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Intracardiac Thrombosis Secondary to the Silent Killer: A Case Report about Monoxide Carbon Poisoning in a Child

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Abstract

Carbon monoxide (CO) poisoning is a frequent cause of emergency room admissions, especially during winter days, the symptoms are varied ranging from a simple headache to a serious cardiac and neurological impairment that can be deadly. Diagnosis is based on the circumstances of occurrence as well as the dosage of carboxyhemoglobin in the blood. Exposure to CO has serious consequences, neurological and cardiac manifestations are not negligible and vary from repolarization disorders to heart attack. Treatment is urgent with normobaric or hyperbaric oxygen therapy. We report a case of a 2-year male child admitted to the emergency room for CO intoxication with an intracardiac thrombus subsequently complicated by an ischemic stroke with a fatal outcome in order to highlight this complication rarely described in literature.

Keywords

Carbon Monoxide (CO), Poisoning, Intracardiac Thrombosis, Child

1. Introduction

Although the exact incidence is not known, the Centers for Disease Control and Prevention in the United States estimate that 50,000 patients were admitted annually to the emergency department due to CO poisoning [1], the mean reason of death is multiorgan failure and encephalopathic brain death by tissue hypoxia [2], therefore, poisoning may cause thromboembolic events in many organs such as heart, mesenteric artery, brain, and skin sometimes [3] but few reports exist on intracardiac thrombus formation following CO poisoning with only two cases reported previously in the literature in English by Yildirim *et al.* [4] and Lee *et*

al. [5].

We report a case of a 2-year male child admitted to the emergency room for CO intoxication with an intracardiac thrombus subsequently complicated by an ischemic stroke in order to highlight thromboembolic complications rarely described in literature.

2. Case Report

A 2-year-old male child with no personal or family history of chronic disease, and in particular no heart disease, presented to emergency room for hemiplegia of the left side of the body, three days prior to this the child was admitted to the emergency room for headaches and vomiting following CO poisoning due to a domestic gas heater that had been left burning overnight. In the initial evaluation at our emergency department, the patient's blood pressure was 88/53 mmHg, pulse was 102 beats/min, respiration rate was 30 breaths/min, body temperature was 36 degree, and oxygen saturation of peroxymeter (SpO_2) was 100% under 100% oxygen supply with good evolution after 24 hours of monitoring, 48 hours later the child was readmitted for a left hemiparesis, he was alert, Glasgow at 15, blood pressure at 90/65 mm Hg, breathing rate at 40 cycle/min with a heart rate at 140/min, he presented a left hemiparesis with preserved sensitivity and fixity of the gaze on the left side, a hepatomegaly with a hepatic arrow at 10 cm, oxygen saturation level at 94% with oxygen mask, all pulses were present and Cardiovascular examination was without abnormalities, A brain CT scan was performed showed an extensive left temporal-parietal ischemic stroke (**Figure 1**), on the other cardiac echocardiography objectified a hypokinetic left ventricular cardiomyopathy with a huge intra-left ventricular thrombus (**Figure 2**), Electrocardiogram showed no abnormal findings, initial laboratory tests revealed a hemoglobin count of 14 mg/dL, leukocyte count of 8700/mm^3, platelet count at 400,000/mm^3, urea of 0.21 mg/L, creatinine of 4 mg/L, Further biological investigations undertaken showed level of Troponin

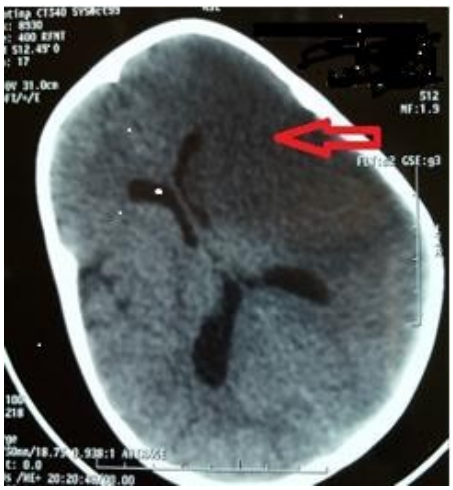

Figure 1. CT scan showed temporo parietal ischemic stroke (arrow).

