Scientific Report

Rare survival of high-tension electrocution shock in a crossbred Jersey cattle: a complete profile on critical care monitoring

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Abstract

Background: Accidental electrocution was more common in animals and death was mostly due to shock and cardiac arrest. Survival of animals or humans could be possible if victims receive immediate medical support. **Case description:** A 3-year-old crossbred Jersey heifer was presented to the Emergency and Critical Care Medicine Referral Clinic of the Veterinary College and Research Institute, Orathanadu, with a history of accidental electrocution by broken high-tension overhead power transmission line during grazing in the paddy fields. The animal was dull and depressed, dark red, and some areas were charred in appearance on the dorsum and limbs. The animal showed difficulty walking due to the electrocution burn injury and was poorly responding to the surroundings. Clinical examination revealed subnormal temperature, polypnea, pale mucous membranes, ruminal atony, and arrhythmias on auscultation. **Findings/treatment and outcome:** On point of care (PoC) hematology testing, leukocytosis, neutrophilia, and microcytosis were observed. PoC electrolyte analysis revealed hypocalcemia (ionized calcium 0.89 mmol/L), mild hypochloremia, and severe hypokalemia (2.81 mmol/L). PoC biochemistry revealed hypoglycemia (41 mg/dl). PoC elevated levels of serum cardiac troponin (0.33 ng/dl) indicated cardiac damage. Aspartate aminotransferase (1794 U/L), CK-MB (699 U/L) and LDH (6.7 U/L) were also elevated. On PoC urinalysis, proteinuria, myoglobinuria, and glucosuria were observed. Evident clinical recovery, wound healing, and improvement in animal activities were observed. **Conclusion:** High-voltage electrocution injury is a serious type of accident with the potential risk of multi-organ damage and death. Early diagnosis of electrocution and immediate management enhances the expectancy of complete recovery.

Key words: Cardiac troponin, Cattle, Electrocution, Shock

Introduction

Animals are more susceptible to accidental electrocution. They not only lack electrical insulation on their feet, which are often wet, but also have more contact with surface materials than humans (Mills and Kersting, 1966). The severity of the electrical shock is associated with high voltage and duration of exposure as well as lowered electrical resistance of the animal and pathway of the current through the body (Aiello, 1998). Depending upon the conditions of the electrical exposure, organ damage may be minor and transient, severe with multi-organ involvement over a lifetime, or lethal. Even the passage of small electric currents through the heart can cause fibrillation. Electrocution injuries lead to necrosis of superficial or deep tissue. The high-voltage current shock causes severe burn injuries, coagulation, and necrosis of affected areas (Tufani *et al*., 2015). Severe electric shock commonly causes unconsciousness, respiratory paralysis, muscle contractions, bone fractures, and cardiac disorders (Radostitis *et al*., 2000). Severely affected animals develop hypovolemic shock, leading to poor organ perfusion, hypoxia, initiation of anaerobic metabolism,

and alteration of circulatory metabolic derangement (Englehart and Schreiber, 2006). Analysis of serum markers including lactate, bicarbonate, and arterial pH is very essential to monitor and assess the severity and therapeutic efficacy of the patient and provide the prognosis (Hobbs *et al*., 2010).

Survival of animals and humans following an electric shock or lightning strike that results in cardiopulmonary arrest is possible if victims receive immediate medical support. However, not many reports are available on survival of electrocuted cattle. Herein, a case of accidental electrocution cattle is reported and its emergency management, critical care monitoring, and post treatment evaluations are also presented and discussed.

Case description

A 3-year-old crossbred Jersey heifer was presented to the Emergency and Critical Care Medicine Referral Clinic of the Veterinary College and Research Institute, Orathanadu, India, with a history of accidental electrocution by broken high-tension overhead power transmission line during grazing in the paddy fields. On presentation, the animal was non-ambulating, dull, depressed, and recumbent for several hours, and had poor response to the surroundings (Fig. 1A). The electrocuted areas became dark red and some areas were charred in appearance, due to the electric burn injuries. Thoracic auscultation revealed cardiac arrhythmia and abnormal respiration. The blood gases (BG) of arterial blood were analyzed (Fig. 1B), which is an important tool in evaluating lung dysfunctions and a basis for respiratory function diagnosis.

Clinical examination revealed subnormal temperature, polypnea, pale mucus membrane, and ruminal atony. The extremities were cold and the animal was poorly responding to stimuli. As these clinical changes and history confirmed that the cow underwent high-tension electrocution.

Clinical samples were taken for point of care (PoC) analysis. The whole blood, serum, and arterial blood were collected for laboratory diagnosis. PoC hematology revealed leukocytosis, neutrophilia, and microcytosis (Table 1). cTnI was assessed in serum samples using the PoC immunoassay i-STAT analyzer (Abbott Healthcare Pvt. Ltd.). Arterial blood collected was from the coccygeal artery and analyzed immediately by i-STAT analyzer.

