

Case Report

길랑-바레 증후군에서 발생한 Takotsubo 심근병

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Takotsubo Cardiomyopathy Associated with Guillain-Barré Syndrome

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A 69-year-old woman presented with a progressive limb weakness. Both clinical and neurophysiological findings were consistent with diagnosis of Guillain-Barré syndrome (GBS). Two days after admission, the patient suffered from an acute coronary syndrome without stenosis at coronary arteriography. Echocardiography revealed left ventricular inferior wall and apical akinesia and decreased ejection fraction. A diagnosis of Takotsubo cardiomyopathy was then made. Left ventricular dysfunction and electrocardiography normalized within one month. Takotsubo cardiomyopathy can be developed as a complication of GBS. (Korean J Clin Neurophysiol 2015;17:73-75)

Key Words: Guillain-Barré syndrome, Heart failure, Takotsubo cardiomyopathy

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Cardiovascular abnormalities in the Guillain-Barré syndrome (GBS) are attributed to autonomic neuropathy and are reported in diverse manifestations such as heart rate and blood pressure variability, cardiomyopathy, and electrocardiographic (ECG) changes.¹ Takotsubo cardiomyopathy (TCM), also called ‘stress-induced cardiomyopathy’, is characterized by a reversible left ventricular dysfunction which needs adequate management and specific therapeutic strategies.² We describe a patient suffering from GBS who had stress-induced cardiomyopathy.

Case Report

A 69-year-old woman presented with a rapidly progressive limb weakness and hyporeflexia. She first noticed difficulty in right arm elevation and limb weakness was rapidly progressed. She had only hypertension as cardiovascular risk factor. A neurological examination demonstrated proximal and distal muscle weakness symmetrically in whole limbs and decreased deep tendon reflexes in biceps, triceps, knee, and ankle. Nerve conduction study was suggestive of axonal sensorimotor polyneuropathy. Cerebrospinal fluid examination did not reveal albuminocytologic dissociation. Cervical spine MRI was unremarkable. Both clinical course and neurophysiological study were consistent with diagnosis of GBS and a treatment by intravenous immunoglobulin over a 5-day period was started. Anti-ganglioside antibodies to GM1, GD1b, GT1a, GalNac-GD1a and GQ1b were all negative. At hospital day 2, she was

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in respiratory distress and mechanical ventilation was started. On the same day, standard 12-lead ECG showed T wave inversion and ST segment elevation in anterolateral leads (Fig. 1A). This ECG was suggestive of acute coronary syndrome and cardiac marker was elevated. She did not have any cardiac symptoms except respiratory distress. Trans-thoracic echocar-

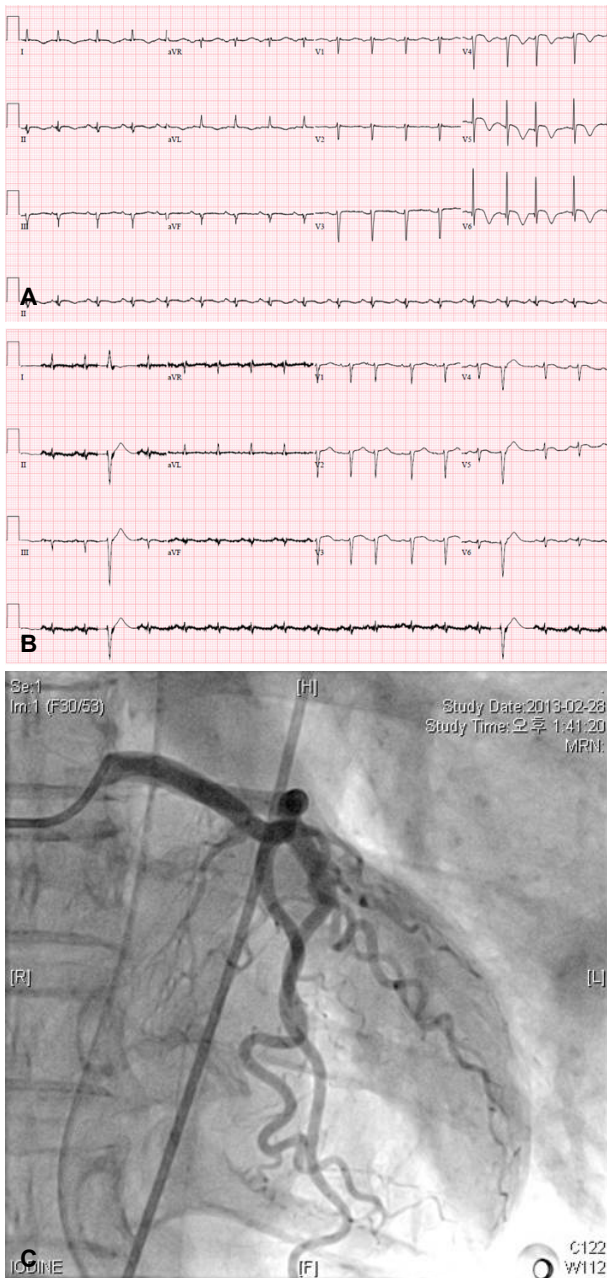


Figure 1. Standard 12-lead electrocardiogram and coronary arteriography of the patient. (A) EKG showed negative T wave in anterolateral leads on the 2nd hospital day. (B) EKG abnormality disappeared on the 3rd hospital day. (C) Coronary arteriography showed normal coronary artery.

diography revealed left ventricular inferior wall and apical akinesis, and decreased left ventricular ejection fraction. However, coronary arteriography did not reveal coronary artery stenosis (Fig. 1C). Left ventriculography revealed segmental wall motion abnormalities. A diagnosis of TCM was then made. Low blood pressure was treated with intravenous dobutamine. After the blood pressure was stable, angiotensin-converting enzyme inhibitor was administered. Left ventricular dysfunction and abnormal ECG normalized within one month (Fig. 1B).

