

Case report of carbon monoxide poisoning in a pregnant patient: mother died, baby survived

一名孕婦的一氧化碳中毒個案：母親死亡，嬰兒生存

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Carbon monoxide poisoning in pregnant women is a relatively rare condition. We report a 32-year-old woman in her 32nd week of pregnancy found unconscious in the bathroom. On arrival, her pulse and blood pressure were undetectable. Cardiopulmonary resuscitation was applied. The mother's carboxyhaemoglobin level was 57%. Due to foetal distress, Caesarean section was performed in the emergency department. The baby was intubated due to the absence of spontaneous respiration. The level of carboxyhaemoglobin in the cord blood was 32%. After staying in the newborn unit for 47 days, the baby was discharged with a sequela of cerebral palsy. (*Hong Kong j.emerg.med.* 2009;16:176-178)

孕婦一氧化碳中毒是比較罕有的情況。我們報告一名32歲懷孕32週的女子，被發現昏迷在浴室內。到達醫院時，測不到血壓脈搏，需要施行心肺復甦法。母親的一氧化碳血紅蛋白水平為57%。因胎兒狀態窘迫，需在急症室進行剖腹產子。因沒有自發性呼吸，嬰兒需氣管插喉。臍帶血的一氧化碳血紅蛋白水平為32%。在新生兒單位住了47天後，嬰兒可以出院，但後遺症為大腦性麻痺。

Keywords: Carbon monoxide poisoning, fetus, pregnancy

關鍵詞：一氧化碳中毒、胎兒、懷孕

Introduction

Carbon monoxide (CO) poisoning in pregnancy is a relatively rare condition, with potentially serious complications for both the mother and baby. Rapid oxygen dissociation and prolonged clearance of CO in

the foetal circulation emphasize the importance of adhering to aggressive treatment protocols.^{1,2} Additionally, a time lag for both uptake and elimination of CO between the mother and foetus has been demonstrated, with the foetus at risk for hypoxia even when the mother's blood level of CO is nontoxic.

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Case report

A 32-year-old non-smoking female at 32-week gestation was admitted to the emergency department (ED) in December 2006 with cardiac arrest due to exposure to CO in the bathroom which was a totally enclosed space. At the scene, the water heater was malfunctioning but was still running and active. The patient was transferred to the hospital around 40 minutes after the exposure. The initial examination showed that the mother was pulseless and apnoeic. Her

pupils were mid-dilated. The Glasgow Coma Scale score was 3/15. The patient was intubated, 100% oxygen support was given and cardiopulmonary resuscitation (CPR) was commenced in accordance with Advanced Cardiac Life Support. A carboxyhaemoglobin (COHb) level of 57% was obtained. Bedside foetal ultrasonography showed a weak foetal heart pulsation and delivery by means of Caesarean section in the ED was decided after emergency gynaecological consultation. CPR was maintained before, during and after the procedure of Caesarean section for 45 minutes. A total of 3 mg of atropine and 9 mg of adrenaline were given in boluses to the mother. The Caesarean section lasted for 15 minutes. The mother was certified dead 15 minutes after the delivery. A 1500 gram baby was delivered and his initial Apgar score was 1. The baby suffered from bradycardia (40 beats per minute) with no spontaneous respiration, and was intubated. Apgar scores were not measured subsequently as the baby was intubated. The COHb level in the cord blood was 32%. The levels of COHb measured at 3-hour intervals were 18%, 9% and 1.1% respectively. The baby was taken to the neonatal intensive care unit. He was given ventilatory support for 24 days, managed with various intravenous antibiotics for sepsis and finally discharged with cerebral palsy on his 47th day of hospitalisation. As hyperbaric oxygen treatment (HBOT) was not available in our centre and the health status of the baby did not permit prolonged transportation, HBOT was not given.

Discussion

CO is responsible for a large percentage of accidental poisonings and deaths reported throughout the world.³ The severity of symptoms depends on various factors such as the duration of exposure, the concentration of CO in the environment, the underlying health status and the susceptibility of the individual.⁴ Even exposure to CO for a short duration at high concentration may lead to coma or fatal events. In our case, the patient stayed in the bathroom with malfunctioning water heater for about 25 minutes, and exposed to CO at high concentration.

The incidence of pregnancy among CO poisoning victims varies between 4.6-8.5%.^{5,6} Breslau reported the intoxication of two pregnant women after a gas explosion.⁷ One woman had an apparently normal child at delivery, but the other suffered a stillbirth: the death occurred one day after exposure. Freund reported a death in utero during the seventh month of gestation.⁸ Farrow et al⁹ reported a foetal death due to accidental nonlethal maternal CO intoxication. From the previous reports, we could not find a case in which the mother died as a result of CO poisoning but the baby was delivered alive.

CO passes through the placenta either by passive diffusion, or by the facilitated mechanism of a carrier.^{10,11} Placental CO diffusion capacity increases with gestational age and in proportion to foetal weight. This is attributable to the rate of placental blood flow and maternal haemoglobin concentration. The increased area of placental exchange and the decreased distance between maternal and foetal blood are partially responsible for this change. Slow dissociation of CO from maternal haemoglobin accounts for the delay in the release of CO and for the accumulation of CO in the foetus. Acute CO toxicity in pregnancy causes foetal tissue hypoxia through two mechanisms: a decreased level of maternal oxygen and the transplacental passage of CO. As maternal COHb rises and the blood oxygen content decreases, oxygen release by haemoglobin diminishes, and oxygen transport across the placenta decreases dramatically. Oxygen in the foetal umbilical vein decreases and tissue hypoxia occurs. CO crosses the placenta and combines with foetal haemoglobin. Foetal COHb levels are approximately 10-15% higher than maternal levels. Elimination is slower in the foetus.¹⁰ In our case, while the initial COHb level of the mother was 57%, the COHb level of the foetus obtained from the cord blood just after the Caesarean section was 32%. The reason of the lower level of COHb in the cord blood may be the result of effective oxygen support and the delivery of the baby at an early stage.

The level of maternal COHb is a poor indicator of foetal toxicity. Despite maternal wellbeing, foetal morbidity or mortality can still occur.¹¹ It was not

known whether the initial symptoms and findings of the baby (bradycardia, absence of spontaneous respiration) were the result of CO toxicity or prematurity.

The pathogenesis of foetal brain damage caused by acute maternal CO intoxication was experimentally investigated in cats.¹² From the distribution and nature of the brain changes, a hypoxic-ischaemic mechanism was proposed as the pathogenesis of foetal brain damage. In the foetuses in middle and early gestational stages, the frequency and severity of brain changes were generally lower than in those in the late gestational stage, and the cerebral white matter and basal ganglia were most often involved, but the thalamus, brain stem and the cerebral cortex were spared.¹² In our case we did not know the exact reason of the cerebral palsy. It might be due to the direct effect of CO intoxication to the brain, maternal arrest and delayed delivery, or a combination of both.

Almost all studies performed in recent years recommended HBOT in severe CO intoxications.^{2,13,14} Although CO intoxication in pregnancy may be clinically insignificant, HBOT is recommended for both mother and baby as foetal CO level is significantly higher than the mother's.^{10,14}

In conclusion, acute CO poisoning during pregnancy is comparatively uncommon, yet can result in foetal death, functional alterations or anatomical malformations in survivors. CO kinetics is different in the foetal circulation. Hyperbaric oxygen seems to be the treatment of choice and all pregnant women with a viable foetus suffering from CO intoxication should be referred for hyperbaric oxygen therapy.

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