Serum biochemistry revealed hypoglycemia (41 mg/dl), the elevation of aspartate aminotransferase (1794 U/L), CK-MB (699 U/L), and LDH (6.7 U/L) (Table 2). PoC cardiac troponin assay revealed elevated levels of serum cardiac troponin (0.33 ng/dl), indicating cardiac damage. Arterial blood gas analysis showed significant variation in lactate and bicarbonate (Table 3). On PoC urinalysis, proteinuria (300 mg/dl), myoglobinuria, and glucosuria (500 mg/dl) with a specific gravity of 1.005 were observed. All these changes contributed to multisystemic damage in the cow. PoC electrolyte

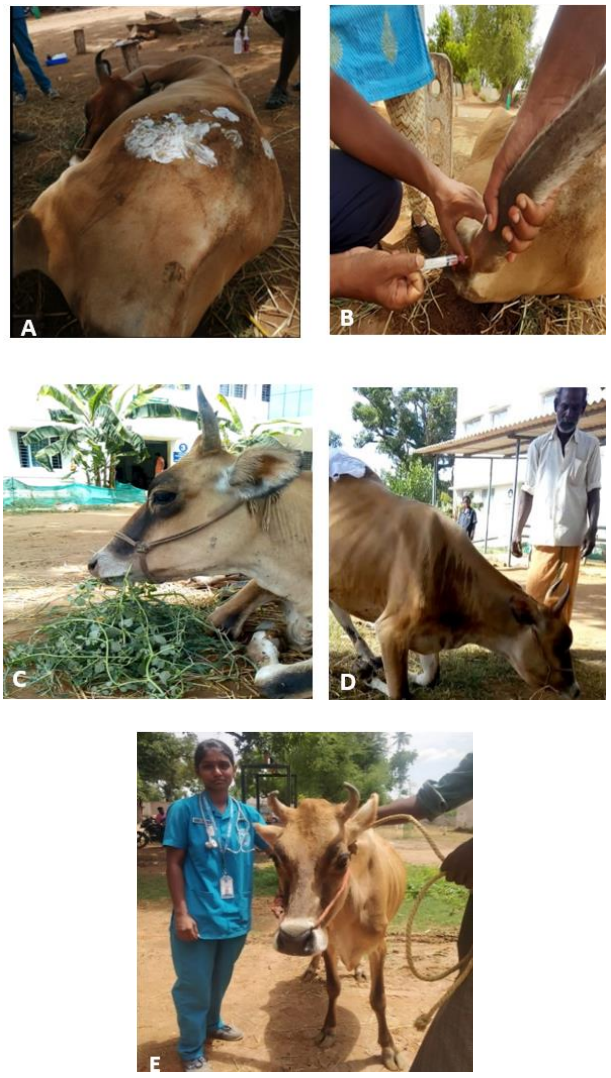

Fig. 1: (**A**) The electrocuted cattle on the day of admission, (**B**) Blood collection from the coccygeal artery, (**C**) After intensive medical management animal revived feeding, (**D**) On the 5th day of admission the animal gained motor activity, and (**E**) On the 15th day of case review

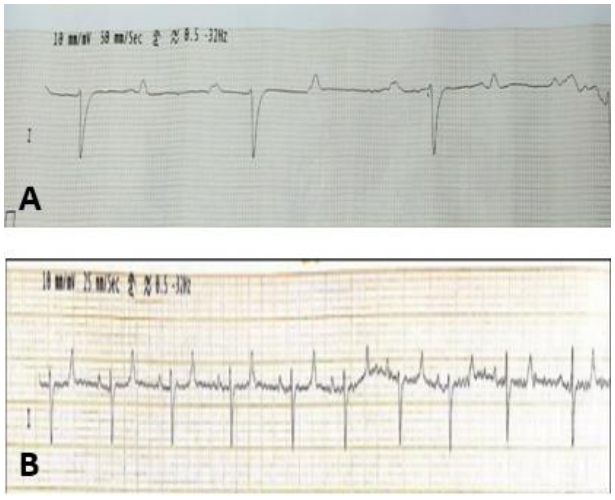

Fig. 2: (**A**) ECG of the electrocuted heifer, showing QT prolongation, before treatment, and (**B**) ECG of the heifer after treatment

analysis revealed hypocalcemia (ionized calcium 0.89 mmol/L), mild hypochloremia, and severe hypokalemia (2.81 mmol/L) (Table 4). Base-apex electrocardiogram of electrocuted heifer showed QT prolongation and tachyarrhythmia at the time of admission. After aggressive treatment and careful monitoring of the heifer, progressive improvement was noticed (Figs. 2A-B).

