Takotsubo cardiomyopathy associated with cerebral infarction following surgery for euthyroid goitre☆

Síndrome de Takotsubo asociado a infarto cerebral en postoperatorio de bocio eutiroideo

Dear Editor:

Takotsubo cardiomyopathy or stress cardiomyopathy is a disease characterised by acute, transient involvement of the left ventricular apex; it may be triggered by emotional or physiological stressors, including neurological complications.[1,2] In the context of thyroid disease, takotsubo cardiomyopathy has mainly been associated with thyrotoxicosis.[3]

We present the case of a patient with takotsubo cardiomyopathy and stroke following thyroidectomy.

Our patient was a 74-year-old woman with arterial hypertension, dyslipidaemia, type 2 diabetes mellitus, and depressive disorder. A CT scan performed due to recurrent bronchitis revealed intrathoracic goitre. The patient reported mild discomfort with swallowing and displayed normal thyroid hormone levels. A thyroidectomy was performed by a cervical approach, with no complications; at discharge, she was instructed to take levothyroxine 100 μg every 24 hours. The patient visited the emergency department 8 days later due to a sudden-onset episode of difficulty speaking and chest pain. In-hospital code stroke was activated. The examination revealed symptoms of moderate aphasia, hemianopsia, and mild right-limb hemiparesis (4/5); the patient scored 7 on the National Institutes of Health Stroke Scale (NIHSS). An emergency CT scan was performed as part of the code stroke protocol. The CT perfusion study revealed a pattern compatible with acute subcortical stroke in the left parietal and piriform areas, with 30% mismatch without large-vessel occlusion. Fibrinolysis was the only reperfusion therapy possible in this case, but was contraindicated due to the recent surgery. The patient was therefore admitted to the stroke unit and remained under observation. Laboratory analyses showed a troponin T level of 67 ng/L (normal range, 0-14), TSH of 6.02 μIU/mL (0.27-4.2), and free T4 of 0.99 ng/dL (0.93-1.7). Electrocardiography showed sinus rhythm at 100 bpm, with negative T waves in leads V4-V6. Echocardiography detected circumferential hypokinesis in the left ventricular apex with ejection fraction in the lower limit of normal. An angiography study of the coronary arteries detected no alterations. These findings are compatible with takotsubo cardiomyopathy. Treatment with anticoagulants and beta blockers started at admission improved the patient's symptoms, and she was discharged 21 days later with mild dysphasia and difficulty reading (NIHSS 1). An echocardiography performed at 3 months showed recovery of cardiac motility and normal ejection fraction. At present, the patient has no neurological or cardiological symptoms.

Takotsubo cardiomyopathy is characterised by transient dysfunction of the left ventricular apex, which presents with symptoms similar to those of myocardial infarction, but in the absence of coronary artery disease. Patients with takotsubo cardiomyopathy present electroencephalography alterations and moderately elevated troponin levels. The pathogenesis of the disease is unclear; an association with a hyperadrenergic state has been suggested.

With regard to thyroid disease, takotsubo cardiomyopathy has mainly been associated with hyperthyroidism,[3–6] but cases have also been described in patients with hypothyroidism, since they may present autonomous nervous system alterations in the form of coronary artery spasm.[8] Our patient, however, presented subclinical hypothyroidism.

She was a postmenopausal woman, a profile that is consistent with most of the cases reported.[7] Some series also report history of such cardiovascular risk factors as arterial hypertension (58%), diabetes mellitus (20%), and dyslipidaemia (37%).[6] Affective disorders may present in up to 42% of patients.[7]

This is the first reported case of takotsubo cardiomyopathy manifesting as stroke after surgery for goitre. The condition is triggered by a stressor in 25%-35% of cases.[6,7] Templin et al.[7] report neurological involvement in 27% of a sample of 1750 patients with takotsubo cardiomyopathy, and other cases have been reported of epileptic seizures,[9] subarachnoid haemorrhage,[10] and ischaemic stroke.[11–13] Likewise, cerebrovascular accidents may present during follow-up as a complication of takotsubo cardiomyopathy in up to 8% of cases.[6]

In our case, it is difficult to determine whether the stroke was the cause or the result of takotsubo cardiomyopathy.[14] The first option seems less likely, since the strokes that cause this type of cadiomyopathy have been associated with left insula involvement,[15] whereas our patient presented a stroke in the left subcortical parietal and piriform region, which is not directly associated with the insula.

