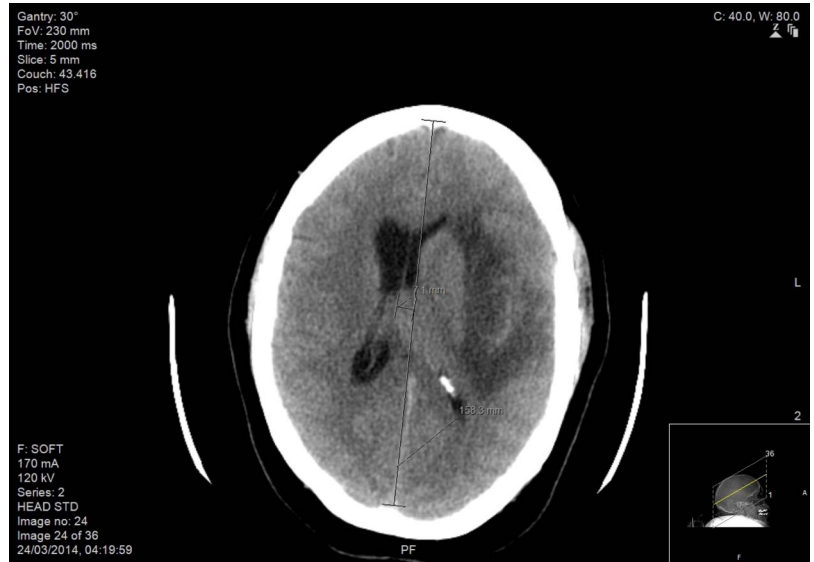


Figure 1 Linear hyperdense configuration corresponding to a thrombosed left middle cerebral artery, associated with extensive infarction involving more than 1/3 of the corresponding vascular territory and loss of corticomedullary differentiation. No signs of haemorrhagic transformation are noted.



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Figure 2 A 7 mm subfalcine and left uncus herniation is noted.



Four days post-decompressive craniectomy, the patient showed marked improvement. Physical examination detailed a resolving aphasia (being capable of understanding and following simple indications yet remaining impaired for verbal expression), resolving right hemiparesis and no hemi-neglect. Slight hemi-sensory defects were still noted, such as unstable gait and moderate cognitive impairment. However, the patient's overall state was good and she displayed rapid improvement. Three days later, she began to show some improvement in counting, naming simple objects, repeating pairs of words and arranging objects by colour and use. Likewise, her sensory functions consistently improved.

Ten days later, she suddenly developed a non-painful swelling over the left side of the upper facial area. However, this quickly resolved within the following days, with no residual defect. There was no neurological deterioration associated with this event.

One month after the initial event, the patient was discharged from our stroke unit and transferred to a specialised neurorehabilitation hospital for further management. At the time of discharge, she was able to transfer without assistance and was able to verbally express ideas adequately, forming short sentences.

A moderate degree of dysarthria and word-finding difficulties remained, however, they were not disabling.

As an outpatient, a bubble-ECHO was performed, without evidence of any persistent foramen ovale. Furthermore, a full transthoracic study did not show evidence, on two-dimension or in colour, of any atrial septal defect. The patient's 24 h cardiac recording did not show any evidence of atrial fibrillation and all investigation pertaining to possible congenital coagulation disorders was negative.

Unfortunately, 5 months after her discharge, the patient returned to the A&E department due to a subacute (approximately 3 weeks) onset of word-finding difficulties, deterioration of memory for recent events, dysarthria, recurrent bi-frontal and left temporal headaches, nausea and fatigue. She did not present any seizure-like activity or loss of consciousness. Her physical examination was remarkable for left seventh cranial nerve palsy, however, she had no limb weakness, and no sensory disturbance or cerebellar dysfunction. A CT of the head with contrast was performed, showing a faint patchy enhancement within a large hypodense area involving the left frontal-temporal region (figures 5 and 6). Owing to these findings, an immediate MRI of the head with contrast was carried out,

Figure 3 Large area of predominantly low-attenuation in the left frontal and temporal lobes, which is relatively sharply demarcated (as seen on the unenhanced CT).