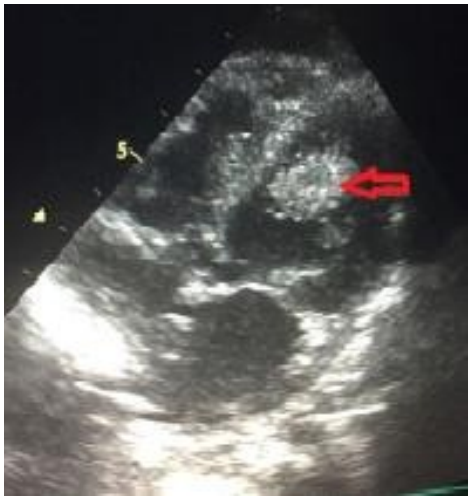

Figure 2. Echocardiogram revealed mobile thrombi in the left ventricle (arrow).

was at 139.9 ng/L, the bleeding profile was normal and the rest of blood tests were without particularities, the carboxyhemoglobin has not been done unfortunately because of lack of resources, and heparinization has been started to prevent aggravation of thrombus formation but unfortunately the evolution was marked a few hours later by respiratory distress complicated by a cardiorespiratory arrest and the infant passed away.

3. Discussion

CO poisoning is the first cause of death by poisoning in France and in the world with an estimated incidence of 4.6% of deaths per million inhabitants [6]. In Morocco, there is no reliable data concerning the real incidence of this type of intoxication, CO causes a decrease in O_2 transport by hemoglobin with an increase in the affinity of CO to hemoglobin forming glycated hemoglobin, which induces a leftward shift in the oxyhemoglobin dissociation curve, decreasing the release of O_2 into tissues. CO also binds to proteins such as myoglobin and cytochrome a3 and disrupts O_2 utilization at the tissue level, particularly by the myocardium that causes tissue hypoxia as well as damage at the cellular level because the affinity of hemoglobin for CO is 200 to 250 times greater than its affinity for oxygen [7], The most frequent signs of intoxication are essentially represented by headaches, asthenia, muscular weakness, especially of the lower limbs [8]. However, these signs remain non-specific and the absence of symptoms is also possible in cases of moderate intoxication [9]. As well as dyspnea and polypnea can be observed. Cardiac manifestations secondary to CO intoxication are not negligible, due to prolonged exposure to CO even at low doses, leading to repolarization disorders (QT prolongation—ST-T change), cardiac dysfunction or intracardiac thrombus formation which remains a rare consequence but can occur during CO intoxication (as the case of our patient), Thromboembolism can occur in oxygen-sensitive organs, the most likely theory of which is that the platelets are affected and there is an exchange between CO and

nitric oxide, resulting in changes in the blood. Oxygen free radicals can cause vascular oxidative stress, increasing platelet aggregation and impairing blood flow, which contributes to endothelial damage and artery stenosis. At the same time, these free radicals disturb hemostasis and inhibit the production of EDRF and prostacyclin by influencing vascular tone and platelet function, thereby increasing thrombus formation, intracardiac thrombosis is rarely reported in literature they were three cases described by Yildirim *et al.* [4] and Lee *et al.* [5]. Concerning the stroke incident, it can be explained by embolic infarction secondary to intracardiac thrombosis and it can also appear many in vital organs such as the lung, brain, and kidney, the prognosis of cases with thromboembolic incidents can be dark with high morbidity and mortality. However, there is yet no consensus for the evaluation and management of cardiac thrombus following CO poisoning. In all of the reported cases of thromboembolic complications in acute CO poisoning, the management consisted of oxygen treatment and anticoagulation therapy with positive results.

4. Conclusion

CO poisoning can be very harmful and causes cardiac injuries by perturbing oxygen delivery. Moreover, it can enhance coagulation pathways. We tried in this report to describe the formation of an intracardiac thrombus induced by CO poisoning in order to remember clinicians that CO intoxication remains the main trigger on intracardiac thrombosis in the absence of any underlying heart or history disease like the case of our patient. The primary care physician should be aware of this eventuality for early management to improve prognosis.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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