Case Report

Tumor thrombus: A rare cause of acute stroke

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ABSTRACT

Stroke is common in our everyday practice as doctors and radiologists. The stroke pathways have steadily improved over the last number of years with the advent of dedicated stroke units; synchronized streamlined care with endovascular stroke therapies, leading to improved outcomes. We present the case of a 57-year-old woman complaining of sudden onset right-sided facial droop and right-sided weakness. The case represents an unusual cause for an atypical stroke with multiple learning outcomes.

Key words: Anticoagulant therapy, Radiologist, Stroke, Tumor

Stroke treatment pathways have steadily improved over recent years with the advent of dedicated stroke units; synchronized streamlined care with endovascular therapies, leading to improved outcomes [1]. The cause of stroke is cryptogenic in 25–30% of cases following standard workup [2]. Most strokes are embolic. In non-lacunar stroke, initial consideration may be given to cardioembolic sources such as left ventricular thrombi, dense atherosclerosis, or even rarer causes such as atrial myxoma. Even less likely is migrated tumor thrombus due to direct tumoral vascular invasion. Decisions related to the management of acute stroke from an alternate source are often complex and require multidisciplinary input. Hence, important decisions must be made regarding providing anticoagulant therapy to the patient and referral to neurointerventional radiology for thrombectomy, if required.

This case aims to highlight the value of a multidisciplinary approach to stroke care and consideration of alternate causes of acute stroke to guide accurate investigations and management.

CASE REPORT

A 57-year-old woman complained of sudden onset right-sided facial droop and weakness of the right upper and lower limbs while having dinner. The patient was subsequently blue-lighted to the University Hospital Galway.

Urgent computed tomography (CT) scan was performed after the initial assessment by the on-call medical team. Assessment by the medical team yielded right-sided hemiparesis and moderate dysarthria. The motor loss affected the upper limbs more than the lower. The National Institutes of Health Stroke Scale score of 12 was calculated [3].

Non-contrast CT revealed diffuse right cerebral low attenuation with loss of gray-white matter differentiation throughout the distribution of the right middle cerebral artery (MCA) (Fig. 1a). Hyperdense right MCA sign was also noted (Fig. 1b). The Alberta Stroke Program Early CT Score was calculated as 4. CT angiogram was performed and revealed an occlusive thrombus in the mid-right M1 segment of the right MCA (Fig. 1c).

Initial urgent findings were discussed with the medical team on call and the neurointerventional radiology department; however, the patient was not suitable for thrombectomy given the established large territory of infarction. Thrombolysis was administered given that the patient was within the appropriate window for treatment. The patient was admitted to the stroke unit for further supportive management.

Request details from intensive care unit (ICU) included new clinical information: There had been an acute change in the patient's status including new seizure-like activity with mydriasis. A clinical question regarding infarct or hemorrhagic transformation was generated. A follow-up CT was emergently performed. CT confirms the presence of a dense region of low attenuation throughout the right MCA territory as expected based on prior known thrombus within the vessel. There was a new midline shift consistent with edema-related mass effect. Interestingly, there were subtle areas of low attenuation in the left occipital lobe and new patchy areas within the left frontal lobe suggesting early multifocal left-sided infarcts (Fig. 2). The patient was intubated, clinically in disseminated intravascular coagulation, and not suitable for any intervention at this stage.

After some time of ICU discussion, it was established that the patient had a history of prior lung cancer. Further, investigations

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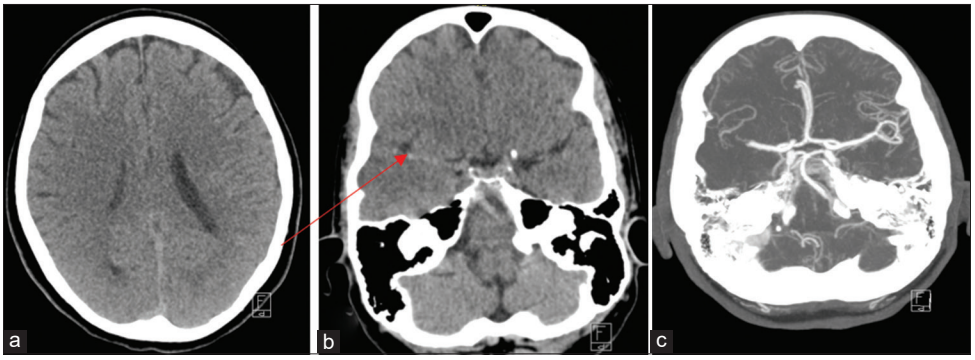

Figure 1: (a) Diffuse low attenuation with loss of gray-white matter differentiation in the right cerebral hemisphere; (b) hyperdense right middle cerebral artery sign; and (c) maximum intensity projection image at the level of the occluded segment of the right middle cerebral artery

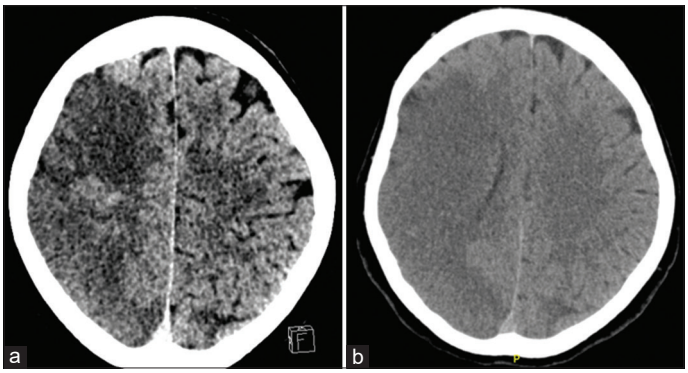

Figure 2: (a) Diffuse low attenuation in the right frontal lobe and (b) Stroke Windows H: 50 W: 50. New low attenuation foci in the left cerebral hemisphere

