Myocardial Fibrosis From Severe Carbon Monoxide Poisoning Detected by Cardiac Magnetic Resonance Imaging

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Carbon monoxide (CO) poisoning is a leading cause of toxicological morbidity and mortality. We recently reported 37% of patients with moderate to severe CO poisoning suffered myocardial injury. In follow-up, 24% of patients died at a median of 7.6 years, a mortality rate 3 times higher than expected compared with age- and sex-specific US mortality rates. Myocardial injury was the major predictor of mortality: 38% of patients who sustained myocardial injury died compared with 15% without myocardial injury. The precise mechanisms responsible for the increase in mortality remain unclear, but cardiovascular death was more common (44% versus 18%) among patients who initially sustained myocardial injury.

We report a case of myocardial fibrosis after severe CO poisoning detected by cardiac MRI. A previously healthy 63-year-old woman presented to an outside hospital with severe CO poisoning. She was comatose, with an initial carboxyhemoglobin level of 28.6%. Baseline troponin I (0.09 ng/mL) and creatine kinase–MB (7.7 ng/mL) were normal, but serial cardiac biomarkers were not obtained. Admission ECG showed sinus tachycardia with ventricular bigeminy and diffuse ST-depression consistent with ischemia. Her clinical course was complicated by neurological dysfunction. At 4-month follow-up, the ECG and echocardiogram were normal (left ventricular ejection fraction 65% without wall-motion abnormalities). Cardiac adenosine MRI showed no rest or stress-induced myocardial perfusion defects but revealed inferolateral midwall myocardial fibrosis (not in the typical distribution of a coronary artery) that spared the endocardium (Figure).

This is the first report of myocardial fibrosis from CO poisoning detected by MRI. Midwall myocardial fibrosis has been reported in dilated cardiomyopathy of unclear origin. In a series of 63 patients, 18 (28%) had midwall enhancement. In a follow-up of 101 consecutive patients with dilated cardiomyopathy, midwall fibrosis (present in 35% of patients) predicted a combined end point of all-cause mortality and cardiovascular hospitalization, as well as sudden cardiac death. Myocardial fibrosis has also been demonstrated in hypertrophic cardiomyopathy and has been linked to known markers for sudden cardiac death, although the independent prognostic value of MRI has yet to be determined. In our case, myocardial fibrosis was present in the setting of a completely normal echocardiogram, which suggests the utility of obtaining cardiac MRI in patients with moderate to severe CO poisoning. The incidence and prognostic significance of myocardial fibrosis after CO poisoning are unclear but deserve further study and may provide a link to adverse long-term outcome for a subset of patients.

Disclosures

None.

References

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