Reversible Left Ventricular Dysfunction Associated with Guillain-Barré Syndrome
—— An Expression of Catecholamine Cardiotoxicity? ——

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The patient was a 76-year-old female who had a history of Guillain-Barré syndrome 3 years previously; ST-segment elevation was noted in association with reversible left ventricular dysfunction. Left ventriculogram and coronary angiograms were normal and ergonovine test was negative during the chronic period of Guillain-Barré syndrome. She was hospitalized again due to the recurrence of Guillain-Barré syndrome. Two days later, ST-segment elevation in leads V2 through V5 prompted us to perform cardiac catheterization, although she did not complain of any chest symptoms. A large akinetic area was found mainly around the apex on left ventriculography, despite the lack of coronary stenoses. Peak creatine kinase and C-reactive protein were 400 IU/ml and 3.5 mg/dl, respectively. Left ventricular dysfunction was normalized within one week.

During the acute phase of the cardiac episode, plasma norepinephrine and epinephrine were 1340 pg/ml and 112 pg/ml, respectively. 123I metaiodobenzylguanidine myocardial scintigram 3 weeks after the episode showed an extensive apical defect which was improved markedly 3 months later.

We think that this reversible left ventricular dysfunction was due to the synergistic toxic effect of mildly increased catecholamine and transiently damaged sympathetic nerve endings in the myocardium, presumably due to Guillain-Barré syndrome.

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G U I L L A I N - B A R R É syndrome is an inflammatory polyradiculoneuropathy in which the autonomic nervous system is sometimes involved! ST-segment and T-wave abnormalities have been reported in cases in which the autonomic nervous system is involved, and sudden death can be attributed to fatal arrhythmia or malignant hypertension? Permanent pacemaker insertion may be necessary3. However, little is known about left ventricular wall motion abnormalities in cases of Guillain-Barré syndrome with ST-segment and T-wave changes in the electrocardiogram (ECG).

We present a case of Guillain-Barré syndrome associated with reversible left ventricular dysfunction and discuss the mechanism of catecholamine cardiotoxicity based on 123I-metaiodobenzylguanidine (MIBG) myocardial scintigraphy.

Key words:
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CASE REPORT

The patient was a 76-year-old female who was hospitalized due to recurrence of Guillain-Barré syndrome. Her history included Guillain-Barré syndrome 3 years previously; ST-segment elevation was observed in leads V4 through V6 and lasted for 3 h during artificial ventilation associated with transient segmental left ventricular dysfunction, although the ECG taken on admission was normal. Coronary angiography performed 2 months after the acute phase was normal and ergonovine test was negative. Left ventricu-
lar end-diastolic volume (EDV) and ejection fraction (EF) were 105 ml and 71%, respectively (Fig. 1).

Two days after the present admission, motor paralysis progressed gradually and ST-segment elevation was noted in leads I, aVL, and V2 through V5, although the ECG obtained on admission was normal (Fig. 2). Peak creatine kinase (CK) and C-reactive protein were 400 IU/ml and 3.5 mg/dl, respectively. Blood pressure was 130/70 mmHg and heart rate was 100 min. On emergent coronary angiography, there was no significant stenosis and or spasm during ST-segment elevation. However, the left ventriculogram showed a large akinetic area around the apex with EDV and EF of 107 ml and 36%, respectively. We did not perform myocardial biopsy. Serial two-dimensional echocardiographic studies revealed no

Fig. 3. Electrocardiogram 4 weeks after the episode on the second admission shows a deep negative T wave in leads V3 through V6.

