A Case of Crush Syndrome With Giant Negative T Waves and Reversible Left Ventricular Dysfunction

Kazumasa Adachi, M.D., Masahito Kawata, M.D., Shun-ichi Araki, M.D.
Akira Matsumoto, M.D., Tomoichiro Mukai, M.D.*
and Teishi Ikoma, M.D.

A 54-year-old male experienced the Hanshin earthquake at 5:46 am on Jan. 17, 1995. He was rescued after being buried under his house for 20 h. After being treated at two hospitals, he was admitted to our hospital with acute renal failure caused by crush syndrome. The maximal serum creatinine kinase level was 35,000 IU/L (CK-MM: 100%), and the maximal myoglobin level was 12,600 ng/ml. An electrocardiogram showed inverted T waves in V_{5,6} which later became giant negative T waves, and a QS pattern in aVL and V₁₋₆. Two-dimensional echocardiography showed a hypokinetic left ventricle. He was treated with hemodialysis 13 times. Cardiac contraction gradually improved to the normal level. Coronary angiography revealed normal coronary arteries and acetylcholine did not induce coronary spasm. Biopsy specimens from the right ventricular septum showed slight degeneration. ²⁰¹Tl imaging showed hypoperfusion except in the anterior wall and ¹²³I-MIBG imaging showed a perfusion defect except in the anterior wall. After about 1 month, only ²⁰¹Tl imaging showed improvement. We believe that myocardial ischemia, cardiac sympathetic nerve damage and/or cardiac contusion caused giant negative T waves and left ventricular dysfunction. This is a rare case which showed significant myocardial damage with crush syndrome.

(*Jpn Circ J* 1996; **60**: 809–814)

AT 5:46 am on Jan. 17, 1995, a terrible earthquake struck the Hanshin-Awaji district in Hyogo prefecture, Japan. Many people were rescued from collapsed houses. Some of them had severe muscle damage followed by crush syndrome. Crush syndrome was first described by Bywaters and Beall in their study of air-raid casualties during World War II! They described patients who had been trapped for extended periods of time in fallen debris. On autopsy, numerous casts, later identified as myoglobin, were

found in the renal tubules. The crush injury of muscles induces myoglobin release, which often causes renal failure? However, crush injury or crush syndrome with myocardial damage is rarely reported^{3,4}. We experienced a case of crush syndrome following the Hanshin earthquake. The electrocardiogram (ECG) showed giant negative T waves, and two-dimensional echocardiography showed severe left ventricular dysfunction.

CASE REPORT

A 54-year-old male experienced the Hanshin earthquake at 5:46 am on Jan. 17, 1995. The wall of his house crushed him on the right side of his chest. He remained alert but

Key words:

Crush syndrome
Giant negative T
Left ventricular dysfunction
Metaiodobenzylguanidine (MIBG)
Emotional stress

(Received September 4, 1995; accepted February 27, 1996)

Departments of Cardiology and *Surgery, Chibune Hospital, Osaka, Japan

Mailing address: Kazumasa Adachi, M.D., Department of Cardiology, Chibune Hospital, 2-2-45 Tsukuda, Nishiyodogawa-ku, Osaka 555, Japan

Fig 1. Changes in ECGs. A, 4 months before admission. The ECG was almost normal. B, on admission (2 days after the earthquake), the ECG showed inverted T waves in V_{5,6}, low voltage in limb leads, and a QS pattern in aVL and all of the chest leads. C, on the 44th hospital day, negative T waves in I, II, III, aVF and V₄₋₆ became maximal, and those in V_{4,5} became giant negative T waves. The QS pattern disappeared. D, after he was discharged, only shallow inverted T waves remained in V_{5,6}.

could not move because of the wall on his chest and waist. He was rescued after being buried under his house for 20 h. After being treated at two hospitals, he was admitted to

our hospital with acute renal failure caused by crush syndrome on Jan. 19. He complained of mild right chest pain without dyspnea. On admission, his blood pressure

