Mechanical thrombectomy in patients with tumour-related ischaemic stroke

Tobias Zander¹, Javier Maynar², Fernando López-Zárraga², Rogelio Herrera¹, Juan José Timiraos-Fernández³, Andrea Saraceni¹ and Manuel Maynar^{1,4}



Interventional Neuroradiology 2016, Vol. 22(6) 705-708 © The Author(s) 2016 Reprints and permissions: sagepub.co.uk/journalsPermissions.nav DOI: 10.1177/1591019916669853 ine.sagepub.com



Abstract

Ischaemic stroke is a common cause of death and incapacity and is related in most cases to vascular disease. Intracranial vessel occlusion due to tumour emboli is a rare entity and adequate treatment for this condition is not defined. The use of mechanical thrombectomy devices is considered the treatment of choice for major intracranial vessel occlusion; however, no recommendation can be made in the case of tumour thrombembolia. This report describes two cases who presented with a middle cerebral artery occlusion due to tumour emboli and that were treated using the Solitaire thrombectomy device.

Keywords

Stroke, thrombectomy, tumour

Received 7 June 2016; accepted 23 August 2016

Introduction

Cerebral ischaemia is one of the most common causes of death and permanent incapacity in the world. Meanwhile, arteriosclerotic disease of the carotid arteries and different cardiac pathologies are responsible for most ischaemic strokes; cerebral ischaemia caused by tumour embolism is a rare phenomenon.

Tumour emboli can be caused by benign and malign tumours; however, the vast majority of cerebral tumour emboli are due to benign tumours. Approximately 50% of thromboembolism is caused by cardiac myxomas that are localised in the left cavities of the heart.¹ Ischaemic stroke due to malign tumour emboli is less likely to occur and is reported in centrally located primary lung tumours or metastases that are infiltrating the left cardiac cavities. Patients with tumour invasion of the pulmonary veins may experience symptoms of systemic arterial embolisation such as transient ischaemic attacks and embolic stroke including acute occlusion of the aorta or its major systemic branches.²

We present two cases of ischaemic cerebral stroke that were caused by tumour occlusion of the intracerebral arteries. Both cases were treated with mechanical thrombectomy using the Solitaire AB device (EV3, Irvine, CA, USA).

Case 1

A 58-year-old man presented at the emergency room with sudden onset of right hemiplegia of 1 hour of

evolution. His medical history included smoking and sporadic haemoptysis during the last few months, with a lack of compliance for consulting a physician. Neurological examination revealed a right hemiplegia, a left gaze deviation and dysarthria with a National Institute of Health stroke scale (NIHSS) of 24. The computed tomography (CT) examination confirmed the absence of intracerebral bleeding and a positive hyperdense left middle cerebral artery sign (Figure 1). The contrast enhanced angio-CT showed a complete occlusion of the M2 segment of the left middle cerebral artery (MCA).

A cerebral angiography demonstrated an MCA occlusion (Figure 2(a)) and under general anaesthesia, a mechanical thrombectomy was performed using the $3 \times 20 \text{ mm}$ Solitaire AB device (EV3) with extraction of the embolus after two passes (Figure 2(b)). A final control confirmed an angiographic TICI 3 result (Figure 2(c)). The extracted material was examined by a pathologist with the diagnosis of an undifferentiated

Corresponding author:

 ¹Endoluminal/Vascular Department, Hospiten Clinic Group, Spain
²Interventional Radiology Department, ARABA University Hospital, Spain
³Stroke Unit, ARABA University Hospital, Spain
⁴University of Las Palmas de Gran Canarias, Spain

Tobias Zander, Endoluminal/Vascular Department, Hospiten Clinic Group, Santa Cruz de Tenerife, Rambla de Santa Cruz 115, Santa Cruz de Tenerife, Spain.