This issue also has therapeutic implications. If cardiomyopathy were caused by stroke, the stroke would be cryptogenic and a more thorough aetiological study would be necessary. If, on the contrary, cardiomyopathy were the cause of stroke, the patient should receive anticoagulation therapy. We opted for the second hypothesis. After prolonged cardiac monitoring, we decided to suspend anticoagulation therapy, assuming that the cause of stroke had resolved.

In conclusion, takotsubo cardiomyopathy is a multifactorial disease that should be considered in some patients with thyroid disorders. Neurological disease may be the cause or a consequence of the syndrome. This issue should be analysed in future studies to gain a deeper understanding of the pathophysiology of the condition in association with neurological involvement.

Bibliografía

1. Akashi YJ, Goldstein DS, Barbaro G, Ueyama T. Takotsubo cardiomyopathy: a new form of acute, reversible heart failure. Circulation. 2008;118:2754—62.

☆ Please cite this article as: Febrero B, Rodríguez JM, Morales A, Parrilla P. Síndrome de Takotsubo asociado a infarto cerebral en postoperatorio de bocio eutiroideo. Neurología. 2020;35:592—593.

2. Ghadri JR, Wittstein IS, Prasad A, Sharkey S, Dote K, Akashi YJ, et al. International expert consensus document on Takotsubo syndrome (part I): clinical characteristics, diagnostic criteria, and pathophysiology. Eur Heart J. 2018;39:2032—46.
3. Eliades M, El-Maouche D, Choudhary C, Zinsmeister B, Burman KD. Takotsubo cardiomyopathy associated with thyrotoxicosis: a case report and review of the literature. Thyroid. 2014;24:383—9.
4. Al-Salameh A, Allain J, Meimoun P, Benali T, Desailloud R. Takotsubo cardiomyopathy can occur in patients with apathetic hyperthyroidism. Thyroid. 2014;24:400—1.
5. Dahdouh Z, Roule V, Bignon M, Grollier G. Síndrome de takotsubo recurrente asociado a hipertiroidismo subclínico. Rev Esp Cardiol. 2011;64:1069—71.
6. Aggarwal S, Papani R, Gupta V. Can thyroid break your heart? Role of thyroid in Takotsubo cardiomyopathy: a single center retrospective study. Int J Cardiol. 2015;184:545—6.
7. Templin C, Ghadri JR, Diekmann J, Napp LC, Bataiosu DR, Jaguszewski M, et al. Clinical features and outcomes of Takotsubo (stress) cardiomyopathy. N Engl J Med. 2015;373:929—38.
8. Rueda D, Aguirre R, Contardo D, Finocchietto P, Hernandez S, di Fonzo H. Takotsubo myocardiopathy and hyperthyroidism: a case report and literature review. Am J Case Rep. 2017;18:865—70.
9. Stollberger C, Wegner C, Finsterer J. Seizure-associated Takotsubo cardiomyopathy. Epilepsia. 2011;52:e160—7.
10. Inamasu J, Ganaha T, Nakae S, Ohmi T, Wakako A, Tanaka R, et al. Therapeutic outcomes for patients with aneurysmal subarachnoid hemorrhage complicated by Takotsubo cardiomyopathy. Acta Neurochir (Wien). 2016;158:885—93.
11. Scheitz JF, Mochmann HC, Witzenbichler B, Fiebach JB, Audebert HJ, Nolte CH. Takotsubo cardiomyopathy following ischemic stroke: a cause of troponin elevation. J Neurol. 2012;259:188—90.
12. Yoshimura S, Toyoda K, Ohara T, Nagasawa H, Ohtani N, Kuwashiro T, et al. Takotsubo cardiomyopathy in acute ischemic stroke. Ann Neurol. 2008;64:547—54.
13. Abi-Saleh B, Iskandar SB, Schoondyke JW, Fahrig S. Takotsubo syndrome as a consequence of transient ischemic attack. Rev Cardiovasc Med. 2006;7:37—41.
14. Finsterer J, Aliyev R. Takotsubo syndrome: consequence or cause of ischemic stroke. Funct Neurol. 2014;29:282.
15. Bersano A, Melchiorre P, Moschwitis G, Tavarini F, Cereda C, Micieli G, et al. Takotsubo syndrome as a consequence and cause of stroke. Funct Neurol. 2014;29:135—7.

