Annals of Nuclear Medicine Vol. 16, No. 8, 557–561, 2002

High-tension electrical injury to the heart as assessed by radionuclide imaging

Hitoshi IINO,* Taishiro CHIKAMORI,* Tsuguhisa HATANO,* Takayuki MORISHIMA,*
Satoshi HIDA,* Hidefumi YANAGISAWA,* Yasuhiro USUI,*
Tatsuya KAMADA,* Katsueki WATANABE** and Akira YAMASHINA*

*Department of Internal Medicine II, Tokyo Medical University
**Department of Plastic Surgery, Tokyo Medical University

To evaluate cardiac complications associated with electrical injury, 7 patients with high-tension electrical injury (6,600 V alternating current) underwent ^{201}Tl and ^{123}I-metaiodobenzylguanidine (MIBG) imaging in addition to conventional electrocardiographic and echocardiographic assessments. Electrocardiography showed transient atrial fibrillation, second degree atrioventricular block, ST-segment depression, and sinus bradycardia in each patient. Echocardiography showed mild hypokinesis of the anterior wall in only 2 patients, but ^{201}Tl and ^{123}I-MIBG myocardial scintigraphy showed an abnormal scan image in 6/7 and 5/6 patients, respectively. Decreased radionuclide accumulation was seen primarily in areas extending from the anterior wall to the septum. Decreased radionuclide accumulation was smaller in extent and milder in degree in ^{123}I-MIBG than in ^{201}Tl imaging. These results suggest that even in patients without definite evidence of severe cardiac complications in conventional examinations, radionuclide imaging detects significant damage due to high-tension electrical injury, in which sympathetic nerve dysfunction might be milder than myocardial cell damage.

Key words: electrical injury, cardiac complication, thallium myocardial imaging, ^{123}I-metaiodobenzylguanidine

INTRODUCTION

HIGH-TENSION ELECTRICAL INJURY can involve almost any organ.[1,2] Although the prognosis of electrical injury depends on cardiac damage, the majority of previous reports relied on conventional tests such as an electrocardiogram and measurement of cardiac enzymes. Recent development of nuclear cardiology facilitates the evaluation of not only myocardial necrosis but also cardiac sympathetic nervous activity with ^{123}I-metaiodobenzylguanidine (MIBG).[3–5] The purpose of the present study was to evaluate cardiac complications associated with high-tension electrical injury (6,600 V alternating current) with ^{201}Tl and ^{123}I-MIBG myocardial imaging since myocardial cell necrosis and abnormal cardiac sympathetic nerve activity are reported in the association with cardiac damage in such patients.[6,7]

Received May 16, 2002, revision accepted September 11, 2002.

For reprint contact: Hitoshi Iino, M.D., Department of Cardiology, Tokyo Medical University Kasumigaura Hospital, 3–20–1 Ami-machi Chyuou, Inashiki-gun, Ibaraki 300–0395, JAPAN.

E-mail: iino@tokyo-med.ac.jp

MATERIALS AND METHODS

Study patients: The subjects were 7 men, aged 20 to 46 years (mean 31), who were admitted to the Tokyo Medical University Hospital because of high-tension electrical injury (AC, 6,600 V). All the patients were admitted to the hospital immediately after the electrical injury. One patient who died from multiple organ failure after electrical injury during this study period was not included in the study. In addition to established management for patients with electrical injury, serum creatine kinase (CK) and CK-MB were measured every 8 hours for

Table 1 Summary of myocardial damage due to high-tension electrical injury in 7 patients

