Case reports

Constitutional predisposition to vasovagal syncope: an additional risk factor in patients exposed to electrical injuries?

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SUMMARY An hour after a 220 V electric shock a patient who was susceptible to mild vasovagal symptoms in response to emotional stress had a severe episode of cardiac arrest in response to insertion of a cannula. No myocardial damage or conduction abnormalities were detected by serial 12 lead electrocardiograms.

Patients with a history of vasovagal reactions may be at high risk of developing lethal sinus node or conduction disturbances after electrical injuries. Psychological stresses should be avoided in the management of such patients.

Cardiac arrhythmias and conduction disturbances are among the most serious clinical manifestations of electrical accidents.¹ After low voltage injuries (<350 V) arrhythmias and conduction disturbances have been found in 11–39% of the patients.² Naturally it is only in fatal cases of high voltage and lightning injuries that the morbid anatomy has been described.^{1,3}

The potential lethal effect of autonomic reactions when there is electrical instability in the heart has been described in patients with ischaemic heart disease and in animal experiments.^{4,5} We report a patient who fainted soon after a 220 V electric shock. About an hour later sinus arrest developed seconds after the insertion of an intravenous cannula.

Case report

A 33 year old electrician was admitted to hospital after a 220 V AC electric shock. When the shock occurred he was fully conscious and locked on to the

wires because of the tetanising effect of the alternating current; he was pulled away by his colleagues. The current, which had passed up one arm and down the other, had caused electrothermal burns on both hands. About 30 seconds after the accident the patient lost consciousness for 10–15 seconds. The pulse was not registered during this event. He regained consciousness spontaneously. At admission to hospital his pulse rate was 80–88 beats/minute, blood pressure was 150/90 mm Hg, and a 12 lead electrocardiogram showed no signs of myocardial injury. A stable sinus rhythm and normal PQ, QRS, and QT intervals were found. The patient's electrocardiogram was monitored continuously. Before the insertion of an intravenous cannula he stated that he was always upset by the sight of blood. He reported fainting after an intramuscular injection and he said that on several occasions he had turned pale and felt dizzy and nauseated at the sight of blood or cannulas. On this occasion as the cannula penetrated his skin (about an hour after the electric shock), sinus bradycardia developed and this led to sinus arrest within 5–10 seconds. The patient lost consciousness, his pupils dilated, and he stopped breathing. Asystole lasted 33 seconds and was interrupted by a

ventricular escape at 17 seconds and a nodal escape at 29 seconds. After 33 seconds a spontaneous sinus impulse was seen but another sinus arrest followed. Electrical cardiac activity then was restored by a blow to the chest and external cardiac compression. During the next three days no arrhythmias or cardiac conduction disturbances were recorded and serial electrocardiograms did not disclose structural damage to the heart. Carotid sinus stimulation was performed two days after the accident and did not provoke bradycardia or conduction disturbances. The patient left hospital in an excellent physical condition after an observation period of three days.

Discussion

Many reports have indicated a relation between psychological stress and sudden death. Engel reviewed 275 patients who died suddenly; 21% died immediately after hearing of the collapse or death of a close relative and another 9% died when such a loss appeared to be a possibility.⁶ Twenty per cent died during the first three weeks of acute grief and 3% during mourning or upon anniversary of a death. A similar link between emotional state and sudden death from coronary artery disease was found in another study.⁷

Cardiac arrest after vasovagal reactions to emotional stimuli in otherwise healthy individuals have only rarely been described.^{8,9} It is known, however, that strong vagal stimulation in healthy people may induce sinus arrest for 4 to 10 seconds.¹⁰ This prompts the question "Was the sinus arrest caused exclusively by a strong vasovagal reaction or was the cardiac dysfunction induced by the electric shock a contributing factor?"

Neither the history nor the patient's subsequent progress suggested that he had ischaemic heart disease; and contemporary and serial electrocardiograms showed no evidence of structural damage to the myocardium by the electric shock. Minor electrophysiological disturbances in intracardiac electrical conduction and the impulse generating system, however, may not induce recognisable abnormalities in a standard electrocardiogram. Furthermore, the pathophysiological basis of arrhyth-

mias that occur after low voltage electrical accidents without myocardial damage is unknown. Finally, the patient had never before shown such severe vasovagal reaction in response to the insertion of a cannula, though he reported milder vasovagal responses to similar stimuli. For these reasons we believe that the electric shock had enhanced the patient's reaction to such stimuli and to such an extent that was potentially lethal.

Patients with a history of vasovagal reactions may be at increased risk of the development of lethal cardiac conduction disturbances after electrical injuries. It seems reasonable to advise medical and nursing staff to avoid interventions that cause psychological stress in such patients.

References

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