CASE REPORT

Carbon monoxide poisoning mimicking long-QT induced syncope

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Carbon monoxide (CO) poisoning is a rare cause of QT prolongation, and is therefore easily missed. The case of a patient with unexplained syncope and QT prolongation on the electrocardiogram that turned out to be related to CO poisoning is reported here. In patients with QT prolongation, uncommon causes also should be looked for.

A 9-year-old girl presented at the emergency department after she had a syncope on a motorboat. She was ready to water ski, and sat on the swim platform. The boat was in an idling position. Her father, who was steering the boat, mentioned that his daughter had suddenly lost consciousness and lay in a prone position on the swim platform. She slowly recovered and was fully conscious by 10 min. At that moment she was given 100% oxygen by facemask. She arrived at the emergency department 1 h after the syncope. Her medical history was uneventful. She did not use any drug. Family history was uneventful for sudden death and syncope.

On examination, no abnormalities were found and the neurological examination. She had a normal temperature (36.2°C) and blood pressure (108/72 mm Hg). Transcutaneous measured oxygen saturation with and without supplemental oxygen was 100%.

Blood gas examination was normal and no electrolyte disturbances were found. A 12-lead electrocardiogram (ECG) showed a normal sinus rhythm of 106 beats/min (bpm). The ECG was further analysed to calculate the corrected QT time (QTc) and the corrected QT dispersion time (QTcdisp). The QTc is the measured QT interval, corrected for heart rate using Bazett's formula ($QT/RR^{1/2}$). Corrected QT dispersion is the difference between the shortest and longest QTc interval on a 12-lead ECG. The QTc was prolonged (481 ms) and the QTc dispersion (107 ms; table 1 and fig 1) was prolonged as well. A diagnosis of long-QT syndrome was suspected until laboratory results showed a carboxyhaemoglobin (COHb) level of 25%. The patient was admitted to the hospital and supplemental oxygen was continued. The COHb level decreased to 16% by

3–4 h and normalised by 7 h. At 12 h after admission, all electrocardiographic abnormalities had normalised as well. QTc had decreased to 441 ms, and QTc dispersion had decreased to 51 ms (table 1). She was discharged home the same day. Follow-up after 3 months did not show any abnormalities, and she had normal electrocardiogram (fig 2). In addition, both parents were tested for electrocardiographic abnormalities, but they turned out to have a normal ECG, including normal QTc of 402 and 384 ms, respectively.

DISCUSSION

Long QT syndrome is related to syncope and sudden cardiac death. As prophylactic use of β -blockers substantially reduces these risks, it is extremely important to make the diagnosis as early as possible. However, other causes of QT prolongation must not be overlooked, as this may lead to unnecessary use of drugs and may postpone a right diagnosis. The present case showed a rare cause of QT prolongation.^{1 2}

Severe carbon monoxide (CO) poisoning can produce symptoms such as seizures, syncope or coma, accompanied by myocardial ischaemia, ventricular arrhythmias, pulmonary oedema and profound lactic acidosis.^{3 4} Acute CO poisoning is usually suspected on the basis of a suggestive history. CO poisoning related to open air exposure to motor boats has been described previously.⁵ However, we made the diagnosis of CO poisoning just after laboratory results became available. CO binds to haemoglobin with much greater affinity than oxygen (approximately 240 times), forming carboxyhaemoglobin and resulting in impaired oxygen transport and utilisation. This results in impairment in tissue oxygen delivery and has a negative effect on the carbon dioxide removal from the tissues. Experimentally induced hypoxia and hypercapnia have both been shown to increase the QTc dispersion.^{6 7}

Our patient showed prolongation of the QTc, especially an increase in the QTc dispersion (table 1). All these values were normalised before discharge. Comparing the QTc dispersion of our patient with normal values, the 51 ms at discharge was normal.⁸ The JT (QT – QRS) values and the standard deviation values are also mentioned; these show a similar normalisation in the ECG before discharge. Although in the present patient CO intoxication was the probable cause of QT prolongation, other secondary causes of QT prolongation have a higher incidence, including electrolyte disturbances, especially low levels of calcium, potassium and magnesium, hypothermia and drug usage. However, our patient did have a normal temperature and normal electrolyte levels; She did not use any drugs. Familial long QT syndrome seems unlikely because of the normal parental ECGs and the normalised ECG during follow-up. Therefore, other causes of her long QT seem less likely.

Table 1 Details of follow-up examination

	ECG 1 (ms)	ECG 2 (ms)	ECG 3 (ms)
QTc	481	441	426
JTc	401	353	374
QTcdisp	107	51	39
JTcdisp	107	63	59
SDQTc	28.7	13.3	14.2
SDJTc	33.4	17.7	17.7

ECG 1, ECG values at admittance; ECG 2, before discharge; ECG 3, ECG during follow up; JTc, corrected JT time; JTcdisp, corrected JT dispersion time; QTc, corrected QT time; QTcdisp, corrected QT dispersion time; SDJTc, standard deviation of JTc; SDQTc, standard deviation of QTc

Abbreviations: COHb, carboxyhaemoglobin; ECG, electrocardiogram; QTc, corrected QT; QTcdisp, corrected QT dispersion

Figure 1 Electrocardiogram at admittance to the hospital. Rate 106 bpm, sinus rhythm, normal axis and conduction. Calculated corrected QT time time was 481 ms, and corrected QT and JT dispersion time of 107 ms.

Figure 2 Electrocardiogram 3 during follow-up. Rate 95 bpm, sinus rhythm, normal axis and conduction. Calculated corrected QT time time was 426 ms, and corrected QT and JT dispersion times 39 and 59 ms, respectively.

CONCLUSION

Our case history describes a patient with unexplained syncope and reversible increases in QTc and QTc dispersion related to CO intoxication. The CO poisoning resulted from direct exposure to CO in the exhaust of a ski boat. Making the diagnosis without delay had important therapeutic implications, as administration of 100% oxygen showed the COHb fraction to decrease rapidly, with concurrent normalisation of the ECG.

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