

## An Unusual CO-conspirator in a Case of Acute Coronary Syndrome

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**Abstract** Carbon monoxide (CO) poisoning is a common cause of morbidity and mortality that exerts its physiological effects by competition with molecular oxygen. Therefore, tissues that extract a high fraction of oxygen from circulating blood are at high risk for injury due to CO toxicity. While neurological complications of CO poisoning have been well described, cardiac damage secondary to CO poisoning also occurs, but has been less frequently reported. We present the case of a young man with CO poisoning who had elevated cardiac biomarkers, a regional wall motion abnormality, and was found to have obstructive coronary disease. Evidence of myocardial necrosis in the setting of CO toxicity should prompt consideration of an evaluation for coronary artery disease, particularly among those with risk factors.

**Keywords** Carbon monoxide poisoning · Acute coronary syndrome · Coronary artery disease · Cardiac biomarkers

### Case Presentation

A 33-year-old man with chronic chest pain of unclear etiology, obesity, hypertension, type II diabetes mellitus, tobacco use, and family history of premature coronary artery disease was found unresponsive at his home. On the

night prior to admission, he had been using his charcoal grill. He brought the grill into his apartment and went to sleep shortly thereafter. He was found unresponsive the next day and was ultimately admitted for carbon monoxide (CO) toxicity.

After treatment with hyperbaric oxygen, his neurologic status improved. On the second hospital day, he experienced chest pain described as a stabbing, substernal discomfort of 4–6/10 in intensity lasting less than 2 minutes and radiating to the left shoulder. The pain was not associated with nausea, vomiting, or diaphoresis, and there was no exertional component. In retrospect, he stated that similar episodes had been occurring once or twice weekly for many years and were unresponsive to nitroglycerin. He has had multiple stress tests in the past, none of which demonstrated ischemia. However, the most recent such test was over 10 years prior to admission.

On exam, he was alert and oriented without any significant neurologic deficits. His chest was clear to auscultation and he had normal heart sounds without murmur. Serial EKGs showed no ischemic features. His CK, CK-MB, and troponin T levels peaked at 216 U/l, 14.8, and 0.337 ng/ml, respectively. He had a leukocytosis of 16,000 cells per microliter with a neutrophil count of 84% and his glucose was elevated at 408 mg/dl; both of these findings may have reflected his myocardial event.

Cardiac catheterization demonstrated a right dominant system with 90% stenosis in the proximal segment of a large right coronary artery (RCA) as well as mild, non-obstructive coronary artery disease in the other epicardial vessels. He received a 4.5 × 16 mm bare-metal stent to his RCA. The left ventricular ejection fraction by contrast ventriculography was estimated as 60% with hypokinesis of the basal inferior wall. The remainder of his hospital course was unremarkable.

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## Discussion

CO exerts its toxic effects by competing with oxygen binding to hemoglobin, myoglobin, and complex IV of the oxidative phosphorylation pathway. The affinity of CO for hemoglobin is two orders of magnitude higher than that of oxygen; therefore, even low quantities of CO can have serious detrimental effects. The toxicity is specific to tissues with high oxygen demand; neurologic effects are most common, with cardiac toxicity second. Cardiac damage may occur in the absence of neurological changes [1–5].

A recent prospective study of 230 patients treated for moderate to severe CO poisoning with hyperbaric oxygen showed that myocardial injury occurs frequently in those hospitalized for CO poisoning; 9-year follow-up data showed increased mortality associated with myocardial injury, as measured by elevated CK-MB, elevated troponin I, or diagnostic EKG changes [6]. Our patient also had diabetes mellitus and hypertension, both of which are significant predictors of adverse 5-year survival after CO poisoning [6]. It is possible that the patient's underlying epicardial coronary artery disease, also a predictor of decreased survival, contributed to the total amount of myocardial necrosis and depressed LV function beyond that which would have been caused by the acute CO toxicity alone. In this context, the episode of CO toxicity may have served as an unintentional 'biochemical stress test'

that ultimately lead to discovery of flow-limiting atherosclerosis. Therefore, in cases of CO toxicity-related acute coronary syndrome, a traditional evaluation for coronary artery disease may be warranted, particularly in the setting of multiple risk factors.

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