Apprentice electrician with electrical injury

EDUCATIONAL CASE REPORT

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A low-voltage electric shock can cause clinical effects of different types and severities, including cardiac arrhythmias and internal burns. The likelihood of injury typically depends on the current, the voltage, the route taken by the current through the body, and the duration of current

exposure. We describe a patient in whom an electrical accident presumed unlikely to cause injury had more severe clinical consequences than expected.

An apprentice electrician in his twenties suffered an electric shock while installing an electrical socket in a junction box in the roofspace above a ceiling. He was working on top of a stepladder, with his arms raised above his head, and his feet approximately 180 cm above the ground. The tips of both thumbs touched live contacts behind the electrical socket and current flowed from one hand to the other, probably via the chest area. The voltage was 230 V according to the electrical grid at his workplace. He did not fall, but probably released contact immediately as he did not have a closed grip on the socket. He perceived the electric shock as lasting only a brief moment. Afterwards, he was dazed and needed a few minutes to recover before he reported the incident to a colleague, who drove him to the Accident and Emergency department.

There are estimated to be around 3 000 potentially serious electrical accidents among electricians in Norway each year, based on subjective assessment of injury potential (1). The incidence appears to be similar in Sweden and Denmark (2). Most of these accidents do not result in contact with the healthcare services, but electricians are aware of the possible dangers of this type of exposure. Recommendations have therefore been prepared regarding when and how persons exposed to electric current should be followed up by the healthcare services in the acute phase (3).

Electrical accidents may have serious acute effects, in particular on the heart, nervous system, skin and musculoskeletal system (3–5). When low-voltage current flows from arm to arm, the key potential complications in the acute phase are disruption of cardiac activity, deep muscle injury owing to tissue heating (internal burns), nerve damage, injuries from falls and complete/partial tearing of muscle insertions (3, 6). In the event of more severe muscle injury – rhabdomyolysis – large amounts of myoglobin and electrolytes are released from the damaged muscle, which may lead to renal failure. Exposure to low-voltage current is generally assumed to pose little risk of muscle injury due to tissue heating, provided that the individual does not remain connected to the current source as a result of muscle spasms. High-voltage accidents, which are more likely to be life-threatening, can have significantly more severe effects, including burns and rhabdomyolysis.

In the Accident and Emergency department, an electrocardiogram (ECG) showed normal sinus rhythm without arrhythmias. The patient felt a little unwell and had to sit down for a while. He had discomfort in his right hand, radiating to the throat, but did not report skin alterations, numbness in the hand, or palpitations. A little while later, it was considered safe to send the patient home as he had a normal ECG and no longer felt unwell. However, it also emerged that the patient had previously suffered episodes of palpitations, which upon investigation had been attributed to nodal reentry tachycardia. Four years prior to the accident, he had undergone a successful ablation procedure.

Given the patient's previous medical history, the Accident and Emergency doctor conferred with the on-call doctor in the hospital's department of internal medicine, and the patient was admitted to the intensive care unit for observation, with monitoring throughout his entire inpatient stay.

Blood samples taken 2 hours 45 minutes after the accident showed elevated total serum creatine kinase (CK) of 37 669 U/l (reference range 50–400) and serum ALT of 201 U/l (10–70) (Table 1). Other tests were within reference ranges ([7]). The results of blood tests performed by the patient's general practitioner before the accident were also available, and showed normal total CK.

Table 1

Selected blood test results prior to the accident and in the acute phase. Other tests were performed and were either within reference ranges both before and after the accident (blood: leukocytes, haemoglobin, platelets; serum: albumin, alkaline phosphatase, sodium), or within reference ranges after the accident but not measured beforehand (serum: CRP, total bilirubin, GT, pancreatic amylase; plasma: chloride).

	Time point		12:45	15:30	21:30	08:00	08:00	
			0 hr	2 hr 45 min	8 hr 45 min	19 hr 15 min	43 hr 15 min	
		1 month before accident	Accident	1st day		2nd day	3rd day	10th day
Analyses	Reference range							
Total serum CK (U/l)	50–400[1,2]	109		37 669*	26 351*	19 217*	9 660*	238
Serum troponin T (ng/l)	< 14			< 10				
Serum creatinine (µmol/l)	60–100	99		87	83	80		
Serum urea (mmol/l)	3.2–8.1[1,2]	4.8		5.7		2.8*		
Serum AST (U/l)	15–45[1]				420*			
Serum ALT (U/l)	10–70[1]			201*		149*		
Plasma potassium (mmol/l)	3.5–4.4			4.0	3.3*	3.9		
Serum calcium, albumin corrected (mmol/l)	2.17–2.47	2.31		2.32	2.07*			

*

Outside of reference range

[1]Reference range for men

[2]Reference range for age group 18–49 years

The patient had an indwelling catheter for the first 24 hours. Urine dipstick testing was performed three times on the day of the accident, with all tests negative for glucose, ketones, leukocytes, albumin and blood. Dark urine was not seen at any time, and urine microscopy was not performed.