Figure 4 Area of increased density within the surrounding oedema, centrally in the left temporal lobe, most likely representing internal haemorrhage.



revealing a 3.6×1.2 cm area of gliosis, which returned an increased signal on T2-weighted imaging (figure 7), fluid-attenuated inversion recovery (figure 8) and T1 (figure 9), and partial restricted diffusion (figure 10), indicating the presence of a haematoma. Also noted was the presence of two areas of nodular enhancement within this region, which enhanced more than the adjacent brain. This immediately raised the suspicion of an underlying malignancy.

After a prompt neurosurgical evaluation, the patient underwent reopening of the craniectomy and 5-ALA-guided left temporal lobectomy with complete resection of the enhancing tumour; an insertion of a left frontotemporal craniocurve cranioplasty was performed. On the postoperative MRI, there appeared to be a complete resection of the enhancing tumour without evidence of postoperative bleeding. The surgical sample was sent to pathology and was confirmed to be an oligoastrocytoma (WHO-2007 grade 2; MIB 5.6% and IDH1 positive). Based on these results, it was decided that the patient would receive postoperative radiotherapy and palliative-support treatment.

INVESTIGATIONS

- ▶ Bubble ECHO, 24 h tape
- ▶ MRI and CT of the head
- ▶ CT angiogram
- ▶ Antibodies, vasculitis screen, thrombophilia screen, tumour markers

DIFFERENTIAL DIAGNOSIS

- ▶ Intracerebral metastasis
- ▶ Primary intracerebral malignancy
- ▶ Haemorrhagic transformation of MCA infarction

TREATMENT

- ▶ Decompressive hemi-craniectomy
- ▶ The patient underwent surgical debulking followed up by radiotherapy and palliative supportive treatment
- ▶ Secondary prevention of stroke with clopidogrel
- ▶ Control of hypertension and cholesterol levels

Figure 5 Faint patchy enhancement noted within the large hypodense area involving the left frontotemporal region.



Figure 6 Faint patchy enhancement noted within the large hypodense area involving the left frontotemporal region.



Figure 7 Increased signal on T2.



Figure 8 Increased signal on fluid-attenuated inversion recovery.

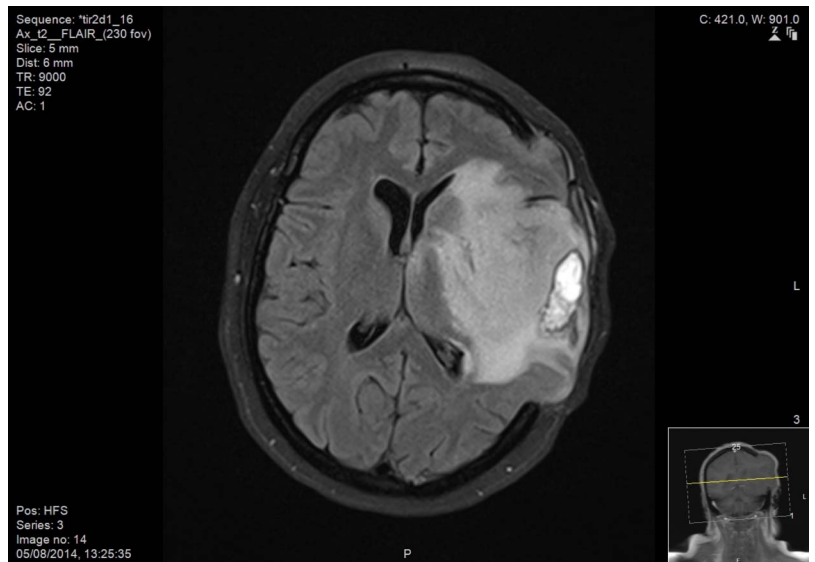


Figure 9 Increased signal on T1.



OUTCOME AND FOLLOW-UP

The patient was followed up in clinic, with signs of improvement. Slight memory impairment and word-finding difficulties were still present, however, there was no aphasia and no other cognitive impairments. Her strength was normal and no sensory loss was present.

