

CASE REPORT

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**Perimortem fixation of the gastric and duodenal mucosa:
a diagnostic indication for oral poisoning**

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Abstract Two cases of fatal oral poisoning are presented. In the first case, a 40-year-old man died due to a lethal dose of mercury (blood concentration 113.8 µg/ml) and in the second, a 34-year-old man died of chloralhydrate overdose with a lethal blood concentration of trichloroethanol (52 µg/ml), the active metabolite of chloralhydrate. In both cases gross examination and histology showed an unusually well preserved gastrointestinal mucosa in addition to unspecific signs of intoxication. The two cases demonstrate that the phenomenon of perimortem fixation is a useful indication for the forensic pathologist and should direct the suspicion to oral poisoning. The detection of fixation facilitates toxicology screening by indicating that the relevant substance must have the capability to precipitate proteins.

Key words Gastric mucosa · Duodenal mucosa · Fixation · Preservation · Intoxication · Poisoning · Mercury · Chloralhydrate

Introduction

In legal autopsies suspicion of acute poisoning is often aroused by the presence of non-specific macroscopic findings, e.g. oedema of the brain and lungs, dilatation of the large bowel, dilatation and full distension of the bladder and suspicious odour or typical appearance of gastric contents. In this paper we describe two cases of fatal poisoning involving mercury and chloralhydrate with unusually well preserved gastric and duodenal mucosa.

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Case reports

Case 1

A 40-year-old man was found unconscious in his apartment with no previous history of disease. He was working as a chemist in a laboratory of the metal processing industry. On admission to hospital, ECG revealed a right bundle-branch block and laboratory data showed partial respiratory insufficiency. Despite intensive medical care including mechanical ventilation, the patient died of cardiorespiratory dysfunction 4 h after admission and pulmonary embolism was suspected. An autopsy was carried out 30 h post-mortem and revealed non-specific signs of intoxication including severe oedema of the lung, oedema of the brain with tonsillar herniation, dilatation of the bowel and marked congestion of the parenchymatous organs. The stomach contained 30 ml of a reddish fluid. Between the gastric folds, the mucosa appeared highly preserved with a brownish discoloration, but streak-like erosions in the exposed parts (Fig. 1). The mucosal surface of the oral cavity and oesophagus also appeared brownish discoloured. Histologically, the preserved areas of the gastric mucosa were totally unaffected by autolysis with an intact epithelial layer (Fig. 2), whereas the eroded areas showed loss of mucosal lining with polymorphonuclear granulocytes and lymphocytes (Fig. 3). Mercury was detected in the epithelial layer in-situ (Fig. 4) using 1,5-diphenylcarbazone staining (0.2% in 96% ethanol). Tubular necrosis was present in the kidneys. Toxicological analysis of the blood revealed amitriptylene in therapeutic concentrations (0.07 µg/ml). Atomic absorption spectrometry was employed to identify metallic compounds, revealing lethal mercury poisoning (blood concentration 113.8 µg/ml). Neither toxicological hair analysis nor histology showed evidence of chronic mercury poisoning. The previous history and police investigations failed to elucidate the background of possible suicidal tendencies. However, the detection of amitriptylene strongly suggested a depressive disorder.

Case 2

A 34-year-old male with a previous history of depressive disorder with suicide attempts, was found dead at home. An empty package of chloralhydrate was found at the scene. Autopsy was performed 4 days postmortem. In addition to non-specific signs of intoxication (oedema of brain and lungs, dilatation of the rectum and fully distended bladder), gross examination revealed 200 mL of a pink, fruity smelling liquid in the stomach and pink gelatinous material adherent to the gastric and duodenal mucosa. Histologically, the epithelial cell lining was well preserved with no evidence of autolysis (Figs. 5, 6). Toxicological analysis showed a lethal blood con-

