

## “Code stroke” activation in a patient with an external ventricular assist device<sup>☆,☆☆</sup>



### Código ictus en paciente portador de dispositivo de asistencia ventricular externa

Dear Editor,

External ventricular assist devices (EVAD) have been available since 2002 as an alternative treatment or bridging therapy to heart transplant for patients with severe systolic dysfunction in situations of advanced refractory (stage D) heart failure (HF). The devices constitute a long-term, percutaneous, mechanical circulatory support comprising an electrically-powered pump driving blood from the left ventricle to the ascending aorta through a system of cannulas.<sup>1,2</sup> First-generation devices produced a pulsatile flow, similar to physiological haemodynamics. However, a subsequent study revealed greater survival and a lower rate of complications with continuous-flow devices<sup>3</sup>; therefore, the latter are most frequently used today.

Stroke is one of the most frequent complications associated with these devices, with an estimated annual incidence rate of 0.19.<sup>4,5</sup> The implication of this is that in the coming years, these devices will be an infrequent but growing reason for code stroke (CS) activation. However, no studies have addressed the treatment of hyperacute stroke in these patients. We report our experience with the management of CS in a patient with an EVAD.

Our patient was a 54-year-old man with a personal history of arterial hypertension, dyslipidaemia, and atrial fibrillation treated with cardioversion. An EVAD (initially an EXCOR<sup>®</sup> Berlin Heart device, replaced by continuous-flow Levitronix<sup>®</sup> CentriMag<sup>®</sup> VAD due to thrombosis in the first device) had been inserted as bridging therapy to the heart transplant (HT) due to ischaemic HF. He had been admitted to another hospital for 27 days due to a spontaneous right parietal occipital haematoma presenting with headache, vomiting, and left homonymous hemianopsia; the patient progressed favourably, becoming asymptomatic (modified Rankin Scale: 1-2). He was receiving anticoagulant medication (perfusion with bemparin; partial thromboplastin time of 54 s) and antiplatelet treatment with acetylsalicylic acid and clopidogrel.

The patient suffered a left-hemisphere stroke, presenting altered level of consciousness, global aphasia, left-sided deviation of the head and eyes, right-sided homonymous hemianopsia, facial paresis, hemiplegia, and

brachiorucral hemianaesthesia (NIHSS score: 21). The baseline brain computed tomography scan revealed no sign of acute ischaemia (ASPECTS score: 10); the angiography study showed occlusion of the M1 segment of the left middle cerebral artery (MCA) with limited leptomeningeal collateral circulation (LCC). Since systemic fibrinolysis was contraindicated due to a recent intracranial haemorrhage and active anticoagulation, the patient was transferred to our hospital to receive neurointerventional treatment. Complete reperfusion of the vascular tree (TICI grade 3) was achieved 3 hours and 45 minutes after symptom onset. The procedure was completed with no complications.

Despite the arteriographic success, infarction of the left MCA with areas of haemorrhagic transformation was confirmed at 24 hours. The patient progressed poorly. His level of consciousness did not improve, so he remained intubated; he presented new bihemispheric ischaemic lesions, fever, and haemodynamic instability. As the lesions were irreversible, CT was ultimately ruled out and treatment was limited. The patient died 11 days after the stroke. No echocardiography or other diagnostic tests were performed to rule out intracavitary thrombi or device thrombosis.

Our case is interesting as it underscores the lack of scientific evidence regarding the management of acute stroke in patients with EVADs. Based on isolated clinical cases<sup>6-9</sup> and expert opinion, some authors recommend proceeding in the same way as with patients without this type of devices<sup>5</sup>; all these patients, with some exceptions, receive anticoagulant treatment and are therefore not eligible for intravenous fibrinolysis. Our patient's clinical outcome was not as favourable as expected, considering how quickly revascularisation was achieved.

After analysing the pathophysiology of the case, we believe that haemodynamics probably played a fundamental role in treatment failure. Some authors<sup>10</sup> have highlighted that if arterial flow is interrupted, the fluid shear stress caused by the increased mechanical pressure of the bloodstream on the endothelium of collateral vessels would activate the preexisting LCC, in a process known as arteriogenesis. Moderately elevated systolic blood pressure (170-190 mm Hg) at the time of stroke has been clinically demonstrated to maintain a more efficient LCC, which was correlated with a better functional prognosis at 3 months,<sup>11</sup> and translated into a recommendation to avoid an excessive decrease of arterial pressure during acute stroke.<sup>12</sup> Our hypothesis is that both the patient's heart failure and the fact that the EVAD maintained a continuous flow at 60-70 mm Hg would have limited his hypertensive response, and consequently the capacity to maintain permeability of the LCC at the time of ischaemia.

This case highlights the need to expand our knowledge on the management of stroke in patients with EVADs. Confirming our hypothesis would require collateral circulation models of both haemodynamic situations (continuous and pulsatile flow), as well as prospective studies. If our hypothesis is correct, we may expect poorer neurological prognosis of stroke in patients with continuous-flow EVADs than in those with pulsatile-flow devices; the therapeutic window for brain reperfusion may also be shorter due to early failure of the compensatory mechanisms.

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<sup>☆☆</sup> This study was presented in poster format at the 13th Annual Meeting of the Neurological Association of Madrid.

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## Muscle atrophy and fasciculations as a manifestation of sporadic Creutzfeldt-Jakob disease: a case report<sup>☆</sup>



### Amiotrofia y fasciculaciones como forma de presentación de la enfermedad de Creutzfeldt-Jakob esporádica. A propósito de un caso

Dear Editor,

Sporadic Creutzfeldt-Jakob disease (CJD) is the most frequent form of presentation of prion diseases. Although the clinical characteristics of this condition are well known,<sup>1,2</sup> lower motor neuron involvement is a rare form of presentation, especially in the early stages of the disease.<sup>3</sup>

Our patient was a healthy 54-year-old man with a 4-month history of poor coordination and gait instability. The neurological examination detected fasciculations in the deltoid, biceps, triceps, and quadriceps muscles, associated with amyotrophy and mild weakness of the proximal muscles of his limbs, especially the quadriceps, deltoid, and periscapular muscles. The patient also displayed global areflexia with mild dysmetria in all limbs, predominantly affecting the left side; orthostatic tremor; and ataxic gait with inability to walk in tandem. He showed no signs of upper motor neuron involvement.

A brain MRI scan revealed cortical alterations on diffusion-weighted sequences only (Fig. 1). Laboratory tests included a complete blood count; a biochemical study; kidney, liver, and thyroid function tests; and vitamin B<sub>12</sub> and B<sub>9</sub> determination. Results were normal. The tests for infection, paraneoplastic antibodies, tumour markers, and markers of autoimmunity yielded negative results. An electroencephalography revealed mild, diffuse disorganization of background activity; electromyography showed extensive denervation in the form of fibrillations, positive waves, and fasciculations in all muscles explored. The nerve conduction study revealed no abnormalities. A neuropsychological study revealed mild visuospatial alterations, short-term memory deficits, and reduced verbal fluency; these findings are compatible with cortico-subcortical cognitive impairment. CSF analysis yielded positive results for 14-3-3 protein and

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