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# Letter to the editor

## Life threatening hemolysis in a 27 year-old drug addict

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A 27 year-old male subject with a ten year history of intravenous drug abuse was admitted to our department. He had not left his apartment for two weeks because of extreme weakness and was found by his mother, who took him to the emergency room. The most remarkable features among his laboratory results were an anemia with a red blood cell count of $0.7 \times 10^6/mm^3$ ($0.7 \times 10^{12}/l$), 2.8% reticulocytes (0.028 percent of 1.00), 12,000 leucocytes/$mm^3$ ($12,000 \times 10^6$ cells/l) and $378 \times 10^3/mm^3$ ($378 \times 10^3/l$) platlet count, hemoglobin 3.2 g/dl (32 g/l), hematocrit 9.3% (0.093), mean corpusular volume 98 $\mu m^3$. Coombs blocking was negative. Haptoglobin serum concentration was reduced (45 mg/dl; 0.45 g/l) total bilirubin was 8.0 mg/dl (136 $\mu$mol/l), conjugated bilirubin 4.3 mg/dl (73 $\mu$mol/l), indirect bilirubin 3.7 mg/dl (63 $\mu$mol/l).

Further blood analysis showed LDH 845 U/l, alkaline phospatase 225 U/I, AST 53 U/l, ALT 28 U/l, GGT 10 U/l and cholinesterse 1340 U/l. The serum concentrations of vitamin B 12 and folic acid were 243 pmol/l and 9.5 nmol/l, respectively. Hbs antigen was proved negative, HbC antibody positive, HCV antibody and HIV antibody negative. The stool examination for occult blood was negative. Blood pressure was 100/70 mmHg. Thus severe hemolysis was established. The patient had been attending a methadone maintenance program receiving 80 mg of methadone daily, and furthermore reported taking heroin and cocaine intravenously. Except for flunitrazepam he denied taking any other drugs.

On the first two days after admission he received five units of red blood cell concentrate, but despite a therapeutic trial with corticoides (80 mg prednisolon/day) the hemolysis did not stop. Since a potential link between drug abuse and hemolysis was suspected, a urine sample was obtained on the fifth day, which proved positive for methadone (half life 25 hours) and opiates (half life 7 hours). Despite the patient's denial, it was proven that he had continued to use heroin during his stay in the hospital. In order to cut off the patient's access to drugs he was transfered to the intensive care unit of the psychiatric hospital. Normalisation occured within a few days. When the patient was confronted with these facts he admitted that he continued taking heroin during admission dissolved in lemon juice or vinegar in order to enhance the desired effect, especially "das Einfahren" (slang for the initial intoxicating effect). On average he had been taking 0.5 g of heroin diluted with about 0.5 to 3 ml lemon juice four times daily. Interestingly, he was using the central venous catheter to administer the opiates during his hospital stay.

Hereditary causes for hemolysis could be ruled out because of normal blood counts from former hospital stays. A paroxysmal noctural hemoglobinuria was unlikely because of its chronic progress. Therefore, after excluding hematologic disorders, the differential diagnosis consisted of an immunhemolytic process, a toxic effect or a pernicious anemia.

An immunhemolytic anemia could be ruled out because of negative Coombs testing and the failure of glucocorticoid therapy. Serum concentration of vitamin B 12 was within normal range (243 pmol/l), so that the direct toxic effect of citric and acetic acid seems to have induced the severe hemolysis.

The cause of hemolysis could be due to local ph changes caused by introduction of the citric acid (ph 1.5) and acetic acid (ph 2.0) into his bloodstream. A hemolytic effect has already been reported for acetic acid (Linden *et al*, 1983). However, the osmolarity of citric and acetic acid are 308 mmol/l and 560 mmol/l, respectively and therefore not a cause of hemolysis.

The repeated incidents of slow hemolysis following the injection of citric and acetic acid also seem to be responsible for the patient's relatively good adaptation to the extreme low red blood cell counts.

The above mentioned case documents the practice of combining intravenous opiates with household acids in order to increase the initial intoxicating effect. This can in turn lead to a life threatening hemolysis. As far as we know, this connection has not been addressed in relevant literature.

Linden C, Berner JM, Kulig K, Rumback BH. Toxicity following systematic absorption. *Vet Hum Toxicol* 1983;25:66

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