Fig. 4. I123 metaidobenzylguanidine myocardial scintigram shows an extensive defect (upper panels) which disappeared 3 month later (lower panels). A: vertical long axis view, B: short axis view, C: horizontal long axis view.
pericardial effusion. Artificial respiration was required due to motor paralysis and a marked change in blood pressure was observed, but heart rate did not fluctuate. Plasma norepinephrine and epinephrine were 1340 pg/ml and 112 pg/ml, respectively, but decreased to 266 pg/ml and 31 pg/ml 2 weeks later. ST-segment elevation persisted for 3 days without the appearance of a new Q wave, followed by deep negative T waves in V3 through V6 4 weeks after the episode (Fig. 3). Left ventricular dysfunction normalized within 1 week and motor paralysis improved gradually by plasmapheresis.

123I-MIBG myocardial scintigram performed 3 weeks after the episode showed a defect around the apex which was improved markedly 3 months later (Fig. 4). She has thus far had no occurrence of chest pain.

DISCUSSION

In the present case, transient left ventricular dysfunction normalized after one week as motor paralysis improved. In the initial episode of Guillain-Barré syndrome 3 years previously, coronary angiography was performed 2 months after ST-segment elevation. There was no significant coronary stenosis and ergonovine test was negative. In the second attack, emergent coronary angiograms were normal while ST-segments was elevated. Therefore, this reversible left ventricular dysfunction was not caused by coronary arterial lesions.

The diffuse ST-segment elevation seen in this patient is also characteristic of viral myocarditis. Guillain-Barré syndrome has been thought to be caused by an autoimmune mechanism, and an altered immune response associated with infection may play an important role in most cases. Causative organisms that have been demonstrated thus far include cytomegalovirus, Epstein-Barr virus, human immunodeficiency virus, Mycoplasma pneumoniae, and Campylobacter jejuni. In contrast, myocarditis is commonly caused by enterovirus species. The slight increase in CK despite the large akinetic area and the rapid improvement in left ventricular motion suggests that myocardial damage was minimal compared with that in viral myocarditis. In both episodes, ST-segment elevation was associated with maximal limb paralysis, but ECG was normal at the initial presentation. Although the development of parainfectious viral myocarditis simultaneously with Guillain-Barré syndrome could not be completely ruled out, it is unlikely that viral myocarditis caused this diffuse ST-segment elevation.

Small doses of catecholamine act as a cardiotoxic, while large doses of catecholamine are notoriously cardiotoxic. Experimentally, infusion of large doses of norepinephrine induces myocardial damage, and pheochromocytoma is a clinical example of left ventricular dysfunction caused by an extraordinary amount of catecholamine. In Guillain-Barré syndrome, hyperadrenergic symptoms have been reported, such as marked fluctuations of both blood pressure and heart rate. In the present case, clinical signs of adrenergic hyperactivity were not marked and the plasma norepinephrine level during the acute episode increased to about 1000 pg/ml, but not enough to induce left ventricular dysfunction.

Cardiac sympathetic innervation was estimated by 123I-MIBG myocardial scintigraphy. An extensive MIBG scintigraphic defect was found 3 weeks after recovery from left ventricular dysfunction and was almost normalized 3 months later. These findings suggest that cardiac sympathetic nerve damage was transiently present during the acute phase of Guillain-Barré syndrome. Since sympathetic function, especially the process of neuronal uptake (Uptake-1) in nerve endings, is the predominant means for terminating the action of the sympathetic neurotransmitter norepinephrine, cardiac sympathetic nerve damage can be expected to cause a local excess of norepinephrine, to which the myocyte is exposed. Therefore, in the present case, since it was presumed that cardiac sympathetic nerve endings were transiently damaged by Guillain-Barré syndrome, plasma catecholamine was able to reach a level sufficient to cause catecholamine-induced myocardial dysfunction.

There have been no cardiac symptoms to date. In the initial episode, ST-segment elevation was detected by electrocardiographic monitoring of the patient during artificial respiration. In the second episode, serial ECG's revealed similar ST-segment elevation. If serial ECG's had not been
taken, this left ventricular dysfunction may not have been discovered. Thus, electrocardiographic monitoring is necessary during the acute phase of Guillain-Barré syndrome.

REFERENCES


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