Email: tobiaszander@gmx.de

Interventional Neuroradiology 22(6)

non-small cell carcinoma of the lung. The next day, the patient presented with a NIHSS of 10 and the control CT demonstrated a hypodensity of the basal nuclei and the ipsilateral temporal lobe without haemorrhagic complications. A transoesophageal ultrasound was normal without intracardiac lesions or valve vegetation. A fibrobronchoscopy and bronchoalveolar lavage was accomplished, which confirmed the diagnosis of a



Figure 1. The computed tomography image does not show any intracerebral bleeding. A hyperdense left middle cerebral artery sign can be observed (white arrows).

pulmonary adenocarcinoma. As the patient presented with a persistent haemoptysis, an embolisation was performed on the third postoperative day using 300–500 microns polyvinyl alcohol particles. A positron emission tomography CT confirmed a hypermetabolic infiltrating mass in the right superior pulmonary lobe with metastasis in the right paratracheal and left paravertebral lymph nodes, as well as abdominal and retroperitoneal metastasis. Therefore, the patient was transferred to the palliative care unit and died on the 14th postoperative day.

Case 2

A 46-year-old woman was transferred to our hospital after presenting in a different institution with sudden onset of weakness of the left side of the body. At arrival she had an onset of symptoms of 4 hours and 15 minutes. Neurological examination revealed a somnolent patient who opened her eyes in response to painful stimuli. A left hemiplegia was found with a deviation of the labial commissure and a right gaze deviation with a NIHSS of 16. The CT, which was provided by the other institution, did not reveal intracranial haemorrhage or hypodense lesions.

The diagnostic angiography confirmed a right MCA occlusion (Figure 3(a and b)) and therefore a mechanical thrombectomy was considered using a 4×20 mm Solitaire AB device (EV3) with complete extraction of the embolus after one pass and obtaining an angiographic TICI 3 result (Figure 3(c)). The extracted material was analysed and the diagnosis of a myxoma was confirmed. A magnetic resonance tomography showed a recent right internal capsule stroke; however, the NIHSS improved from 6 directly after the procedure to 2, 6 days after the procedure.



Figure 2. (a) Diagnostic angiography of the left carotid artery demonstrates an occlusion of the M2 segment of the left middle cerebral artery (arrow). (b) After mechanical thrombectomy a small tissue fragment can be extracted. (c) A TICI 3 result was obtained, which was confirmed in the control angiography.



Figure 3. (a) The image shows an incomplete occlusion of the right distal M1 segment of the middle cerebral artery (arrow). (b) Close-up image after microcatheter passage confirms M1 segment occlusion. (c) Control angiography after mechanical thrombectomy demonstrates a TICI 3 angiographic result.

A transesophageal ultrasound confirmed an 11.5×3.3 cm mass in the left atrium that protruded into the left ventricle. Six days after the initial stroke, the patient underwent cardiac surgery with complete resection of the myxoma. Further intrahospital evolution was uneventful and the patient was discharged at day 15 after the stroke with a NIHSS of 0 and a modified Rankin scale of 0. At the 2-year follow-up the patient was completely asymptomatic with incorporation into working life.

Discussion

Stroke caused by tumour embolic occlusion of intracranial arteries is a rare entity and is mostly related to benign tumour lesions of the left cavities of the heart. The most common benign tumour is the cardiac myxoma, responsible for 0.5% of all ischaemic strokes.³ Malign tumour embolism is rare in the setting of ischaemic stroke and should be considered in patients with centrally located pulmonary tumours, embolic distributions infarctions and intracardiac masses.⁴ As clinical distinction and differentiation by imaging modalities may be difficult, in the setting of an acute stroke these findings should not exclude an invasive treatment if a definitive diagnosis has not been established.

The stroke described in the first case of this report was due to a malign tumour embolism. The patient died 14 days after the mechanical thrombectomy; therefore, one might argue that this patient would have had a contraindication for endovascular thrombectomy. The patient had presented with episodes of haemoptysis for the previous months but did not seek medical advice. Therefore, the diagnosis of a malign tumour disease was not established at the time of his admission, which was the reason why mechanical thrombectomy was considered.

Treatment in tumour stroke has been described before; however, most of these reports used intravenous or intra-arterial thrombolytic therapy in order to achieve patency in occluded intracerebral arteries. Thrombolytic treatment has the potential to achieve patency if a fresh thrombus is present. Tumour emboli are unlikely to lyse upon thrombolytic therapy as the occluding material is composed of the tumour itself, adherent thrombotic material or a combination of both.⁵ Some tumours, such as non-small cell lung carcinoma, may produce hypercoagulability due to prothrombotic mutations that could lead to thrombotic emboli.⁶ However, in the present case report, in both patients, the pathological examination of the extracted material confirmed tumour tissue to be responsible for the intracranial vascular occlusion.