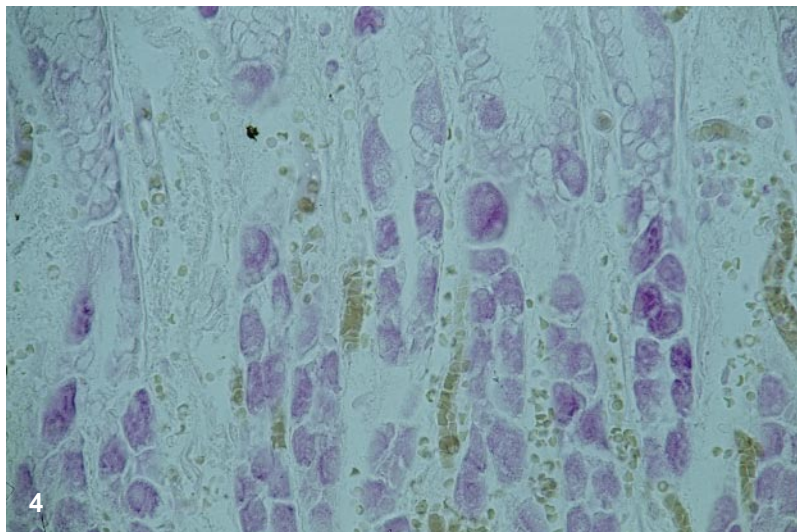
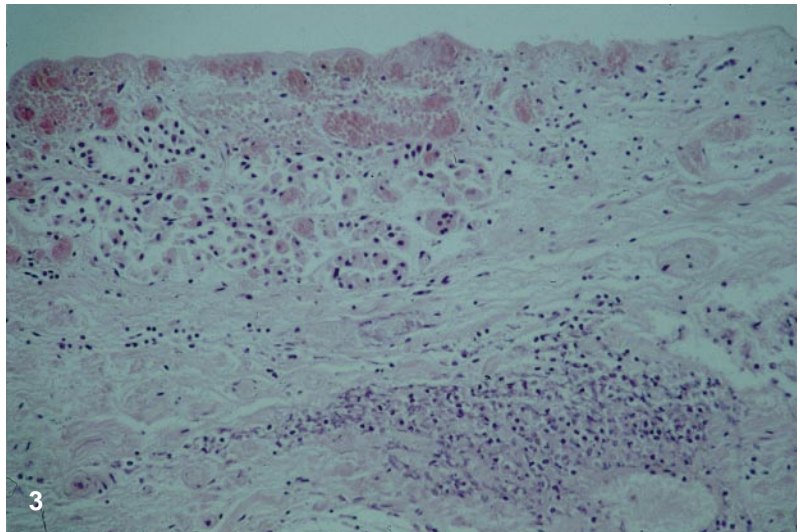
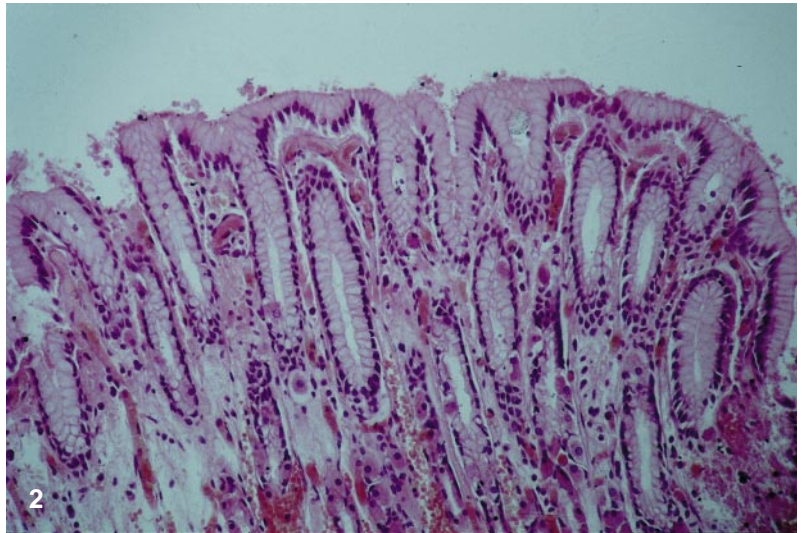