Fig 2. ^{201}Tl and ^{123}I -MIBG images at rest. SPECT imaging was performed with ^{201}Tl and ^{123}I -MIBG. Representative early images in the vertical long axis, short axis and horizontal long axis views are shown. A, ^{201}Tl images on the 35th hospital day show hypoperfusion except in the left ventricular anterior wall. B, ^{123}I -MIBG images on the 35th hospital day show a perfusion defect, except in part of the left ventricular anterior wall. C, ^{201}Tl images on the 65th hospital day show mild hypoperfusion in the posterior septal and apical walls. The uptake had recovered compared to that in Fig 2A. D, ^{123}I -MIBG images on the 65th hospital day show a perfusion defect, except in part of the left ventricular anterior wall. There is little change compared to those in Fig 2B.

was 120/90 mmHg and his heart rate was 70 min. Chest X-ray showed mild lung congestion. The cardiothoracic ratio was 54%. Laboratory findings on admission were as follows: red blood cell count 450×10^4 ml, hemoglobin 13.6 g/dl, hematocrit 42.4%, white blood cell count 12,100 ml, platelet 9.6×10^4 ml, serum total protein 5.1 g/dl, albumin 3.7 g/dl, creatinine kinase (CK) 35,000 IU/L (CK-MM: 100%), myoglobin 12,600 ng/ml, aspartate aminotransferase (GOT) 704 IU/L, alanine aminotransferase (GPT) 332 IU/L, lactate dehydrogenase (LDH) 3,700 IU/L, blood urea nitrogen 100 mg/dl, creatinine 5.7 mg/dl, Na 133 mmol/L, K 6.8 mmol/L, Cl 102 mmol/L and Ca 7.0 mg/dl. He also showed anuria. The ECG showed negative T waves in $V_{5,6}$, and a QS pattern in aV_L and V_{1-6} (Fig 1B). Two-dimensional and Doppler echocardiography showed left ventricular hypokinesis (% frac-

tional shortening was 10%) without valvular regurgitation or pericardial effusion. He was diagnosed as acute heart failure and renal failure. Hemodialysis was performed for 2 to 3 h every day until the ninth hospital day and the urinary volume gradually increased to 800 ml per day. Thereafter, hemodialysis was performed 2 times a week, and was finally discontinued on the 19th hospital day. The urinary volume rose to about 2,000 ml per day and renal failure disappeared. On the 30th hospital day, serum epinephrine, noradrenaline, and dopamine levels were 18 pg/ml, 314 pg/ml, and 19 pg/ml, respectively. Left ventricular contraction gradually improved and echocardiography showed a % fractional shortening of 30% on the 10th hospital day. ECGs also changed. An ECG 4 months before the earthquake showed neither negative T waves nor poor R wave progression (Fig 1A). However, the ECG

on admission showed inverted T waves in $V_{5,6}$, low voltage in limb leads, and a QS pattern in aV_L and all of the chest leads (Fig 1B). On the 44th hospital day, the voltage of negative T waves in I, II, III, aV_F and V_{4-6} became maximal, and giant negative T waves (more than 1 mV) appeared in $V_{4,5}$ without Q waves (Fig 1C). After he was discharged, only shallow inverted T waves remained in $V_{5,6}$ (Fig 1D). On the 43rd hospital day, coronary angiogram showed normal coronary arteries and intracoronary injection of acetylcholine did not induce coronary arterial spasm. Left ventriculography showed a hypokinetic left ventricle, except in the basal portions. The ejection fraction of the left ventricle was 49%. Myocardial biopsy specimens from the right ventricular septum demonstrated slight degeneration of cardiac muscle cells without clear necrosis or fibrosis. On the 35th hospital day, single-photon emission computed tomography (SPECT) imaging using $^{201}\text{TlCl}$ showed hypoperfusion, except in the left ventricular anterior wall (Fig 2A). On the same day, SPECT imaging using ^{123}I -metaiodobenzyl-guanidine (MIBG) showed a perfusion defect, except in part of the left ventricular anterior wall (Fig 2B). On the 65th hospital day, ^{123}I -MIBG imaging still showed a broad defect, except in the left ventricular anterior wall, while ^{201}Tl imaging showed hypoperfusion in the posterior septal and apical walls (Fig 2, C and D). Heart failure disappeared 2 weeks after admission. Due to right sciatic nerve palsy, he was discharged after 2 months of walking rehabilitation. About 1 year after the earthquake, brain computed tomography showed no evidence of cerebrovascular attack. ECG on the same day showed no T wave abnormality and mild poor R wave progression in V_{1-3} . Echocardiography showed normal left ventricular contraction (% fractional shortening was 36%).