Case	Age	ECG	CK-MB max (% to CK)	Wall motion	^{201}Tl defects	^{123}I-MIBG defects	Size of defects	CAG
1	34	AF	28 U/*l* (8%)	normal	AS	AS	^{201}Tl > ^{123}I-MIBG	normal
2	46	normal	9 U/*l* (11.1%)	normal	Ap	normal	^{201}Tl > ^{123}I-MIBG	—
3	44	AV block	2088 U/*l* (8%)	normal	Sep	—	—	normal
4	26	normal	11 U/*l* (10.8%)	Ant hypokinesis	AS	AS	^{201}Tl = ^{123}I-MIBG	normal
5	20	normal	12 U/*l* (9.9%)	normal	Ap, AL Ant, Inf, Lat	Ap, Ant Inf, Lat	^{201}Tl > ^{123}I-MIBG	—
6	24	ST ↓	15 U/*l* (7.7%)	AS hypokinesis	Ap, AL AS, Inf	Ap, AL AS, Inf	^{201}Tl = ^{123}I-MIBG	normal
7	23	normal	19 U/*l* (2.3%)	normal	normal	normal	^{201}Tl = ^{123}I-MIBG	—

AF = atrial fibrillation; AL = antero-lateral, Ant = anterior; Ap = apical; AS = antero-septal; AV = atrioventricular; CAG = coronary angiogram; CK = creatine kinase; ECG = electrocardiogram; ^{123}I-MIBG = iodine-123 metaiodobenzylguanidine; Inf = inferior; Lat = lateral; Sep=septal; ST ↓ = ST depression; ^{201}Tl = thallium-201

24 hours after the admission. In patients with a high serum CK level when first tested, CK was monitored at 8 hour intervals for another 24 hours and then daily until the serum CK level normalized. The normal serum CK and CK-MB values for men were 34 to 152 U/*l* and 0 to 23 U/*l* (0–6.8%), respectively in our laboratory. Written informed consent was obtained from the all patients for detailed assessment of cardiac complications of high-tension electrical injury.

Echocardiography: An echocardiographic study was performed by a specified echocardiographer with an HP Sonos 1000 instrument (Hewlett-Packard; Stanford, California) and a 3.5-mHz transducer. Complete M-mode, 2-dimensional and Doppler echocardiography were recorded on videotape for subsequent analysis. Measurements of chamber dimensions and cardiac function were made from the M-mode echocardiogram as described previously.[8]

Radionuclide imaging of the heart: Within 40 days after injury, all patients underwent ^{201}Tl myocardial imaging to assess the extent and severity of myocardial cell damage, as well as ^{123}I-MIBG myocardial imaging to assess sympathetic nerve damage, except for one patient on whom MIBG myocardial imaging was performed 8 months after the injury. Without any stress testing, 111 MBq of ^{201}Tl chloride was injected intravenously at rest, and image acquisition was started 10 minutes after the injection. On a separate day, 111 MBq of ^{123}I-MIBG was injected at rest, and image acquisition was started 15 minutes later. An identical delayed image was acquired 4 hours later.

A digital gamma camera equipped with a low-energy high-resolution parallel multi-hole collimator (Prism 2000 XP, Picker; Cleveland, Ohio) was rotated over a 180° arc. SPECT image processing was conducted on an image data processor (Odyssey VP, Picker) with a Butterworth filter (with a cutoff value of 0.25 and an order of 8) and a Ramp filter. Scintigraphic findings were visually assessed by 3 cardiologists, and images were interpreted as normal or abnormal on visual inspection of the short axis, vertical long-axis, and horizontal long-axis views. Disagreement was resolved by consensus.

Cardiac catheterization: Left-sided cardiac catheterization was performed in 4 patients who had significant wall motion abnormality and/or severe myocardial perfusion defects as assessed by noninvasive tests. Patients underwent coronary angiography by Sones' technique with multiple projections, and left ventriculograms were obtained with biplane views. The ejection fraction was calculated from the right anterior oblique projection by the area-length method.

RESULTS

General findings of electrical injury: All patients met with the accident during work near 6,600 V AC high-voltage electrical wires. None of these patients had a previous history of cardiac disease before the injury. Although 1 patient had lost consciousness at the time of injury, no patient experienced cardiac arrest or ventricular fibrillation that necessitated cardiopulmonary resuscitation. Entry of the electrical current was from the right hand in 5 patients and from the left hand in 2 patients. Exit of the current was through the left hand in 2 patients, left upper extremity in 1, neck in 1 and no exit of the current was identified in the remaining 3 patients. The extent of skin damage due to the electrical injury was limited in every case, 1 patient with 5% of the total body surface area, and the remaining patients with 1% or less. No definite evidence of severe organ damage was observed in the brain, lungs, liver, intestines or kidneys in any of the patients.

