Exhibit 4

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MARTINSVILLE VIRGINIA CIRCUIT COURT CASE NO. CR19000009-00

UNITED STATES DISTRICT COURT CASE NO. 1:13-CR-435-1
MIDDLE DISTRICT OF NORTH CAROLINA

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TRANSIENT CARDIAC DYSFUNCTION IN ACUTE CARBON MONOXIDE POISONING

To the Editor:

Carbon monoxide inhalation is a leading cause of poison-related morbidity and mortality in the United States (1). We report a patient with reversible right bundle branch block and severe cardiac dysfunction. This case describing a transient cardiac conduction block compliments those reported by others (2).

A 49-year-old woman was found in an idling automobile in a closed garage. First responders arrived within 30 minutes and found her to have sinus tachycardia with a heart rate of 100 beats per minute, systolic blood pressure of 80 mm Hg, respiratory rate of 8 breaths per minute with room air oxygen saturation of 85%, and a Glasgow Coma Scale of 6. The patient was intubated, given intravenous crystalloid, and transported to a community hospital.

On arrival, the patient had a carboxyhemoglobin level of 35%, a lactate level of 5.0 mmol/L (45.5 mg/dL), and a negative toxicology screen. The electrocardiogram (ECG) showed sinus tachycardia. She was treated with intravenous crystalloid followed by dopamine for hypotension, and then flown to a level 1 trauma center and treated with hyperbaric oxygen therapy (3).

At this facility, her carboxyhemoglobin level was 5.7%, creatine kinase level was 3161 mg/dL, and creatine kinase–MB level was 30.7 mg/dL with a creatine kinase–MB/creatine kinase ratio of 1%. Troponin I level was 2.6 ng/mL. Chest radiograph showed bilateral lower lobe infiltrates. Dopamine was changed to norepinephrine because of refractory hypotension. An ECG demonstrated a new right bundle branch block (Figure 1, left). Troponin I levels peaked at 23.4 ng/mL on hospital day 2. The patient had no history of coronary artery disease and had no associated risk factors. An echocardiogram on hospital day 2 demonstrated a left ventricular ejection fraction of 20% with global hypokinesis and moderately reduced right ventricular function. The right bundle branch block resolved by hospital day 3 (Figure 1, right). Norepinephrine was discontinued on hospital day 3. A repeat echocardiogram on hospital day 4 demonstrated a left ventricular ejection fraction of 40% with mild global hypokinesis and normal right ventricular function. A myocardial perfusion scan on hospital day 8 demonstrated no evidence of ischemia or infarct and a left ventricular ejection fraction of 70% (Figure 2).

Neurologically the patient improved. She was extubated on day 6, and the Glasgow Coma Scale was 14. She exhibited cognitive dysfunction and balance problems. She was in rehabilitation from day 8 to 15 and was then discharged to an inpatient psychiatry unit. At 6 months, cognitive testing showed moderate depression and only subtle cognitive impairments.

This patient developed a reversible cardiac conduction block and severe, reversible left ventricular dysfunction. This case compliments prior reports of reversible left ventricular dysfunction with carbon monoxide inhalation (2). Reversible intraventricular cardiac conduction delays in carbon monoxide poisoning have been described in 2 cases (4), neither of which involved complete bundle branch block.

The patient had elevated troponin I levels suggesting cardiac myonecrosis. Prior reports of carbon monoxide poisoning suggest that cardiac toxicity is likely caused by acute, generalized tissue hypoxia and toxic effects on myocardial mitochondria (5). Throughout the patient's hospitalization, she had no ischemic ECG findings, such as ST-T abnormalities or Q waves. The patient's lack of known coronary artery disease and absence of risk factors, and normal follow-up nuclear perfusion study, suggest that her reversible cardiac dysfunction was due to acute carbon monoxide poisoning and not vascular obstruction, which agrees with a case report of acute myocardial infarction following carbon monoxide poisoning and subsequent normal coronary angiography (5).