DISCUSSION

Crush syndrome consists of various events following crush injury. Serious soft tissue and muscle compression leads to muscle injury and necrosis followed by hypovolemia, compartment syndrome, rhabdomyolysis, electrolyte imbalances, coagulopathy and

renal failure? Although crush syndrome is a muscle-related disease, myocardial damage in crush syndrome has rarely been reported^{3,4}. In our case, severe myocardial damage appeared in a patient who had been buried under his house for 20 h after an earthquake. Chest X-ray, ECG, echocardiography, ventriculography and scintigraphy showed cardiac abnormalities. Interestingly, the coronary arteries seemed intact and the myocardial damage was reversible. Among these data, we will mainly discuss giant negative T waves, low uptake of ^{201}Tl and ^{123}I -MIBG and left ventricular dysfunction, which were apparent but difficult to clearly explain.

Giant negative T waves were defined as negative T waves of more than 1 mV⁵. Based on the appearance of the initial ECG, we suspected silent old anterior myocardial infarction. Abnormal Q waves suggested transmural myocardial necrosis. However, coronary angiography showed normal coronary arteries which did not respond to intracoronary injection of acetylcholine; ie, there was little possibility of extramyocardial coronary vasospasm. Giant negative T waves are often observed in apical hypertrophic cardiomyopathy⁶. However, echocardiography showed no left ventricular hypertrophy in our case. Echocardiography and left ventriculography also showed diffuse hypokinesis in both the anterior and posterior walls. It is intriguing that left ventricular contraction gradually recovered and finally became almost normal.

^{123}I -MIBG imaging showed a perfusion defect which persisted up to the 65th hospital day. On the other hand, the area of hypoperfusion of the ^{201}Tl images was smaller on the 65th hospital day. These phenomena indicate that left ventricular myocardial blood flow decreased on the 35th hospital day and had recovered about 1 month later. Coronary angiography showed adequate coronary blood flow on the 43rd hospital day. Therefore, these coronary and scintigraphical data suggest that there was a temporary depletion of myocardial blood flow, followed by its recovery. Abnormal Q waves and contraction abnormality indicate the presence of transmural necrosis. However, a myocardial biopsy specimen from the right ventricular septum showed no necrosis

in the myocardium and abnormal Q waves disappeared 1 month later. Curiously, once the myocardium had become necrotic, it should not have been able to recover within 2 or 3 months. To resolve this contradiction, these changes may not have been due to myocardial necrosis, but rather to temporary myocardial ischemia or damage.