Conventional assessment of the heart (Table 1): Electrocardiography showed transient atrial fibrillation, second degree atrioventricular block (Wenckebach type), ST-segment depression, and sinus bradycardia in each

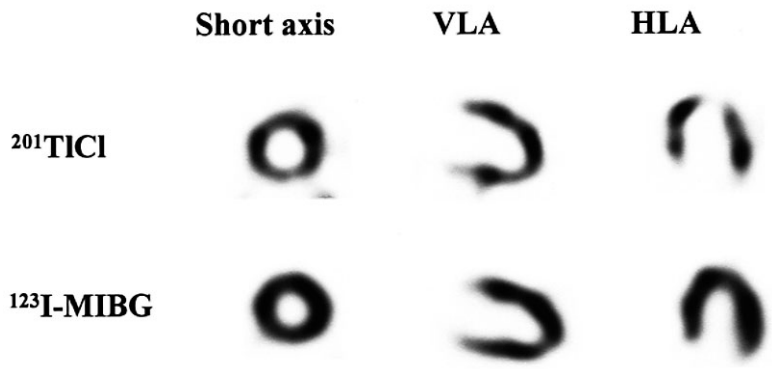

Fig. 1 ^{201}TlCl and ^{123}I-MIBG myocardial imagings of Case 2. Thallium perfusion defect was found in the apex of the horizontal long axis view, whereas ^{123}I-MIBG showed normal image. HLA = horizontal long axis; ^{123}I-MIBG = iodine-123 metaiodobenzylguanidine; ^{201}TlCl = thallium-201; VLA = vertical long axis.

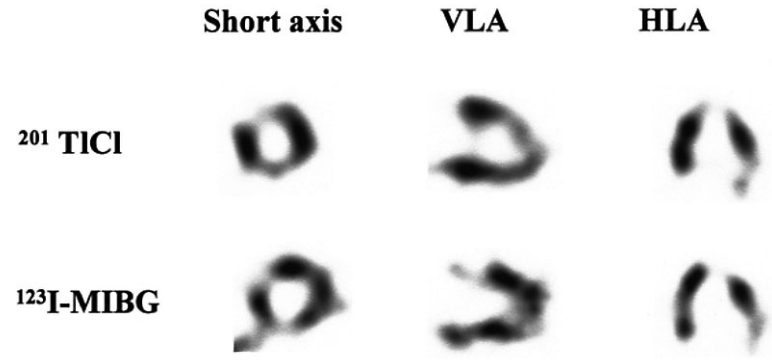

Fig. 2 ^{201}TlCl and ^{123}I-MIBG myocardial imagings of Case 5. Thallium perfusion defects were observed in the anterior and inferior segments in the short axis view, in the apical and anterolateral segments in the vertical long axis view, and in the apical and lateral segments in the horizontal long axis view. Decreased ^{123}I-MIBG accumulations were observed in the anterior segments and inferior segments in the short axis view, in the apical segment in the vertical long axis view, and apical and lateral segments in the horizontal long axis view. However, the extent of most perfusion abnormalities was less in ^{123}I-MIBG compared with ^{201}Tl imaging. In addition, a localized area with decreased ^{123}I-MIBG accumulations were detected in the septum in the horizontal long axis view. HLA = horizontal long axis; ^{123}I-MIBG = iodine-123 metaiodobenzylguanidine; ^{201}TlCl = thallium-201; VLA = vertical long axis.

patient (Cases 1, 3, 6 and 7). All these abnormal findings in electrocardiography, however, were sustained for only 1 to 3 days. In the remaining 3 patients, the electrocardiogram was normal. Serum CK-MB levels increased in only 2 of the 7 patients: 28 and 2,088 U/*l*, respectively (Cases 1 and 3). Echocardiography revealed normal left ventricular wall motion in most patients except for 2 patients who had mild hypokinesis of the anterior or anteroseptal wall (Cases 4 and 6). Coronary angiogram demonstrated normal coronary arteries in these 2 patients. Left ventricular ejection fraction as assessed by echocardiography and/or left ventriculogram was ≥60% in all patients.

