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Pneumology

Pulmonary Thromboembolism after Carbon Monoxide Poisoning

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Abstract **Case Report**

Carbon monoxide (CO) poisoning is known to increase thrombotic tendency, and the risk of deep vein thrombosis in individuals who have experienced CO poisoning is higher than in the general population. We report the case of a 42-year-old male patient with extensive pulmonary thromboembolism after carbon monoxide poisoning.

Keywords: Pulmonary thromboembolism, case report, carbon monoxide poisoning.

INTRODUCTION

Carbon monoxide (CO) poisoning is a major toxi- cological cause of morbidity and mortality world-wide. CO concentrations ≥667 ppm can cause ≤50% of the body's haemoglobin to convert to carboxyhaemoglobin, which interferes with the transport of oxygen to tissues by haemoglobina [1]. Carbon monoxide (CO) poisoning has been associated with thrombotic tendency; previous reports have described thrombosis in various organs [2-4]. Cerebral, cardiac, mesenteric, and deep vein thrombosis (DVT) are associated with CO poisoning [5]. One nationwide cohort study demonstrated that the risk of DVT is significantly higher in patients with CO poisoning [6].

We report the case of a 42-year-old male patient with pulmonary thromboembolism after carbon monoxide poisoning. In order to describe the clinical and paraclinical presentation and the therapeutic management of this rare disease.

PATIENT AND OBSERVATION

A 42 -year-old man was transferred from reanimation for accidentally CO poisoning due to a malfunctioning heater at home. He appeared awake but confused, with recent memory loss, nausea and vomiting, diffuse tremors, complicated to unconsciousness for which the patient is admitted to the intensive care unit where he was intubated. He had no chronic disease.

Arterial blood gas analysis (BGA) in oxygen showeda lactic acidosis with high carboxyhemoglobin. Frontal chest radiography had objectified left basal triangular opacities with hilar vertex and prepherical base. A chest CT scan had noted an image of partially obstructing endoluminal thrombus in the right lower lobe extended to the laterobasal fig 1.

Laboratory tests revealed leukocytosis, increased troponin I values (109 pg/ml, normal range 0.00 to 0.13 pg/ml), and D-dimer.

The patient received Low molecular weight heparin and was later switched to rivaroxaban, with a good clinical outcome.