It is very difficult to clarify the cause of myocardial damage in this case. However, there are several possibilities: (1) vasospasm; (2) microvascular thrombosis; (3) sympathetic myocardial damage; and (4) mechanical injury of coronary arteries or myocardium. Although we could not induce vasospasm of extramyocardial coronary arteries, it is possible that the acetylcholine provocation test gave a false negative, or that ischemia was induced at the microvascular level. Severe emotional stress and dehydration during his accident may have increased the plasma viscosity followed by microvascular thrombosis in coronary arteries. Emotional stress induces platelet activation and sometimes triggers acute myocardial infarction.⁷⁻¹⁰ The number of acute myocardial infarctions actually increased during the Hanshin earthquake.¹¹ In our case, excessive emotional stress may have induced intracoronary microthrombi which dissolved in a short time, while ischemic injury remained. Left ventricular wall motion appears to have recovered faster than the changes in the ECG. This discrepancy indicates some other cause in this case. Since ¹²³I-MIBG uptake represents cardiac sympathetic nervous activity, perfusion defects in ¹²³I-MIBG imaging indicate sympathetic nervous dysfunction which was induced by excessive emotional stress and catecholamines released during the life-threatening accident.^{12,13} Although we could not analyze the serum catecholamine levels on admission, similar cases have been reported in brain damage in cerebrovascular attacks and neurosurgical operations. Hondo et al described 11 cases with inverted T waves, including 5 cases with wall motion abnormalities, among 40 acute cerebrovascular attacks.¹⁴ Hanaoka et al at Kobe University encountered 14 cases who showed inverted T waves without myocardial infarction after the Hanshin earthquake (unpublished data). ¹²³I-MIBG imaging showed focal defects in 11 cases and

complete defects in 3 cases. They suggested that abnormal sympathetic nervous function may cause ECG changes. Sympathetic nervous damage may explain the rather fast recovery of left ventricular hypokinesis and the slow change in ECGs.

Finally, coronary injury or myocardial contusion may have been the cause in this case, as described in severe blunt chest trauma, such as in traffic accidents.^{3,4,15,16} In one study, one third of the cases of traumatic myocardial infarction showed intact coronary arteries, probably because of spontaneous reperfusion or vasospasm.¹⁷ As of 1993, 31 cases of acute myocardial infarction due to blunt chest trauma had been reported, in which coronary injury was confirmed by coronary angiography.¹⁸ Most cases of cardiac contusion are due to motor vehicle accidents.^{4,16} Cardiac contusion is rarely caused by crush injury.^{3,4} Malangorni et al reported 12 cases of blunt cardiac injury, one of which had crush injury to the chest.³ Nine of 147 blunt chest injury cases were due to crush injury, and echocardiography showed wall motion abnormality in only four of the 147 cases.⁴ Cardiac contusion involves hemorrhage and edema of myocardial tissue, in addition to patchy necrosis.¹⁹ The accurate diagnosis of cardiac contusion is difficult because of imperfect diagnostic criteria and the absence of reliable indicators of myocardial injury other than necropsy.²⁰ Electrocardiography is a sensitive method, but has low specificity.⁴ Echocardiography is a good method, but it is technically difficult to obtain adequate echocardiograms from traumatic patients.⁴ Recently, SPECT imaging with ²⁰¹TlCl and ¹¹¹In-antimyosin scintigraphy have been used to detect myocardial injury, although their sensitivity is low.^{20,21} CK-MB also has limited sensitivity and specificity because of the large amount of CK-MM released in traumatic patients.⁴ It is possible that our case had cardiac contusion or coronary injury, based on the spontaneous recovery of ECG changes and cardiac function. However, giant negative T waves have not been previously associated with cardiac contusion. Furthermore, the damaged area may have been too large to attribute our case to cardiac contusion or coronary injury.

In conclusion, although it is very difficult

to determine the cause of myocardial damage in our case, it is important to recognize that crush syndrome may be accompanied by cardiac disorders, although this is rather rare. It is also important to examine patients with crush syndrome using noninvasive methods such as electrocardiography, echocardiography and myocardial scintigraphy.