An increase in total serum CK reflects damage to muscle tissue and may occur in a number of different circumstances, including after muscle spasms; physical trauma or injury to muscles after strenuous exercise; muscle ischaemia, for example after CO poisoning, and in primary neuromuscular diseases such as Duchenne muscular dystrophy. Viral and bacterial infections can both lead to elevated total serum CK, as can various medications, such as neuroleptics (neuroleptic malignant syndrome), cyclic antidepressants and antihistamines. Toxic substances such as alcohol, barbiturates or amphetamines can also have the same effect (8). The anticipated CK increase varies depending on the cause (9).

A mild to moderate increase in ALT levels can be seen in a variety of conditions, particularly those affecting the liver, but also upon damage to skeletal muscle. In the latter case, the increase is always coincident with increased serum AST and total serum CK (7, 10).

Myoglobinuria can be part of the clinical picture in rhabdomyolysis; however, in certain datasets myoglobinuria has been reported in less than one-fifth of cases (11). It is therefore an unreliable measure that is dependent on very rapid release of myoglobin in injured muscles. Serum myoglobin normalises within 24 hours (8), and the presence of myoglobin in the urine depends on urine flow, the person's body weight, and not least on the time since the injury; moreover, it only occurs if renal capacity is exceeded (8, 12).

The patient's test results could be consistent with muscle damage (rhabdomyolysis) despite the short duration of the electric shock. In order to prevent kidney damage, he was therefore admitted and treated with forced alkaline diuresis. A total volume of 6 250 ml saline was administered in the first 24 hours, with diuresis of 3 190 ml measured between 6 pm on the day of admission and 6 am the following morning. Six hours after treatment initiation, total serum CK was 26 351 U/l. After 24 hours, it was 18 338 U/l. Serum AST was 420 U/l and still elevated, while plasma potassium and serum calcium were fairly low nine hours after the accident, i.e. during treatment with forced diuresis.
Other measures (blood: leukocytes, haemoglobin, platelets; serum: albumin, creatinine, alkaline phosphatase, sodium) were within reference ranges both before and after the accident.
Upon discharge two days after admission, he appeared clinically normal. All symptoms (discomfort in the right hand radiating to the throat) had resolved completely, but he still had elevated total serum CK of 9 660 U/l (ref. 50–400)

and was informed of the importance of drinking plenty of fluids over the next few days. An outpatient check-up after ten days showed normal total serum CK (Table 1).

Discussion

This case report describes a patient who suffered an electrical accident, but whose good general condition upon initial assessment, along with the course of events in the accident and the presumed current exposure, could easily have resulted in him receiving insufficient testing and observation. Further testing revealed that there were indications for intervening to prevent renal damage as a result of muscle injury. Since test results prior to the accident were normal, and there was a seemingly obvious causal trigger, no further differential diagnoses were considered.

The patient did not describe either muscle spasms that could have kept him in contact with the circuit, or strong muscle contractions, and he did not fall from the stepladder on which he had been working. Such brief exposure to low-voltage current as seems to be the case here, is generally considered relatively unlikely to cause injury as a result of tissue heating. In Norway, electricians often colloquially refer to such exposures as 'candies'.

When assessing electrical accidents, it is important to describe the current flow in such a way as to allow exposure to subsequently be estimated. Keywords are current type, voltage level, probable route of the current through the body, and perceived or observed duration of the event. Any findings from examinations in the acute phase that could add to the patient's description of the exposure, such as spasms in the arms or legs, the surface and moisture level of the contact points (which affect the resistance), and skin alterations, should also be included (2).

The most common type of electrical accident involves exposure to low-voltage alternating current – 230 V or 400 V – with a frequency of 50 Hz. However, direct current is also used, especially in the workplace. Direct current and alternating current have somewhat different effects on the human body. The most important difference is that the 50 Hz frequency of alternating current can contribute to tetanic muscle contractions that may prevent a person who is holding onto a conductor from being able to let go. This will increase the duration of current exposure and thus the risk of tissue damage, including muscle injury. Tissues in the path of the current that have high resistance, such as bones and tendons, are particularly likely to undergo heating. This means that areas around joint and muscle attachments are vulnerable to injury (13). Exposure to direct current normally does not cause significant muscle spasms and is therefore typically of shorter duration than exposure to alternating current. In the case of lightning strikes or other high-voltage accidents, the type and duration of the exposure are of less significance given that large amounts of energy are transmitted regardless.