Emergency care management and follow-up

As part of the initial emergency care, the animal was treated with epinephrine (1:10000) @ 0.01 mg/kg Bwt IV (Tufani *et al*., 2015), Chlorpheniramine malate @ 20 ml IM, dexamethasone @ 2 mg/kg Bwt IM along with fluid therapy with 5% dextrose and normal saline @ 90 ml/kg Bwt. Epinephrine was given for labored breathing, attributed to the damage of blood vessels by electricity and the following fluid leakage that fills the lungs to manage the shock. Fluid support and adrenaline play an important role in the condition of shock and restore blood pressure and cardiac output. Antibiotic coverage was applied to prevent local and systemic sepsis. Other supportive medications instituted were useful in early recovery. Enrofloxacin @ 5 mg/kg Bwt twice daily was given IM to combat any secondary bacterial infections. Burns were dressed with antiseptic solutions; fly repellent spray was also applied to help recovery. Due to the concurrent hypokalemia and hypocalcemia, intravenous infusion of calcium + magnesium phosphate (Mifex®) was given at a dose of 350 ml by slow IV along with diluted potassium chloride 5 ml under ECG monitoring. The case was monitored hourly basis and follow-up therapy was given. Significant recovery was observed by the second week. All the laboratory and PoC analysis become within the reference interval and the same is represented in Tables (1-4). Evident clinical recovery was observed from day seven followed by wound healing, improvement in the animal activities like standing with itself, and appetite (Figs. 1C-E).

Table 3: Arterial blood gas analysis of the electrocuted heifer

Parameters	Day 1	Day 15	Reference range (Harold *et al*., 2000)
pH	8.089	7.4	7.46 ± 0.02
PCO_2 (mmHg)	6.1	27	36.7 ± 3.36
PO_2 (mmHg)	147	112	108 ± 12.6
HCO_3 (mmol/L)	18.5	27.8	26.7 ± 3.10
SO_2 (%)	100	100	99.6 ± 0.62
Lac (mmol/L)	1.90	2.9	3.31 ± 2.17

Table 1: Hematological parameters in electrocuted heifer

Variables	Day 1	Day 2	Day 3	Day 7	Day 15	Reference (Merck Vet Manual)
Erythrocyte (10^6/µL)	8.00	8.2	8.4	8.7	9	5-10
PCV (%)	30	32	32.5	34	37	24-46
HB (g/dl)	12.2	12.6	12.6	12.8	12.4	8-15
WBC (10^3/µL)	14.34	11.12	9.12	8.6	8.9	4-12
Neutrophils (%)	62	65.3	68	54	36	15-33
Lymphocyte (%)	33	32.1	39	44	61	62-63
Monocyte (%)	4	1.3	1	1	1	0-8
Eosinophil (%)	1	1.3	1	1	2	0-20
MVC (fl)	38	38	39	41	45	40-60
MCHC (g/dl)	40.2	39.2	38.1	37.3	33	30-36
MCH (pg)	15.3	14.8	13.4	13.1	15.3	11-17

Table 2: Biochemical parameters at different treatment days of the electrocuted heifer

Variables	Day 1	Day 2	Day 3	Day 7	Day 15	Reference (Venkatesan *et al*., 2020)
Glucose (mg/dl)	41	49	62	81	65	52.60 ± 3.27
Cholesterol (mg/dl)	119	74	78	127	121	107.12 ± 0.29
AST (U/L)	179	153	147	132	107	101.30 ± 7.59
LDH (U/L)	977	654	468	411	380.12	205.20 ± 7.64
Total protein (g/dl)	5.6	5.7	6.3	6.7	8.1	8.8 ± 0.35
Albumin (g/dl)	2.89	2.81	3.05	3.5	4.6	4.4 ± 0.11
Urea (mg/dl)	24	25	24	22	22	22.80 ± 1.5900
Creatinine (mg/dl)	0.98	0.99	0.74	0.98	0.69	0.72 ± 0.075
Cardiac troponin I (ng/dl)	0.33	0.17	0.03	0.03	0.00	0.0204 ± 0.011
CK-MB (U/L)	699	607	502	358	121	76.45 ± 28.24

Table 4: Electrolyte values at different treatment days of the electrocuted heifer