DISCUSSION

The patient's young age, her acute clinical presentation and the initial clinical assessments were suggestive of an acute ischaemic event. The initial neuroimaging was also more suggestive of an ischaemic rather than a tumour-infiltrated region. However, on subsequent imaging, it became obvious that there was indeed a malignancy. The challenge of this case was to differentiate a tumour from an infarcted area; this challenge has been recognised for some time. A large space occupying the MCA or hemispheric ischaemic brain infarcts is usually associated with the development of massive brain oedema, which may lead to herniation, as in the case of our patient. This condition, which has been described as malignant MCA infarction, is associated with 80% mortality due to herniation during the first week, despite

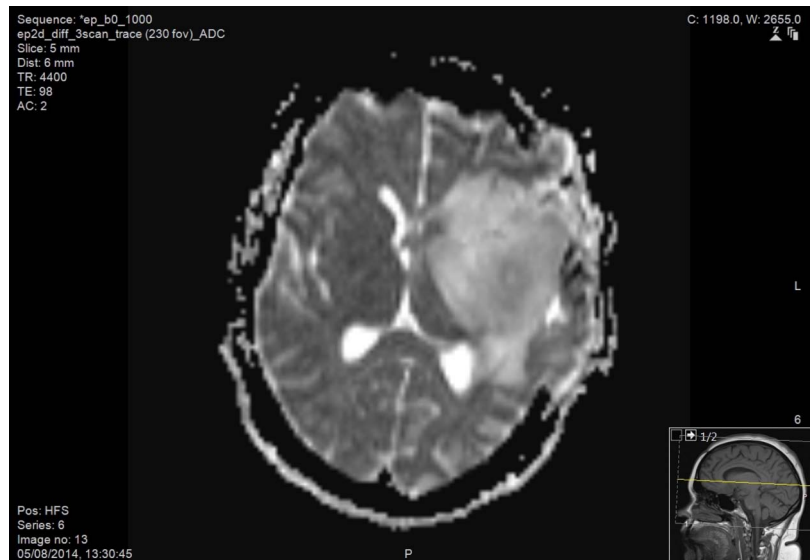
maximal conservative treatment. Similarly, intracerebral tumours can present with herniation and massive oedema.

Intracerebral tumours can occasionally have a stroke-like presentation. Cerebral infarction can mimic a brain tumour by its cystic appearance and contrast enhancement in various stages of its evolution. But ischaemia prior to the detection of the tumour is rare and there should be a high index of suspicion, for the proper management.¹

Several mechanisms have been shown to be capable of causing stroke in brain tumour patients—either in isolation or in combination. These include low-flow infarct due to vessel compression and occlusion; vessel dissection due to neoplastic infiltration of the vessel wall and, at times, haemorrhage.² Furthermore, several hypotheses have been proposed to explain the development of cerebral ischaemia prior to vascularisation of the tumour on imaging. There are some reports mentioning the tumour cells encompassing and infiltrating the MCA with rupture³ and dissection of the vasculature with stroke presentation.⁴ Malignant tumours such as gliomas can invade the arterial walls and rupture the vessel.

In this case, the diagnosis of the underlying malignancy was delayed primarily due to three factors: (1) the absence of

Figure 10 Apparent diffusion coefficient map—partial restricted diffusion.



Learning points

- ▶ Decompressive craniectomy for malignant middle cerebral artery (MCA) infarction has proven to be a beneficial procedure with positive outcome in appropriately selected patients. The appropriate selection of patients should be based on current evidence provided from the trials DESTINY I and II, and must necessarily require an onset of symptoms >12 and <36 h before the surgical procedure in a patient between 18–60 years of age, with a well-documented unilateral MCA infarction, including at least 2/3 of the territory and including at least part of the basal ganglia, with or without additional ipsilateral infarction of the anterior or posterior cerebral artery; also required is an National Institute of Health Stroke Scale (NIHSS) of >18 for lesions of the non-dominant hemisphere and >20 for lesions of the dominant hemisphere.
- ▶ Extensive strokes in young patients—especially with no previous risk factors—must include an MRI evaluation to rule out any underlying malignancy. CT scans are not a reliable method in these cases as, on many occasions, they fail to detect the initial stages of malignancy.
- ▶ Appropriate imaging in a timely fashion is paramount in such young patients with stroke, as the decision for decompressive craniectomy must be taken without delay.

suggestive signs on the initial head scans, (2) the absence of symptoms of intracranial hypertension, mainly because of the decompressive hemicraniectomy, and, finally, (3) due to the lack of clinical signs suggestive of a malignancy.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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