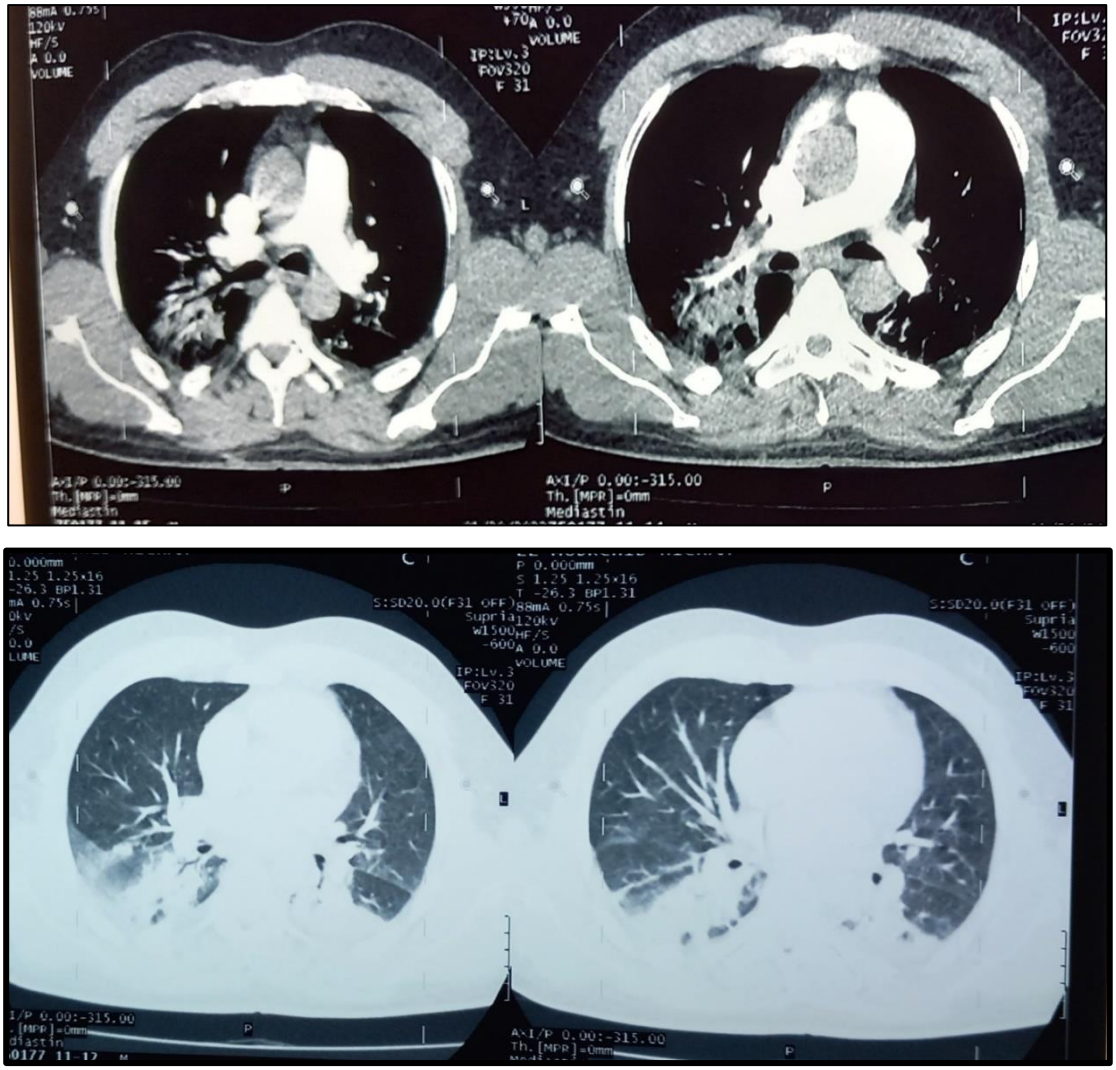

Fig. 1: A chest CT.

DISCUSSION

CO is a colorless, odorless, and tasteless gas that binds with high affinity to many ferrous heme-containing proteins involved in different cellular pathways [7, 8], especially to hemoglobin; thus, CO-bound hemoglobin has reduced oxygen carrying and delivery capacity. Furthermore, CO inhibits mitochondrial respiration impairing adenosine triphosphate production in tissues. Excessive CO concentration can activate platelets by displacement of nitric oxide from platelet surface hemoproteins; displaced free nitric oxide can react with superoxide to produce peroxynitrite, further inhibiting mitochondrial function and increasing platelet and neutrophils activation. This process leads to a vicious circle enhancing inflammation and oxidative stress.

The epidemiological causes of increased risk of DVT in patients with CO poisoning are unclear. Exogenous exposure to high amounts of CO can result in toxic effects, causing hypoxia and inflammation [9]. CO enters the blood toinduce hypoxia through the formation of carboxyhaemoglobin, which causes a leftward shift in the oxyhaemoglobin dissociation curve, and binds to protein to impair mitochondrial function [10]. CO also causes inflammation by increasing the levels of cytosolic and oxygenase-1 protein, and inducing the release of myeloperoxidase, protease and reactive oxygen species, which results in oxidative stress. The pathogenesis of VTE is reportedly associated with inflammation and oxidative stress [11].

The patients with CO poisoning who presented with ARF were associated with substantially increased risk of DVT compared with the control patient. ARF-induced hypoxaemia, associated with hypercoagulability, endothelial dysfunction and immobilisation, might explain our observations of markedly increased risk of DVT in CO poisoning patients with ARF. The patients with CO poisoning receiving hyperbaric oxygen therapy exhibited a considerable risk of DVT, which may be associated with disease severity. The CO poisoning patients with coexisting comorbidities were also associated with substantially increased risk of DVT compared with the comparison patients. Therefore, clinicians should

consider providing DVT prophylactic medication to CO poisoning patients with ARF or coexisting comorbidities [11, 12].

CO cardiac toxicity is due to hypoxic damage and adverse effects at the cellular and molecular level. In particular, because CO binds the heme group of myoglobin with greater affinity than oxygen, its presence in the blood compromises oxygen supply to the cardiomyocyte mitochondria, causing a switch to anaerobic metabolism with consequent hypoxia, lactic acidosis, and apoptosis. Moreover, CO triggers endothelial dysfunction through enhanced transcapillary efflux, leukocyte sequestration within the endothelial lining, and increased oxidation of plasma lipoproteins [13].