Myocardial perfusion imaging (Table 1): ^{201}Tl and ^{123}I-MIBG myocardial scintigraphy revealed abnormal scan images in 6/7 and 5/6 patients, respectively. Perfusion defects and/or decreased radionuclide accumulation were seen primarily in areas extending from the anterior wall to the septum. The amount of decreased accumulation was smaller in extent and milder in degree in ^{123}I-MIBG than in ^{201}Tl imaging. Myocardial images obtained with ^{201}Tl and ^{123}I-MIBG in case 2 are shown in Figure 1. A thallium perfusion defect was found in the apex of the horizontal long axis view, whereas ^{123}I-MIBG showed a normal myocardial image. In case 5, large perfusion defects were observed in the anterior and inferior segments in the short axis view, in the apical and anterolateral segments in the vertical long axis view, and in the apical and lateral segments in the horizontal long axis view (Fig. 2). Decreased ^{123}I-MIBG accumulations were also observed in the anterior and inferior segments in the short axis view, in the apical segment in the vertical long axis view, and apical and lateral segments in the horizontal

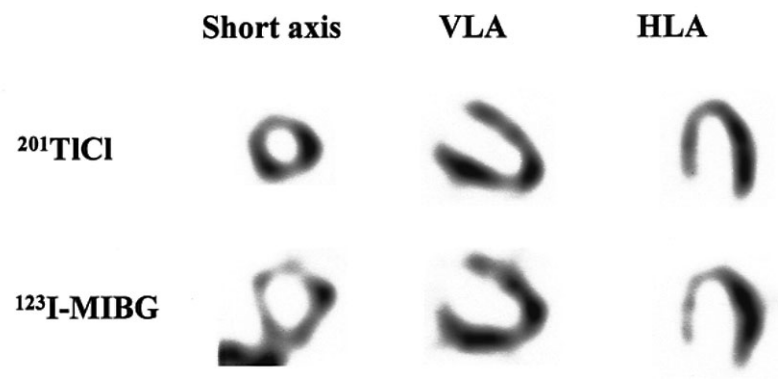

Fig. 3 ^{201}TlCl and ^{123}I-MIBG myocardial imagings of Case 6. Thallium perfusion defects were observed in the anteroseptal and inferior segments in the short axis view, in the apical and anterolateral segments in the vertical long axis view, and in the apical and septal segments in the horizontal long axis view. Although extensive areas with decreased ^{123}I-MIBG accumulations were also observed in this patient, the extent and severity in these perfusion abnormalities were similar. HLA = horizontal long axis; ^{123}I-MIBG = iodine-123 metaiodobenzylguanidine; ^{201}TlCl = thallium-201; VLA = vertical long axis.

long axis view, but the extent of most perfusion abnormalities was less in ^{123}I-MIBG than in ^{201}Tl imaging. In addition, a localized area with decreased ^{123}I-MIBG accumulation was detected in the septum in the horizontal long axis view. In case 6, extensive perfusion defects were observed in the anteroseptal and inferior segments in the short axis view, in the apical and anterolateral segments in the vertical long axis view, and in the apical and septal segments in the horizontal long axis view (Fig. 3). Although extensive areas with decreased ^{123}I-MIBG accumulations were also observed in this patient, the extent and severity in these perfusion abnormalities were similar. A coronary angiogram of this patient showed normal coronary arteries.