B. Febrero[a,*], J.M. Rodríguez[a], A. Morales[b], P. Parrilla[a]

[a] *Unidad de Cirugía Endocrina, Servicio de Cirugía General, Hospital Clínico Universitario Virgen de la Arrixaca, Instituto Murciano de Investigación Biomédica (IMIB), El Palmar, Murcia, Spain*
[b] *Servicio de Neurología, Hospital Clínico Universitario Virgen de la Arrixaca, Instituto Murciano de Investigación Biomédica (IMIB), El Palmar, Murcia, Spain*

* Corresponding author.
E-mail address: beatrizfebrero@hotmail.com (B. Febrero).

27 October 2018

https://doi.org/10.1016/j.nrleng.2019.01.012
2173-5808/

Acute lower limb paresis in a patient with rheumatoid arthritis: emergency neuroimaging findings*

Paresia aguda de miembro inferior en paciente con artritis reumatoide: hallazgos en la neuroimagen de urgencia

Dear Editor:

Rheumatoid arthritis is a chronic, inflammatory, systemic disease that mainly affects ligaments, joints, and ultimately bone. Its pathophysiology involves genetic, autoimmune, and environmental factors. The condition is caused by synovial inflammation, leading to destruction of the joints.[1] However, the condition has also been associated with cardiovascular, pulmonary, digestive, haematological, and more rarely neurological manifestations.[2] Rheumatoid arthritis is one of the most frequent rheumatic disorders worldwide, with an estimated prevalence of 0.5%-1% in the adult population.[3,4]

We present the case of a 76-year-old woman with no known vascular risk factors, under treatment with methotrexate and folic acid due to chronic rheumatoid arthritis. She visited our hospital's emergency department due to inability to walk as a result of loss of muscle strength in the right leg of less than 24 hours' progression. She presented no neck pain and reported no history of trauma. The examination revealed paresis in the proximal part of the right leg (3/5), mild hyperreflexia of the right limbs, and extensor plantar reflex in the right foot, associated with impaired proprioceptive sensitivity (arthrokinetic and positional) in the right leg. Examination of cranial nerves and tactile and pain sensitivity revealed no alterations. An emergency blood analysis including a biochemistry study and a complete blood count yielded normal results, and a head CT scan revealed no alterations in the brain parenchyma. However, it did show incipient compression of the medulla oblongata by the dens of the axis, with a posterior atlantodental interval < 13 mm (radiological measurement between the posterior surface of the odontoid process and the posterior arch of the atlas,[5] with normal values being > 14 mm) (Fig. 1).

Clinical findings were anatomically correlated with radiological findings, and compatible with incomplete right

* Please cite this article as: Macías-García D, Jurado Serrano J, Parada Blazquez MJ, Moniche F. Paresia aguda de miembro inferior en paciente con artritis reumatoide: hallazgos en la neuroimagen de urgencia. Neurología. 2020;35:593—595.