When assessing potential muscle damage, further tests should be conducted in the event of abnormally high total serum CK. CK is found primarily in skeletal muscle and myocardium, but also in the brain, intestines, kidney, thyroid and prostate. The creatine kinase enzyme consists of polypeptide chains that can be of two types: B and M. Total CK is a measure of three isoenzymes: CK-BB (brain type), CK-MB (cardiac muscle type – a marker for myocardial infarction) and CK-MM (skeletal muscle type). Of these isoenzymes, only CK-MB is measured routinely. The normal range for total serum CK in men aged 18–49 years is around 50–400 U/l, varying slightly between laboratories. A person's baseline level correlates with muscle mass, and muscle use can lead to significant increases in total serum CK, with even moderate muscle use increasing activity by 50 % (14).

Throughout the patient's hospitalisation, we observed highly elevated total serum CK levels. These may occur as a result of widespread damage to muscle tissue/rhabdomyolysis or severe myocardial infarction, for example. Measurement of total serum CK is therefore indicated as part of the workup and monitoring of muscle injuries, and when assessing the indication for treatment in cases of rhabdomyolysis (8, 12). The total CK serum concentration reflects to a large extent the amount of muscle tissue affected, and 10 000 U/l is sometimes used as a threshold for initiating forced diuresis (15, 16). In critically ill patients with widespread damage to muscle tissue, measurement of plasma myoglobin has greater sensitivity and specificity than measurement of serum CK for assessing the risk of acute renal failure (17). Plasma myoglobin was not quantified in this patient.

Knowing precisely when the exposure occurred makes it easier to interpret the total serum CK level. This is because total serum CK will be in the normal range shortly after muscle damage, before starting to increase after 2–12 hours (18). After about 24 hours, serum CK reaches its peak (8, 19), where it remains stable for up to 3 days (8) before gradually decreasing and returning to normal after 2–3 (19) or 4–5 days (8). However, extensive variation is seen, depending on the degree of hydration and on renal function. The relative timing of the injury and of sampling is therefore important as samples taken too soon after the injury will still be in the normal range, while levels may already have started to decrease after about two days. Our patient had an increase in total serum CK after 2 hours 45 minutes. Given the known variation, it may be worth taking multiple samples over the course of the first 12 hours post-injury to help ensure that any increase is detected, so that treatment can be initiated if required.

Documenting an increase in total serum CK could also prove important should the patient subsequently experience medical problems related to the arms, and there is reason to assess whether this is an occupational injury occurring in the wake of the previous accident.

In this case, the patient's previous medical history contributed to further testing, which in turn led to admission and treatment. The initial blood test was therefore crucial in ensuring that the patient received the treatment he needed to achieve the best possible outcome after the accident.

Upon suspicion of acute myocardial injury, especially when current is likely to have travelled through the chest region or where there is chest pain following the accident, troponin T (or I) should be measured, possibly in addition to CK-MB. In this patient, the troponin level was within the reference range of < 14 ng/l (99th percentile) when measured four hours after the accident (< 10 ng/l). The results thus suggest that striated muscle was affected to a greater degree by the accident (given the elevated total serum CK) than cardiac muscle. This is further supported by the fact that the ECG did not provide evidence of arrhythmias. Thus, according to test results, the accident probably did not lead to an increased risk of cardiac complications.

The blood tests following the accident strongly suggest muscle damage, although this was not expected on the basis of the clinical findings. The exposure to current may thus have been more serious in reality than the initial description suggested. However, it may also be that the threshold for muscle damage is lower than assumed, with the result that such damage is often overlooked and undertreated. The initial finding of normal serum potassium and high total serum CK is surprising, and difficult to explain. Normally levels of these two substances track one another after cell damage (20). Rhabdomyolysis is a known complication of high-voltage accidents (8, 21), but we are not aware of it having been described following exposure to low-voltage current.

Recommendations have been published for inpatient examination of those with electrical injuries (3). We recommend that hospital emergency departments review these when developing internal procedures.

Conclusion

This case history shows that muscle damage may occur following more minor electrical accidents than had previously been assumed. Furthermore, decisions on admission and monitoring in such cases cannot be based solely on the risk of cardiac complications, as the current case is an example of skeletal muscle involvement without signs of cardiac involvement. Total CK in serum is an important marker for assessing muscle damage. It is crucial to consider the time of sampling in relation to the time of the accident, so that any damage can be detected as early as possible. In the event of normal findings within the first 5–6 hours after injury, sampling should be repeated because of the latency for CK increases, and in any case after 24 hours (8, 19). This will ensure that the maximum total serum CK level is detected and that any muscle damage is not overlooked.

The patient has consented to the publication of this article.

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