Parameters	Day 1	Day 2	Day 3	Day 7	Day 15	Reference (Yogeshpriya *et al*., 2019)
Potassium (mmol/L)	2.81	3.1	3.55	4.2	4.7	3.6-4.9
Sodium (mmol/L)	143	143.6	144.5	143.4	144.3	136-144
Chloride (mmol/L)	91	90	119.2	105	100.5	99-107
Ionized calcium (mmol/L)	0.89	0.87	0.92	1.00	1.08	1.20
Total calcium (mmol/L)	2.14	2.16	2.21	2.26	2.4	2-2.8
pH	8.04	7.93	7.93	7.5	7.5	7.4

Discussion

Bae *et al*. (2008) reported the accidental electrocution of Korean native cattle by a fallen electric wire, which caused the death of 13 animals. When sudden death of animals occurs in a confined area like pens or stanchions, electrocution should be considered (Radostitis *et al*., 2000). Tufani *et al*. (2015) reported that electrocution in a Rhesus monkey was thrown away on the road after high-voltage electrocution and the monkey was unconscious and had poor response to the surroundings, pale conjunctiva membranes, and sunken eyeball.

In this study, after intensive therapeutic measures, heart and respiration became normal; burns were healing properly, and the animal was gradually returning to normal. Not many reports are available on the survival of cattle following high-tension electrocution shock and the present case was recovered uneventfully. Kisner and Casini (1998) reported that more than 2 amps of current cause significant internal organ and cardiac damage, leading to sudden death.

Since blood is a good conductor of electricity, current tends to flow along blood vessels, causing damage to endothelial cells and myocytes, and resulting in thrombosis. These lesions may develop at any time after the accident, even after several weeks (Price and Cooper, 2013). The Liver enzyme levels consistently increased after the electrical injury and peaked at 3 h (Huitong *et al*., 2016). This finding correlated with our study, which suggests that hepatocytes might experience extensive injuries shortly after electrical injury.

An electric shock occurs when a person or animal comes into contact with an electrical energy source. Electrical energy flows through a portion of the body and causes a shock and burn injury due to the heating effect of the current. At low-voltage contacts, burns are usually localized at the point of contact with the electrical source. Cardiac arrest, either from systole or ventricular fibrillation, may commonly be presented in electrical accidents (Primavesi, 2009).

Biao *et al*. (1998) found that the levels of CK and CK-MB in 32 patients who had experienced an electrical shock were significantly greater than those of normal people. CK-MB is sensitive to skeletal and cardiac muscle damage (Primavesi, 2009) which was elevated in this case. Severe electrical injury is often associated with acute rhabdomyolysis, evident from massively elevated serum creatine (CK) levels, along with presence of other muscle fiber constituents in the serum and urine, resulting specifically in myoglobinuria. Hypocalcemia can result from a combination of hyperphosphatemia and calcium salt accumulation in injured muscle cells in severe cases of rhabdomyolysis (metastatic calcification). This could explain why the animal had myoglobinuria when they were present with a history of myoglobinuria. Understanding the basic structure and function of skeletal muscle, as well as the aetiology of rhabdomyolysis associated with severe electrical injury, is critical to enhancing medical management and increasing the effectiveness of patient care measures (Brumback *et al*., 1995).

Electricity can disrupt cardiac function resulting in arrhythmia causing the animal's death by cardiac arrest. In severely affected, brain damage, unconsciousness or even instant death can occur. Some of the cases of electrical accidents in farm animals with low-voltage power lines also indicated only signs of acute circulatory failure, as reported by Lehman *et al*. (2007). Raised troponin level in the present case is possibly due to myocardial tissue injury and necrosis that had happened because of overheating rather than acute vascular ischemic events, particularly in the absence of major cardiac symptoms. Furthermore, this cTnI level became within the reference range on day 14. Bose *et al*. (2016) reported that the cTnI level was high within the first 5 h of high voltage electric shock due to myocardial damage, and it became normal after 72 h of treatment and further stated that uneventful recovery was evident without any cardiac problem during follow-up of more than four weeks. Based on these findings, cardiac damage due to electric shock could be restored if the cases were presented early to the hospital and immediately initiated critical care therapeutic protocols.

Blood gas analysis has been considered a gold standard method for the assessment of metabolic and respiratory acid-base disorders. Also, it provides information on the interrelationship between ventilation, oxygenation, and metabolic status of the patient and further therapeutic protocols to stabilize the patient (Soltésová *et al*., 2015). Values of HCO_3 and BE observed in our study corresponded to the dynamics of blood pH (Helena *et al*., 2014). The results confirm that the consequences of global respiratory insufficiency affect the respiratory system and the overall system of acid-base balance. Myocardial necrosis and infarction are reported to be rare in human victims of electrocution but have not been reported in cattle.

High-voltage electrocution injury is a serious type of accident with the potential risk of multi-organ damage and death. Early diagnosis of electrocution and immediate management enhances the expectancy of complete recovery.

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Conflict of interest

The authors declare that they have no conflict of interest.

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