The patient had cardiogenic shock requiring norepinephrine. Others have also reported reversible and persistent right and left ventricular dysfunction following acute carbon monoxide poisoning (4). It is uncertain how myocardial damage from carbon monoxide poisoning is differ-

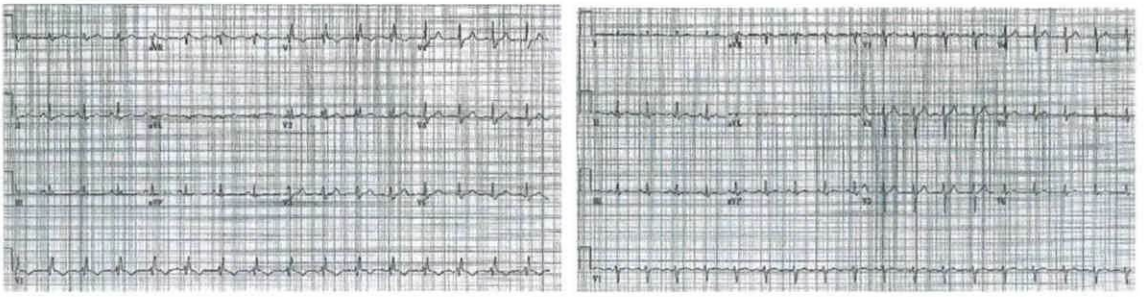

Figure 1. Electrocardiogram of patient on arrival at the tertiary care center (left) and on hospital day 3 (right). The right bundle branch block resolved following hyperbaric treatments with the QRS narrowing from 130 to 90 ms.

ent from acute coronary arterial ischemia, and why the ventricular dysfunction may be reversible. Myocardial stunning may be responsible. Also, carbon monoxide poisoning causes oxidative stress (1), which could reduce nitric oxide levels, causing the heart to become stiffer (6), as well as causing direct myocardial injury. In addition to enhancing patient care, further understanding of the reversibility of carbon monoxide–induced myocardial dysfunction is important because carbon monoxide–poisoned patients are a potentially underutilized source of heart donors for transplantation (7).

Along with substantial troponin I elevation and reversible echocardiographic changes, the patient also demonstrated a transient right bundle branch block. The reversible nature of her conduction disease could be consistent with acute carbon monoxide–induced myocardial stunning (8). ECG abnormalities have been described before in cases of carbon monoxide poisoning, including ST-segment and T-wave abnormalities, premature atrial and ventricular contractions, and atrial and ventricular fibrillation. We also discovered 2 other cases describing intraventricular conduction delays, but neither demonstrated a reversible or persistent right bundle branch block (9).

Myocardial injury with transiently abnormal ventricular function and electrical conduction can result from acute carbon monoxide poisoning, yet have a favorable cardiac outcome.

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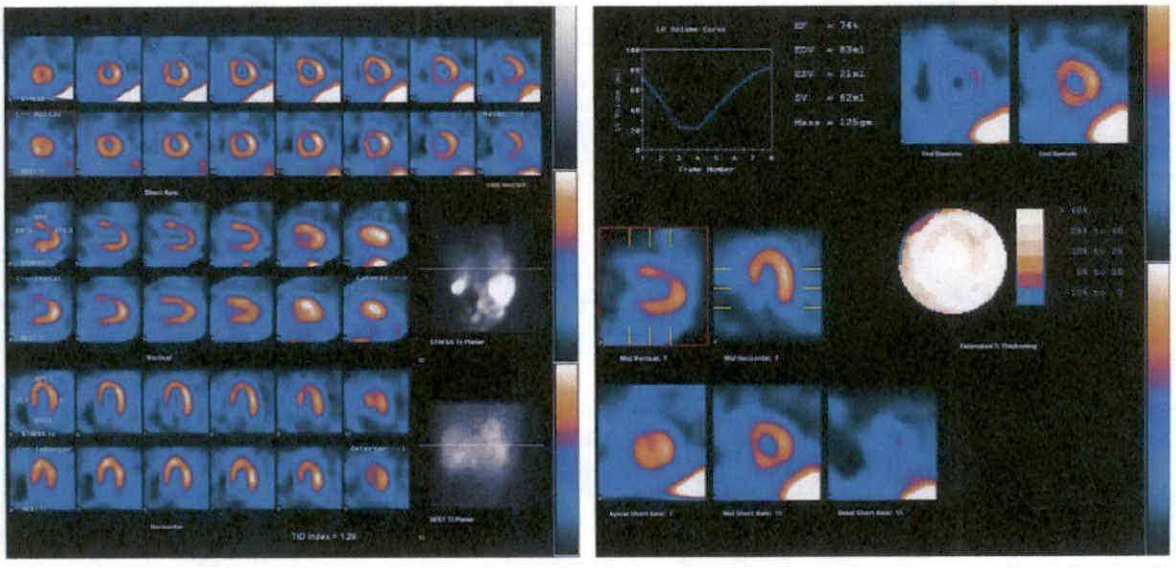

Figure 2. Dual isotope nuclear medicine perfusion study. Noninvasive stress imaging demonstrated no reversible or fixed perfusion defects and a normal left ventricular ejection fraction of 70%.