SEVERE PARACETAMOL POISONING WITH METHEMOGLOBINEMIA
Olsson Eva¹, Lemberg Marie². 1. Swedish Poisons Information Centre, Stockholm, Sweden; 2. Department of Anaesthesia and Intensive Care, Västerås Central Hospital, Sweden

Objective
Paracetamol poisoning may cause metabolic acidosis and coma after large ingestion, and treatment with CCVHD in severe paracetamol poisoning has been described previously. We present a case of massive paracetamol ingestion with coma, metabolic acidosis, methemoglobinemia, rapid onset of multiorgan failure and death within 48 hours.

Case report
A 45-year old woman with a history of gastric bypass surgery and depression was found unconscious after an estimated intake of 190 grams paracetamol. She had not been in contact with her relatives for 24 hours and was surrounded by boxes of paracetamol with approximately 1000 additional tablets.

On hospital arrival she was comatose, hypotensive with systolic blood pressure 45 mmHg and hypothermic 26 degrees C.

She had severe metabolic acidosis with pH 6.96, base excess -23 mmol/L and serum lactate 18 mmol/L. Blood glucose was 1.0 mmol/L. ASAT was <0.2 mikrokat/L, ALAT 4.69 mikrokat/L and PK(INR) 1.2.

She also had a methemoglobinemia of 28.6%.

CT scan of the brain showed no bleeding or signs of cerebral edema.

Treatment with intravenous N-acetylcysteine (NAC) was started within an hour without having the serum paracetamol level. Serum paracetamol was initially 11 812 mikromol/L, and 2 hours later 10 314 mikromol/L.

The circulation was stabilized but the metabolic acidosis worsened despite intensive supportive treatment with vasoactive drugs, inotropic support, fluids and buffer treatment.

Conclusion
This is to our knowledge the first case presenting with massive paracetamol poisoning and methemoglobinemia. Since there were no other known substances involved it is likely that the methemoglobinemia was a direct consequence of the paracetamol poisoning.

Due to the severe metabolic acidosis, the extremely high serum paracetamol concentration and an incipient kidney failure CVVHD was initiated. Infusion with NAC was doubled during dialysis.

The methemoglobinemia was treated with methylene blue in repeated doses and fell from 28 to 16%.

Twelve hours after hospital arrival the patient was transferred to a university hospital liver unit for possible MARS treatment and acute liver transplantation.

On arrival the serum paracetamol was 6100 mikromol/L, ASAT 19.1 mikrokat/L, ALAT 7.21 mikrokat/L and PK(INR) 2.6.

A few hours later her circulation failed and was unresponsive to resuscitation.

Due to the severe metabolic acidosis, the extremely high serum paracetamol concentration and an incipient kidney failure CVVHD was initiated. Infusion with NAC was doubled during dialysis.

The methemoglobinemia was treated with methylene blue in repeated doses and fell from 28 to 16%.

Twelve hours after hospital arrival the patient was transferred to a university hospital liver unit for possible MARS treatment and acute liver transplantation.

On arrival the serum paracetamol was 6100 mikromol/L, ASAT 19.1 mikrokat/L, ALAT 7.21 mikrokat/L and PK(INR) 2.6.

A few hours later her circulation failed and was unresponsive to resuscitation.