Case Report

Rapidly Progressive Deteriorated Left Ventricular Wall Motion Associated with Tetanus: A Case Report

Kanji IGA and Kenjiro HORI

A case of tetanus accompanied by severe reversible left ventricular motion impairment is reported. Emergent coronary angiography showed no abnormalities and large akinesis was present on left ventriculography.

The plasma catecholamine level was not too high in the acute phase, therefore it is thought that the cause of this depressed left ventricular wall motion abnormality was due to some metabolite derived from tetanus.

Key words: Tetanus, Echocardiography, Left ventricular disturbance, Congestive heart failure, Catecholamine

Cardiac arrest is the second most common cause of death due to tetanus. A histological study of autopsied hearts showed that toxic myocarditis is present (1). However, there has been only one report in which reversible left ventricular dysfunction was

documented by echocardiography (2). We describe here a case of tetanus accompanied by reversible left ventricular motion impairment. Coronary angiography revealed no significant stenosis in the acute phase. The cause of this left ventricular motion

Table 1. Clinical course.

	19/July	26/July	29/July	15/Aug.	19/Sep.	
		throat discomfort				
		dysphagia				
		dysarthria				
		difficulty in opening mouth				
		admission				
TIG		3000IU	1500IU	3000IU		
NE (<400pg/mL*)		5912	579	1512	112	254
E (<100pg/mL*)		1305	89	127	<10	<10
DA (<30pg/mL*)		570	10	36	<10	<10
EDV (mL)		84	61		57	
ESV (mL)		68	35		27	
EF (%)		20	42		53	

Abbreviation: TIG=Tetanus immunoglobulin, NE=Norepinephrine, E=Epinephrine, DA=Dopamine, EDV=End-diastolic volume (by echocardiography), ESV=End-systolic volume (by echocardiography), EF=Ejection fraction * normal range

From Department of Cardiology, Tenri Hospital, Tenri
Received for publication August 17, 1989; Accepted for publication April 25, 1990
Reprint requests should be addressed to Kanji Iga, MD, Department of Cardiology, Tenri Hospital,
200 Mishimacho, Tenri 632, Japan

abnormality is discussed.

CASE REPORT

A 73-year-old man was healthy until one week prior to admission when he noticed throat discomfort. Three days prior to admission, dysphagia and dysarthria developed, and his family noticed that his voice had become feeble. On the day of admission, trismus was present and he complained of dyspnea. He was admitted to the emergency ward (see Table 1, the clinical course). On physical examination, he appeared acutely ill and dyspneic. His eyes would

not open fully. His pulse rate was 90/min, blood pressure 184/90 mmHg, respiration rate 20/min and body temperature 36.5°C.

The chest X-ray revealed an interstitial lung shadow compatible with pulmonary edema. Two-dimensional echocardiography revealed that left ventricular wall motion deteriorated progressively in a few hours. As the electrocardiogram showed a QS pattern in leads V1 through V4 (Fig. 1), coronary angiography was performed to rule out acute myocardial infarction. Left ventricular end-diastolic pressure was 22 mmHg and cardiac output was 4.1

J.I. 73M 1988

Fig. 1. Serial electrocardiogram. QS pattern is seen in V1 through V4 on July 26 (admission day). Diffuse T-wave abnormalities seen on all chest leads on Aug. 17 returned to normal on Oct. 17.

l/min. The coronary artery was not significantly stenotic (Fig. 2-A) and left ventriculogram revealed a diffuse akinesis except for a postero-basal and antero-basal segment with ejection fraction 35% (Fig. 2-B). Creatine-phosphokinase was 860 U/l and the MB fraction was 3%.

Toxic myocarditis associated with tetanus was suspected and respiratory support with tracheostomy was performed. Tetanus-immune-human gamma-globulin (1500 IU) was injected three times thereafter. Although blood pressure and pulse rate fluctuated greatly, the patient's general condition improved within one week, and left ventricular motion, seen on echocardiography, rapidly returned to almost normal with an ejection fraction of 53% (Fig. 3). Plasma norepinephrine and epinephrine were increased maximally to 5912 pg/ml (normal <400) and to 1305 pg/ml (normal <100), respectively during the acute phase, and returned to normal after tetanus was cured. Local tetanus infection was not detected.

DISCUSSION

The diagnosis of tetanus is usually made clinically. Dysarthria, dysphagia, difficulty in opening the mouth and trismus form the typical time

sequence of tetanus. Although no focus of local infection could be detected in this patient, the diagnosis was definite. Left ventricular wall motion was severely disturbed in the acute phase of tetanus, but was not associated with coronary artery disease. It was normalized within a week.

Left ventricular wall motion impairment has been observed in pheochromocytoma patients, and experimental infusion of large amounts of catecholamine is known to result in myocarditis. Histological study of myocardial damage secondary to tetanus is very similar to that of pheochromocytoma (1). Sympathetic hyperactivity has been reported in cases of tetanus (3), and labile hypertension and profuse sweating were observed in this case. Iga et al reported a case of severe reversible left ventricular motion impairment in pheochromocytoma (4). The plasma epinephrine and norepinephrine levels were extraordinarily high, greater than one million pg/ml. The plasma catecholamine level during the acute phase of tetanus was about 15 times the normal maximum level, but not high enough to produce left ventricular dysfunction; it returned to normal after tetanus was cured.

We think that the reversible left ventricular wall motion abnormality in this case was due to a

Fig. 2. A) Coronary arteriography showed no significant stenosis. B) Left ventriculography of right anterior oblique view revealed akinesis except for the postero-basal and antero-basal segment. ED, end-diastole; ES, end-systole; EF, ejection fraction; EDV, end-diastolic volume; ESV, end-systolic volume

Fig. 3. Two-dimensional echocardiography of end-diastolic and end-systolic phase. Left ventricle showed mild concentric hypertrophy with an almost normal ejection fraction. ED, end-diastole; ES, end-systole; EF, ejection fraction; EDV, end-diastolic volume; ESV, end-systolic volume

myocardial depressant factor secondary to tetanus, as is occasionally observed in cases of sepsis (5, 6), rather than due to excessive catecholamine.

REFERENCES

- 1) Rose AG. Catecholamine-induced myocardial damage associated with pheochromocytoma and tetanus. *S Afr Med J* **48**: 1285, 1974.
- 2) Lieshout JJ, Wieling W, Romjin JA, et al. Hyperadrenergic syndrome with hypertension, hypotension and myocardial necrosis in tetanus. *Neth J Med* **33**: 33, 1988.
- 3) Tueda K, Oliver PB, Richter RW. Cardiovascular manifestation of tetanus. *Anesthesiology* **40**: 588, 1974.
- 4) Iga K, Gen H, Matumura T, et al. Reversible left ventricular wall motion impairment in pheochromocytoma: A case report. *Jpn Circ J* **53**: 813, 1989.
- 5) Elldodt AG, Riedinger MS, Kimchi A, et al. Left ventricular performance in septic shock: Reversible segmental and global abnormalities. *Am Heart J* **110**: 402, 1985.
- 6) Parrillo JE, Burch C, Shelhamer JH, et al. A circulating myocardial depressant substance in human septic shock. *J Clin Invest* **76**: 1539, 1985.