REFERENCES

1. Michaelson M: Crush injury and crush syndrome. *World J Surg* 1992; **16**: 899–903
2. Cheney P: Early management and physiologic changes in crush syndrome. *Crit Care Nurs Q* 1994; **17**: 62–73
3. Malangoni MA, McHenry CR, Jacobs DG: Outcome of serious blunt cardiac injury. *Surgery* 1994; **116**: 628–633
4. Paone RF, Peacock JB, Smith DLT: Diagnosis of myocardial contusion. *Southern Med J* 1993; **86**: 867–870
5. Yamaguchi H, Ishimura T, Nishiyama S, Nagasaki F, Nakanishi S, Takatsu F et al: Hypertrophic nonobstructive cardiomyopathy with giant negative T waves (apical hypertrophy): ventriculographic and echocardiographic features in 30 patients. *Am J Cardiol* 1979; **44**: 401–412
6. Usui M, Inoue H, Suzuki J, Watanabe F, Sugimoto T, Nishikawa J: Relationship between distribution of hypertrophy and electrocardiographic changes in hypertrophic cardiomyopathy. *Am Heart J* 1993; **126**: 177–183
7. Markovitz JH, Matthews KA: Platelets and coronary heart disease: potential psychophysiologic mechanisms. *Psychosom Med* 1991; **53**: 643–668
8. Grignani G, Pacchiarini L, Zucchella M, Tacconi F, Canevari A, Soffiantino F et al: Effect of mental stress on platelet function in normal subjects and in patients with coronary artery disease. *Haemostasis* (Switzerland) 1992; **22**: 138–146
9. Smith M, Little WC: Potential precipitating factors of the onset of myocardial infarction. *Am J Med Sci* 1992; **303**: 141–144
10. Gelernt MD, Hochman JS: Acute myocardial infarction triggered by emotional stress. *Am J Cardiol* 1992; **69**: 1512–1513
11. Suzuki S, Sakamoto S, Miki T, Matsuo T: Hanshin-Awaji earthquake and acute myocardial infarction. *Lancet* 1995; **345**: 981
12. Kline RC, Swanson DP, Wieland DM, Thrall JH, Gross MD, Pitt B et al: Myocardial imaging in man with I-123 meta-iodobenzylguanidine. *J Nucl Med* 1981; **22**: 129–132
13. Sisson JC, Lynch JJ, Johnson J, Jaques Jr S, Wu D, Bolgos G et al: Scintigraphic detection of regional disruption of adrenergic neurons in the heart. *Am Heart J* 1988; **116**: 67–76
14. Hondo T, Shimizu W, Karakawa S, Kurosawa J, Shigemoto E, Yamane T et al: Electrocardiographic abnormalities and left ventricular asynergy in patients with acute cerebrovascular accidents. *Heart (Japan)* 1993; **25**: 406–414
15. Cachecho R, Grindlinger GA, Lee VW: The clinical significance of myocardial contusion. *J Trauma* 1992; **33**: 68–73
16. McLean RF, Devitt JH, McLellan BA, Dubbin J, Ehrlich LE, Dirksen D: Significance of myocardial contusion following blunt chest trauma. *J Trauma* 1992; **33**: 240–243
17. Gaspard P, Clermont A, Villard J, Amiel M: Non-iatrogenic trauma of the coronary arteries and myocardium: contribution of angiography - Report of six cases and literature review. *Cardiovasc Intervent Radiol* 1983; **6**: 20–29
18. Akiyama H, Masuda T, Kurosawa T, Kashiwaya Y, Tsuchiya N, Ohwada T: Coronary artery dissection accompanied by acute myocardial infarction and myocardial contusion secondary to blunt chest trauma. *JJAAM* 1993; **4**: 643–647
19. Parmley LF, Manion WC, Mattingly TW: Non-penetrating traumatic injury of the heart. *Circulation* 1958; **18**: 371–396
20. McCarthy MC, Pavlina PM, Evans DK, Broadie TA, Park HM, Schauwecker DS: The value of SPECT-Thallium scanning in screening for myocardial contusion. *Cardiovasc Intervent Radiol* 1991; **14**: 238–240
21. Hendel RC, Cohn S, Aurigemma G, Whitfield S, Dahlberg S, Pape L et al: Focal myocardial injury following blunt chest trauma: a comparison of indium-111 antimony scintigraphy with other noninvasive methods. *Am Heart J* 1992; **123**: 1208–1215