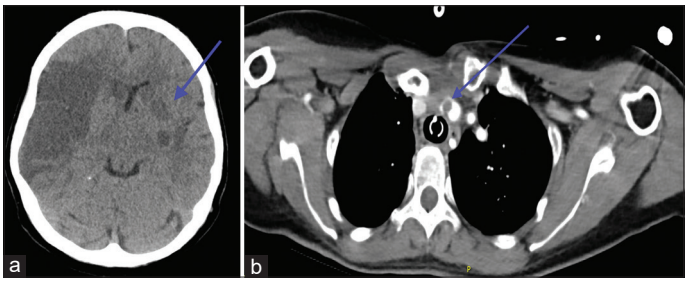

Figure 3: (a) Non-contrast computed tomography brain revealing established dense right-sided infarct with multiple new areas of low attenuation in the left head of caudate, basal ganglia, and insular cortex and (b) filling defect within the arch of the aorta and soft-tissue mass anteriorly invading the vessel with extensive tumor thrombus

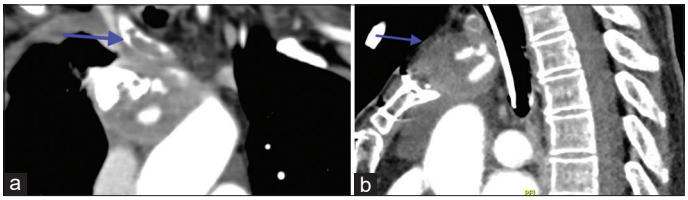

Figure 4: (a) Coronal zoomed image demonstrating extensive tumor thrombus extending from anterior mediastinal mass in the arch of aorta and brachiocephalic arteryand (b) sagittal zoomed image at the level of the upper mediastinum revealing the invasive soft-tissue mass abutting the intubated trachea posteriorly

requested including a repeat CT brain and CT thorax, abdomen, and pelvis (CT TAP) were performed to establish the underlying cause of the multifocal infarcts.

The previous CT brains had revealed the atypical clinical presentation of multifocal infarcts raising the probability of an embolic source. The bedside echocardiogram in ICU was clear. A contrast-enhanced CT TAP was performed immediately after the CT brain (Fig. 3a). CT TAP revealed a soft-tissue mass in the anterior mediastinum directly invading the arch of the aorta with tumor thrombus present within the arch of aorta and brachiocephalic artery (Fig. 3b). CT TAP revealed a soft-tissue mass in the anterior mediastinum invading the arch of aorta posteriorly with multiple filling defects within the arch of aorta and brachiocephalic artery (Fig. 4).

The aggressive mass was seen to invade the sternum with pathological subluxation of the right sternoclavicular joint. Given the prior history of lung adenocarcinoma for which the patient had undergone a prior lobectomy, the recurrent disease was thought to be the most likely cause for the new invasive anterior mediastinal mass. Other less likely differential considered was a metastatic deposit.

DISCUSSION

Direct tumor thromboembolism from recurrent lung carcinoma is rare. The original paper describing this phenomenon was a direct pulmonary arterial invasion causing stroke [4]. A number of case reports have described the occurrence of stroke either from lung tumor invasion into the pulmonary veins [5] or direct invasion of the atria [6]. Another case report documented extensive tumor thrombus within the abdominal aorta causing lower limb ischemia and further intracranial infarct [7].

The optimum treatment for embolic tumor-causing stroke is mechanical thrombectomy as proven in other studies [8]. Unfortunately, this patient did not progress to mechanical thrombectomy given the extent of the established infarct and progressive deterioration of the clinical condition. A recent Japanese study by Siegler *et al*. found that mechanical thrombectomy is potentially beneficial in returning the patient to baseline up to 24 h after the initial infarct with large vessel occlusion (LVO) [9].

CT perfusion will continue to improve the interpretation of the ischemic penumbra and hopefully inform therapeutic benefits from salvage thrombectomy [10]. To the best of our knowledge, no exact case report involving a recurrent lung tumor invading the arch of aorta with subsequent tumor thrombus leading to multifocal acute infarct has been described in the literature. There have been several studies discussing the phenomenon of acute stroke as the first presentation of atrial myxoma. In one study, it was suggested that systemic thrombolysis is suitable for patients with stroke from atrial myxoma [11].

Based on available case studies, mechanical thrombectomy forms the mainstay of management in acute infarct secondary to tumor thrombus [8,12-14]. Unfortunately, in this case, the extent of initial infarction ruled out the possibility of thrombectomy from the treatment options available. The subsequent left-sided infarcts may not have been preventable given the extent of direct tumor invasion. This case demonstrates a good example of a rare cause of multifocal stroke secondary to tumor thromboembolism. This should always be considered in atypical presentations and in patients with relevant clinical history.

CONCLUSION

Stroke is a common presentation to hospitals around the world and the etiology is frequently unknown. Our case highlights the need to always consider an atypical source of emboli, particularly in the setting of multifocal infarcts. Mechanical thrombectomy forms the mainstay of management in this context; however, consideration must be given to the risk of further infarcts based on the embolic source. Early discussion with interventional services is advised; however, patients can benefit up to 24 h after the initial insult.

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