Discussion

The GBS is an autoimmune disease affecting the peripheral nervous system and autonomic neuropathy is an important and common complication of GBS.³ In GBS, in addition to autonomic dysfunction involving the heart, TCM can occur. Up to now, there are few case reports about TCM in GBS.⁴⁻⁷ TCM is characterized by a reversible left ventricular systolic dysfunction due to enhanced central or focal sympathetic stimulation, triggered by emotional or physical stress. Clinical presentation and ECG changes mimic acute coronary syndrome or myocardial infarction. Contrary to myocardial infarction, coronary angiography is normal. There is a strong preference for post-menopausal females. Typically, echocardiographic and ECG abnormalities resolve within weeks if heart failure and concomitant arrhythmia are appropriately treated.^{8,9}

We describe the case of a 69-year-old woman who was diagnosed with GBS and developed acute coronary syndrome without coronary artery stenosis, consistent with the diagnosis of a TCM. TCM has been occasionally reported in association with neuromuscular disorders. Among them, myasthenia gravis was most commonly associated with TCM.¹⁰ However, it is under debate if the underlying neuromuscular disorder is involved in the pathogenesis of TCM and if there is a causal relationship between the neuromuscular disorders and TCM. Neuromuscular disorders seem to favor the development of TCM under stress condition.

The pathophysiology of TCM remains unclear but the tool of catecholamine-mediated myocardial stunning may be predominant.² Most cardiovascular effects of the GBS are related primarily to autonomic dysfunction due to an immune injury of the autonomic nervous system.³ Triggers of TCM in GBS may be catecholamine treatment,⁷ immunoglobulin treatment

or involvement of the autonomic fibers in GBS,⁶ or autonomic dysfunction.^{4,5} In TCM, supportive treatment associating β -blocker and angiotensin-converting enzyme inhibitor therapies is recommended until complete recovery of left ventricular ejection fraction. GBS occurrence can be the stressful trigger of TCM. Therefore, frequent monitoring is needed and trans-thoracic echocardiography should be performed when electrocardiographic abnormalities are present in GBS to rule out a TCM.

Conflict of Interest

The authors declare that they have no financial or other conflicts of interest in relation to this research and its publication.

REFERENCES

1. Mukerji S, Aloka F, Farooq MU, Kassab MY, Abela GS. Cardiovascular complications of the Guillain-Barré syndrome. *Am J Cardiol* 2009;104:1452-1455.
2. Bybee KA, Prasad A. Stress-related cardiomyopathy syndromes. *Circulation* 2008;118:397-409.
3. Zochodne DW. Autonomic involvement in Guillain-Barré syndrome: a review. *Muscle Nerve* 1994;17:1145-1155.
4. Fugate JE, Wijdicks EF, Kumar G, Rabinstein AA. One thing leads to another: GBS complicated by PRES and Takotsubo cardiomyopathy. *Neurocrit care* 2009;11:395-397.
5. Iga K, Himura Y, Izumi C, Miyamoto T, Kijima K, Gen H, et al. Reversible left ventricular dysfunction associated with Guillain-Barré syndrome-an expression of catecholamine cardiotoxicity? *Jpn Circ J* 1995;59:236-240.
6. Martins RP, Barbarot N, Coquerel N, Baruteau AE, Kolev I, Vérin M. Takotsubo cardiomyopathy associated with Guillain-Barré syndrome: a differential diagnosis from dysautonomia not to be missed. *J Neurol Sci* 2010;291:100-102.
7. Quick S, Quick C, Schneider R, Sveric K, Katzke S, Strasser RH, et al. Guillain-Barré syndrome and catecholamine therapy. A potential risk for developing takotsubo cardiomyopathy? *Int J Cardiol* 2013;165:43-44.
8. Porto I, Della Bona R, Leo A, Proietti R, Pieroni M, Caltagirone C, et al. Stress cardiomyopathy (Tako-tsubo) triggered by nervous system disease: a systemic review of the reported cases. *Int J Cardiol* 2013;167:2441-2448.
9. Tsuchihashi K, Ueshima K, Uchida T, Oh-mura N, Kimura K, Owa M, et al. Transient left ventricular apical ballooning without coronary artery stenosis: a novel heart syndrome mimicking acute myocardial infarction. Angina Pectoris-Myocardial Infarction Investigations in Japan. *J Am Coll Cardiol* 2001;38:11-18.
10. Finsterer J, Stöllberger C. Neuromuscular disorders and Takotsubo syndrome. *Int J Cardiol* 2013;168:4293-4294.