Fig. 1 Case 1: Stomach and distal third of the esophagus. Well preserved and brownish discoloured mucosa between the gastric folds, streak-like erosions in the exposed parts

Fig. 2 Case 1: Gastric mucosa 30 h postmortem unaffected by autolysis with intact epithelial layer (hematoxylin-eosin, $\times 225$)

Fig. 3 Case 1: Gastric mucosa 30 h postmortem. Loss of mucosal lining with hyperaemia, polymorphonuclear granulocytes and lymphocytes (hematoxylin-eosin, $\times 225$)

Fig. 4 Case 1: Detection of mercury in the gastric mucosa (1,5 diphenylcarbazone, $\times 450$)

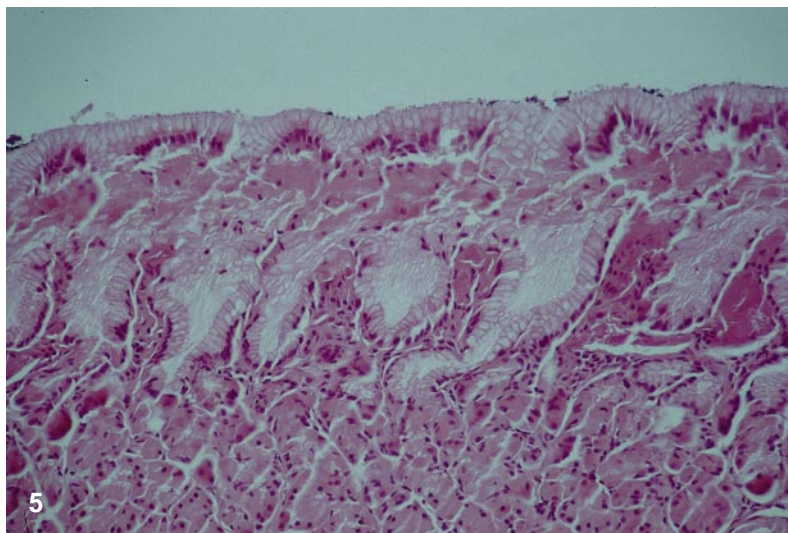


Fig. 5 Case 2: Gastric mucosa 4 days postmortem. Intact epithelial layer (hematoxylin-eosin, $\times 225$)

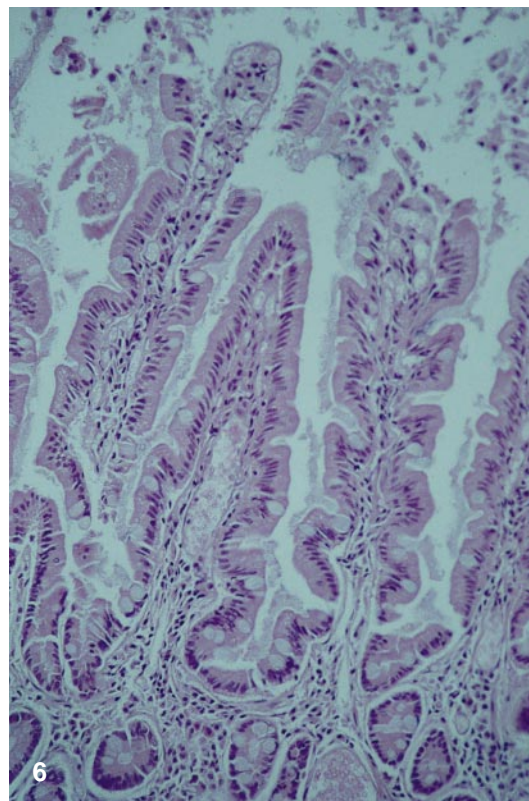
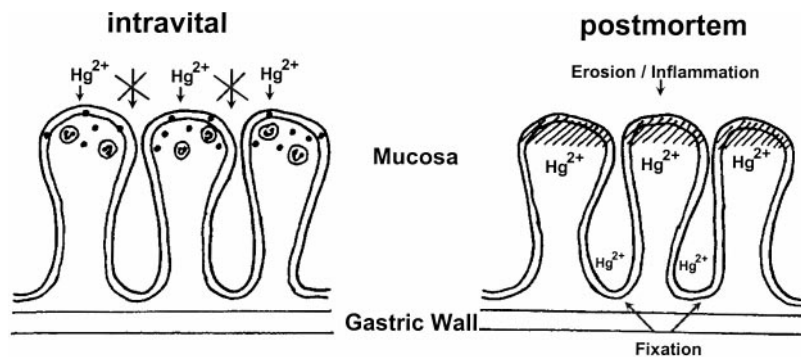


