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Case Report

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Compartment syndrome of the lower limb: a rare complication of carbon monoxide poisoning

Abstract

Carbon monoxide poisoning (CO) nicknamed "Silent killer" remains the leading cause of poisoning deaths in most countries. In Tunisia, an estimated number of 2000 to 4000 cases of poisoning was noted per a year with an estimated percentage of death up to 90% at the site of intoxication. This intoxication is very exceptionally complicated by a compartment syndrome that can aggravate the functional and vital prognosis when it is dominated by other symptoms, particularly neurological ones. We report the case of a patient who presented a compartment syndrome during CO intoxication that evolved favorably.

Volume 6 Issue 5 - 2018

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Received: October 08, 2018 | **Published:** November 15, 2018

Introduction

Carbon monoxide is a colorless, odourless, non-irritating gas. It is particularly toxic to mammals but undetectable by them giving it an insidious "Silent killer" character. In humans, it is the cause of much domestic intoxication, often fatal. Its causes are most often accidental, due to malfunction or misuse of heating means. CO poisoning remains the leading cause of toxic morbidity and mortality worldwide. It represents a real public health problem.[1,2] The compartmental syndrome is an exceptional complication of intoxication which can be serious.

Patient observation

A 47-year-old patient was admitted to the emergency department for carbon monoxide poisoning. He was victim of CO intoxication by a bath heater. He had headache with vertigo and then he fell unconscious with an inhalation syndrome (Mendelson syndrome). He initially consulted the emergencies for an acute respiratory failure with edema of the lower left limb. As a deep vein thrombosis complicated by a pulmonary embolism was suspected, he had a venous Doppler ultrasound and a thoracic angiography which rejected the pulmonary embolism. He had a cold, swollen left lower limb from the root of the leg to to the toes, with a weak pulse. The delay of capillary refill was prolonged without paresthesia and with a conserved sensitivity evoking a compartmental syndrome. On the admission, he was sleepy and had headache. His blood pressure was 150/80 mm Hg, his heart rate was 115 beats per minute. On the other hand, he was polypneic at 31 cycles per minute and his saturation under 15L of oxygen was 97%. The x-ray of the leg was without abnormality, the Doppler ultrasound of the left lower limb showed a normal caliber arterial network with absence of Doppler flow at the level of the superficial femoral vein which is compressible.

The electrocardiogram was normal. The biological assessment noted a leukocytosis at 14000 elements per mm3, a CRP at 266mg/l, a creatine phosphokinase level at 95291IU/l in addition to a kidney failure with a urea level at 15.17mmol/L and serum creatine at 349µmol/l. Overall, there was severe CO poisoning with an inhalation syndrome causing acute respiratory failure associated with a compartmental syndrome of the left lower limb syndrome causing acute kidney failure (tubular necrosis) with rhabdomyolysis requiring intubation associated to mechanical ventilation with emergency hemodialysis sessions. With regard to the compartmental syndrome, two fasciotomies in emergency were made in our service. Neurologically, with regard to the evolution, he was extubated after 6 days of mechanical ventilation, he kept a preserved state of consciousness but he presented a delirium made of hallucination well treated by Haldol®. On the respiratory level, he was put on antibiotic therapy for inhalation pneumonia which has favorably evolved. As for the metabolic level, the evolution was marked by the persistence of organic anuric renal failure requiring the use of hemodialysis 3 sessions per week to fight against hyperkalemia. Orthopedically, the patient had shown a necrosis of muscle tissue which required an excision of necrotic tissue. After 2 resumptions, the evolution was marked by the improvement of the wound condition. One year after the incident, the patient is asymptomatic, he resumed the diuresis, with a steady respiratory and hemodynamic systems and excellent wound condition in the lower left limb .However, it keeps a paralysis of the SPE and the SPI which are being explored.

Discussion

Carbon monoxide (CO) nicknamed Silent killer by the Anglo-Saxons represents the first cause of mortality by accidental and voluntary poisoning in Europe as well as in the United States.[1] Musculoskeletal manifestations were rare and accounted for only 0.1% of cases. However the neurological signs represented 28%.[2,3] The appearance of a compartmental syndrome during CO poisoning is a very rare situation. The first to describe this association was Larrey in 1812 in Napoleon's soldiers during the Berlin occupation. Other publications have also dealt with this issue. The compartmental syndrome is manifested by pain, tension and deficit signs related to the nerves passing through the compartment are most characteristic and reliable for positive and topographical diagnosis. Problems of consciousness, secondary to CO intoxication, can make diagnosis difficult, and the signs for the compartmental syndrome are those of rhabdomyolysis.

Several authors have reported cases of compartment syndromes associated with CO intoxication on different lodges. For example, Orizaga M. reported two cases of compartment syndrome associated with CO poisoning, one of which involving the forearm and the arm was of a positional character.[4] In addition, Abdul-Ghaffar N. reported five cases of CO poisoning. Two victims died and the other three survivors presented one or more compartmental syndromes. The duration of their exposure was too long in a small confined room suggesting that rhabdomyolysis and the compartmental syndrome, whether postural or not, are the inescapable evolution of CO poisoning.[5] The association of rhabdomyolysis with carbon monoxide poisoning has been described by several authors. This rhabdomyolysis, affecting several muscle groups, seems to be related to a longer duration of CO exposure than to a postural character. Consequently, in addition to the prolonged postural nature that can explain the occurrence of a syndrome of lodge during a CO intoxication, a second mechanism may be involved, it is rhabdomyolysis related to hypoxia and toxicity cell generated by CO. Several mechanisms may be responsible for rhabdomyolysis during CO poisoning, the peripheral hypoxia due to decreased oxygen transport by hemoglobin and the formation of HbCO, the direct toxicity of CO on cells muscle by attachment of CO on myoglobin (MbCO) as well as the formation of radical oxygen species.

However, rhabdomyolysis during CO poisoning is not always synonym of the installation of compartmental syndrome. In contrast, edema is reported in almost all cases of rhabdomyolysis with CO intoxication and may be the cause of the development of compartmental syndrome and gangrenes. The treatment of secondary compartment syndrome during carbon monoxide poisoning is a medical and surgical emergency. After evacuating the patient, it is necessary to dose the concentration of CO in the atmosphere if possible, find the source to stop it, ventilate the room and start oxygen therapy with a mask. Oxygen can be administered at normal pressure by using the high concentration mask 12 to 15liters/minute or at high pressure in hyperbaric chambers. The decompressive fasciotomy must be performed as soon as possible in order to limit very serious aftereffects, at the latest in the first 6hours by necessity. "It is better to operate for nothing than to let a compartment syndrome evolve.[3]

Conclusion

Although the presence of a compartmental syndrome is rare during carbon monoxide poisoning, it could worsen the prognosis when it is delayed.

Acknowledgements

None.

Conflict of interest

Authors declare no conflict of interest.

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Citation: Drissi G, Khaled M, Kraiem H, et al. Compartment syndrome of the lower limb: a rare complication of carbon monoxide poisoning. *Int Clin Pathol J*. 2018;6(5):180–181. DOI: 10.15406/icpjl.2018.06.00184