In the two cases presented, intra-arterial thrombectomy was performed without the use of fibrinolytic agents. Intravenous fibrinolysis using recombinant tissue-type plasminogen activator (r-tPA) carries the potential risk of intracranial haemorrhage. Large randomised controlled trials could demonstrate that haemorrhage rates were no higher in intra-arterial thrombectomy compared with control patients, most of whom received r-tPA, suggesting that most of the bleeding in both arms in these studies was because of the r-tPA as mentioned by Grotta and Hacke.⁷ They furthermore propose that r-tPA might be omitted in patients who can be quickly moved to the endovascular suite and who have a higher bleeding risk from r-tPA.

In addition, intravenous fibrinolysis in the setting of ischaemic tumour stroke carries the risk of intracranial haemorrhage.⁸ In particular, patients suffering from myxoma-induced ischaemic stroke have a higher haemorrhagic risk under intravenous thrombolytic therapy, because these patients may present with cerebral microaneurysms.⁹ Mechanical thrombectomy using a stent

retriever has demonstrated its beneficial effect on functional outcome.^{10,11} Mechanical thrombectomy in this setting has the advantage that the mechanism of clot extraction is not only effective in a fresh blood clot but also for the extraction of solid tissue and without increasing the risk of bleeding.⁵ For both cases in this report, extraction of the tumour was possible with a good technical result (TICI 3) and without bleeding complications.

In conclusion, occlusion of intracranial vessels due to tumour embolism is a rare entity. Our single observation suggests that mechanical thrombectomy using a Solitaire device can be a good alternative to achieve patency of intracranial vessel occlusion due to tumour emboli. Mechanical thrombectomy may decrease the procedural risk compared to thrombolytic treatment and could be more effective for tumour emboli extraction.

Ethical approval

The authors declare that they have obtained approval from the institutional review board.

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Declaration of conflicting interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

References

- Reynen K. Cardiac myxomas. N Engl J Med 1995; 333(24): 1610–1617.
- Gandhi AK, Pearson AC and Orsinelli DA. Tumor invasion of the pulmonary veins: a unique source of systemic embolism detected by transesophageal echocardiography. *J Am Soc Echocardiogr* 1995; 8(1): 97–99.
- Knepper LE, Biller J, Adams Jr, HP, et al. Neurologic manifestations of atrial myxoma. A 12-year experience and review. *Stroke* 1988; 19(11): 1435–1440.
- Navi BB, Kawaguchi K, Hriljac I, et al. Multifocal stroke from tumor emboli. *Arch Neurol* 2009; 66(9): 1174–1175.
- van den Wijngaard I, Wermer M, van Walderveen M, et al. Intra-arterial treatment in a child with embolic stroke due to atrial myxoma. *Interv Neuroradiol* 2014; 20(3): 345–351.
- Aoun EG, Musallam KM, Abou-Ghazal M, et al. Malignancy and hypercoagulability: a two-way association revisited. *J Thromb Thrombolysis* 2010; 30(3): 340–341.
- Grotta JC and Hacke W. Stroke neurologist's perspective on the new endovascular trials. *Stroke* 2015; 46(6): 1447–1452.
- 8. Chong JY, Vraniak P, Etienne M, et al. Intravenous thrombolytic treatment of acute ischemic stroke associated with left atrial myxoma: a case report. *J Stroke Cerebrovasc Dis* 2005; 14(1): 39–41.
- Nagy CD, Levy M, Mulhearn TJt, et al. Safe and effective intravenous thrombolysis for acute ischemic stroke caused by left atrial myxoma. J Stroke Cerebrovasc Dis 2009; 18(5): 398–402.
- Berkhemer OA, Fransen PS, Beumer D, et al. MR CLEAN Investigators. A randomized trial of intraarterial treatment for acute ischemic stroke. *N Engl J Med* 2015; 372(1): 11–20.
- Zander T, Vicente S, Garcia de Casasola C, et al. Trombectomía mecánica primaria en oclusión aguda de arterias intracerebrales. *Emergencias* 2016; 28(1): 41–44.