Fig. 6 Case 2: Duodenal mucosa 4 days postmortem. Intact epithelial layer (hematoxylin-eosin, $\times 450$)

Fig. 7 Potential mechanism of mercury fixation of the gastric mucosa



centration of trichloroethanol ($52 \mu\text{g/ml}$), the major active metabolite of the sleeping tablet chloralhydrate.

Discussion

Various alterations of the gastrointestinal mucosa associated with fatal poisoning by orally ingested substances are well known including for example discolouration by chromate compounds [1], oedema and diffuse gastric hemorrhage following mercury oxycyanide ingestion [2], gastritis caused by chloralhydrate overdose and mercuric compounds [3, 4], erosion following benzalkonium chloride poisoning [5] and metaphasic mitotic figures in gastric and small bowel epithelium due to colchicine poisoning [6]. Additionally, suspicious gastric contents, e.g. dark brownish fluid or bloodstained contents in cases involving pentoxifylline, moclobemide and perazine, have been described [7, 8].

The two cases presented here showed an unusually well preserved gastric mucosa despite a postmortem interval of 30 h and 4 days respectively, with corresponding autolysis of the other internal organs.

In case 1 the preservation of the gastric mucosa was most likely due to the capability of mercuric salts to precipitate proteins to albuminate [4]. However, erosive lesions were also present. It seems reasonable to assume that intravital exposure to mercury compounds might have led to an inflammatory reaction of the exposed mucosa, whereas the mucosal lining within the gastric folds might have been protected from the irritant. Therefore, no inflammatory reaction but perimortem or postmortem fixation occurred in these areas (Fig. 7). In 1977 Scheidegger reported on the discovery of medieval grave sites that had been found in churches around the city of Basel, Switzerland [9]. The exhumed bodies were mummified and remarkably well preserved and it was emphasised that the

intestinal tract was in a surprising good condition. Chemical analysis revealed high concentrations of mercury in the internal organs, which led to the diagnosis of mercury poisoning. According to our findings, there might be a direct connection between the preservation of these bodies and the mercury intoxication. A remarkably good preservation of dead bodies has also been described in cases of arsenic poisoning [10].

In case 2 the fixation of gastric and duodenal mucosa can easily be explained by the formalin-like protein-denaturing properties of the aldehyde chloralhydrate. Erosions, ecchymoses, hemorrhages and inflammation of the gastric mucosa due to chloralhydrate poisoning have been previously reported [3, 11]. The absence of inflammatory changes in case 2 suggests that death might have occurred shortly after ingestion of the poison. In addition, the solidified mucus might have functioned as a protective layer against the gastric acid and therefore prevented the onset of autodigestion. Similar phenomena have been described by Lewin [3] in cases of fatal formalin poisoning, that presented a leathery composition of the internal organs and solidified mucus in the nose and pharynx.

The two cases presented here demonstrate that the phenomenon of perimortal fixation is a useful indication for the forensic pathologist. Good preservation of the gastric and duodenal mucosa inconsistent with the duration of the postmortem interval should always direct the suspicion to oral poisoning. Moreover, the detection of the fixation phenomenon facilitates toxicology screening by indicating that the relevant substance must have the capability to precipitate proteins.

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