DISCUSSION

Tachyarrhythmias, conduction disturbances and left ventricular wall motion abnormality are reported as common cardiac complications in electrical injury.[10,11] The clinical picture in case reports often showed significant enzymatic, electrocardiographic and echocardiographic abnormalities that resembled myocardial infarction.[12–15] The present study demonstrated that the incidence of electrocardiographic abnormalities, enzymatic increase and left ventricular wall motion abnormality were rather uncommon and mild in 7 patients who survived high-tension electrical injury of 6,600 V AC, but multiple perfusion defects and/or decreased accumulation of radioisotopes were observed in the majority of patients with both ^{201}Tl and ^{123}I-MIBG myocardial imagings. The cardiac damage due to electrical injury in these patients may be less severe than reported in the literature,[10,12–14] since no patient in this study sustained cardiac arrest or ventricular fibrillation that necessitated cardiopulmonary resuscitation. In contrast to previous reports, the present study indicates that even in patients without definite evidence of severe cardiac abnormality in the conventional test, radionuclide myocardial imaging detects significant myocardial damage due to electrical injury.

Myocardial perfusion defects and/or decreased accumulation of radioisotopes were seen primarily in areas extending from the anterior wall to the septum with either ^{201}Tl or ^{123}I-MIBG myocardial imagings. Myocardial infarction was not considered to be the cause of these perfusion defects. In most patients with abnormal radionuclide imaging, the maximal serum CK level of cardiac origin was within the normal range. Moreover, no obstructive coronary artery lesion was observed on the coronary angiogram in these patients who underwent invasive examination because of left ventricular wall motion abnormality and/or extensive ^{201}Tl perfusion defects. Instead, the etiology of myocardial damage in these patients may be due to thermal injury due to high-tension electrical current. The entry of the electrical current was from the hand in all of the patients, and the obvious exit was through the frontal surface of the body in the majority of the patients. Therefore, the high-tension electrical current might pass through the anterior part of the heart and leave myocardial damage due to thermal injury as was observed with myocardial imaging.

Several reports suggested the etiology of cardiac damage due to electrical injury as coronary vasospasm because an electrogenic membrane mechanism of human coronary smooth muscle activation may exist.[6,15,16] In addition, Xenopoulos et al. reported an autopsy case with myocardial necrosis in the absence of coronary thrombus or significant atherosclerosis.[6] To test this hypothesis, ^{123}I-MIBG was selected as a radionuclide to evaluate those patients surviving high-tension electrical injury in this study because abnormal ^{123}I-MIBG findings were frequently observed in patients with vasospastic angina

because of abnormal cardiac sympathetic nervous activity.[17–20] If coronary spasm induced by electrical injury is the primary etiology of myocardial damage, any abnormal finding in ^{123}I-MIBG myocardial imaging that is related to abnormal sympathetic nervous activity should be remarkable. In the present study, however, a scintigraphic study revealed that the amount of decreased radionuclide accumulation was smaller in extent and milder in degree with ^{123}I-MIBG than with ^{201}Tl myocardial imaging in the majority of the patients. Moreover, the locations of decreased accumulation of ^{123}I-MIBG were almost identical to the areas of ^{201}Tl perfusion defects in most patients. Therefore, the cause of abnormal finding in ^{123}I-MIBG myocardial imaging due to high-tension electrical current may be the same as that in ^{201}Tl myocardial imaging, and was most likely to be related to thermal injury that resulted in myocardial denervation. Nevertheless, the potential role of increased coronary tonus cannot be ruled out in this study since the extent of perfusion abnormalities was larger with ^{123}I-MIBG than with ^{201}Tl in a few patients.

Many factors such as voltage, tissue resistance, tissue susceptibility, type of current, current pathway, site and conduction of electrical contact determine the severity of electrical injury to the heart.[16] Previous reports and our observations indicate a wide spectrum of cardiac damage, from ventricular fibrillation to normal ventricular function without any electrocardiographic abnormality. Even in the latter case, as was observed in the present study, radionuclide imaging is a sensitive method for detecting cardiac damage caused by electrical injury and may be useful for long-term follow-up of patients surviving electrical injury.