By virtue of the complex pathophysiology outlined above, there are several reasons why CO poisoning may enhance risk of thrombosis, fig 2.

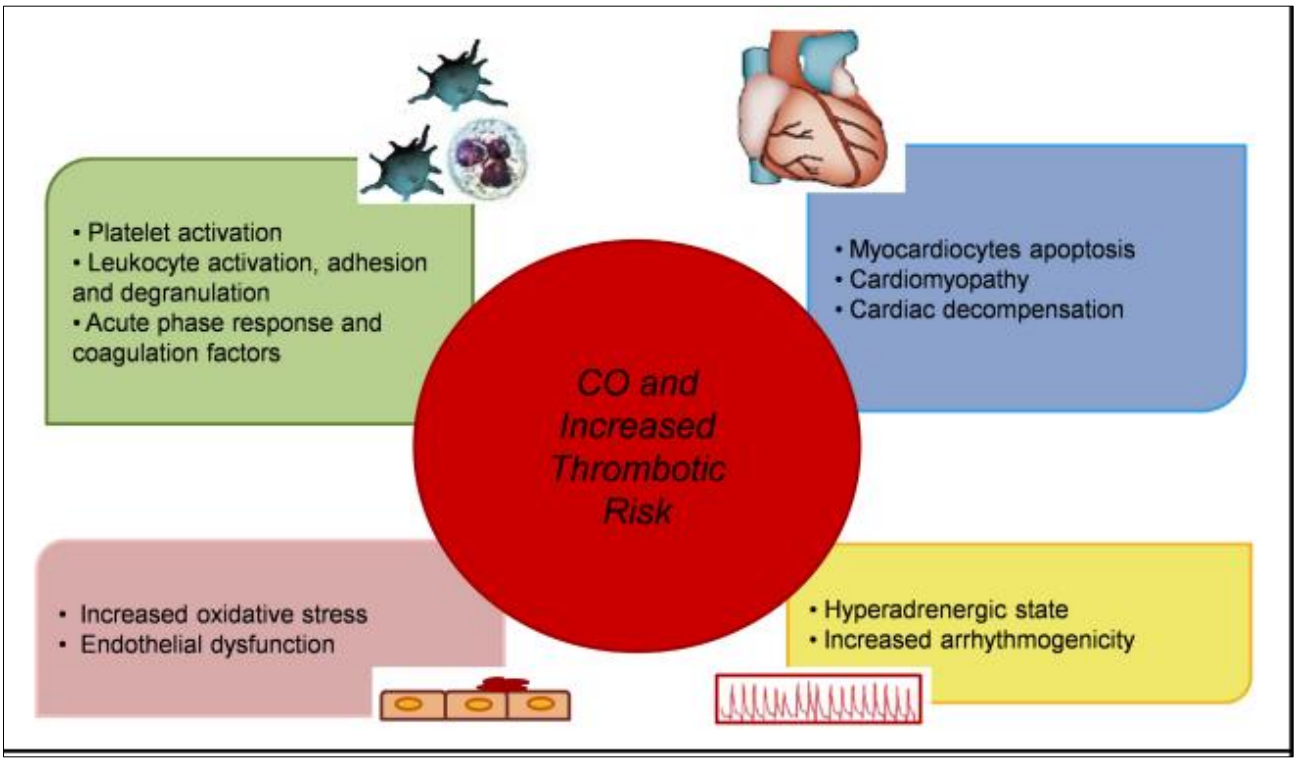

Fig. 2: Possible Mechanisms Involved in Increased Prothrombotic Risk of CO Poisonin

In the largest epidemiological study available [14]. CO poisoning was associated with a 3.85-fold higher risk of DVT, although it was non-significantly associated with PE. Conversely, no association between CO poisoning and bleeding risk has been reported. Despite these data, neither PE nor CO poisoning guidelines mention the potentially lethal association of CO poisoning and DVT/PE [15]. Notably, our patient developed life-threatening PEin the absence of other obvious risk factors such as immobilization, trauma, or genetic evidence of thrombophilia.

CONCLUSION

In agreement with pertinent literature, our observation suggests that CO poisoning is associated with the risk DVT/PE, so active surveillance and prophylaxis for DVT/PE should be considered in severe CO poisoning, including patients suffering from severe CO poisoning.

Compliance with Ethical Standards

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Disclosure of Conflict of Interest

No conflicts of interest in the subject matter.

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