REFERENCES

1. DiVincenti FC, Moncrief JA, Pruitt BA Jr. Electrical injuries: a review of 65 cases. *J Trauma* 1969; 9: 497–507.
2. Hammond JS, Ward CG. High-voltage electrical injuries: management and outcome of 60 cases. *South Med J* 1988; 81: 1351–1352.
3. 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.
4. Sisson JC, Shapiro B, Meyers L, Mallette S, Mangner TJ, Wieland DM, et al. Metaiodobenzylguanidine to map scintigraphically the adrenergic nervous system in man. *J Nucl Med* 1987; 28: 1625–1636.
5. Sisson JC, Lynch JJ, Johnson J, Jaques S Jr, Wu D, Bolgos G, et al. Scintigraphic detection of regional disruption of adrenergic neurons in the heart. *Am Heart J* 1988; 116 (1 Pt 1): 67–76.
6. Xenopoulos N, Movahed A, Hudson P, Reeves WC. Myocardial injury in electrocution. *Am Heart J* 1991; 122: 1481–1484.
7. Lewin RF, Arditti A, Sclarovsky S. Non-invasive evaluation of electrical cardiac injury. *Br Heart J* 1983; 49: 190–192.
8. Feigenbaum H. Echocardiographic measurements and normal values. In: Feigenbaum H, ed. *Echocardiography*, 5th ed. Philadelphia; Lea & Febiger, 1994: 658–683.
9. Sandler MP. The expanding role of MIBG in clinical medicine. *J Nucl Med* 1988; 29: 1457–1459.
10. Housinger TA, Green L, Shahangian S, Saffle JR, Warden GD. A prospective study of myocardial damage in electrical injuries. *J Trauma* 1985; 25: 122–124.
11. Jensen PJ, Thomsen PE, Bagger JP, Norgaard A, Baandrup U. Electrical injury causing ventricular arrhythmias. *Br Heart J* 1987; 57: 279–283.
12. Lichtenberg R, Dries D, Ward K, Marshall W, Scanlon P. Cardiovascular effects of lightning strikes. *J Am Coll Cardiol* 1993; 21: 531–536.
13. Arrowsmith J, Usgaocar RP, Dickson WA. Electrical injury and the frequency of cardiac complications. *Burns* 1997; 23: 576–578.
14. Carleton SC. Cardiac problems associated with electrical injury. *Cardiol Clin* 1995; 13: 263–266.
15. Ku CS, Lin SL, Hsu TL, Wang SP, Chang MS. Myocardial damage associated with electrical injury. *Am Heart J* 1989; 118: 621–624.
16. Robinson NM, Chamberlain DA. Electrical injury to the heart may cause long-term damage to conducting tissue: a hypothesis and review of the literature. *Int J Cardiol* 1996; 53: 273–277.
17. Sakata K, Miura F, Sugino H, Saegusa T, Shirotani M, Yoshida H, et al. Assessment of regional sympathetic nerve activity in vasospastic angina: analysis of iodine 123-labeled metaiodobenzylguanidine scintigraphy. *Am Heart J* 1997; 133: 484–489.
18. Inobe Y, Kugiyama K, Miyagi H, Ohgushi M, Tomiguchi S, Takahashi M, et al. Long-lasting abnormalities in cardiac sympathetic nervous system in patients with coronary spastic angina: quantitative analysis with iodine 123 metaiodobenzylguanidine myocardial scintigraphy. *Am Heart J* 1997; 134: 112–118.
19. Sakata K, Shirotani M, Yoshida H, Kurata C. Iodine-123 metaiodobenzylguanidine cardiac imaging to identify and localize vasospastic angina without significant coronary artery narrowing. *J Am Coll Cardiol* 1997; 30: 370–376.
20. Taki J, Yasuhara S, Takamatsu T, Nakajima K, Tatami R, Ishise S, et al. Value of iodine-123 metaiodobenzylguanidine scintigraphy in patients with vasospastic angina. *Eur J Nucl Med* 1998; 25: 229–234.