H74 Death Following Intravenous Administration of Sucralphate

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After attending this presentation, attendees will understand how sucralphate — a drug used orally to prevent gastric ulcerations — can cause severe complications when administered intravenously and how to identify intravascular presence.

This presentation will impact the forensic science community by presenting a case report which serves as an example of the importance of applying unconventional analyses to answer a particular question in routine postmortem casework.

Sucralphate is a drug used for prevention of gastric and duodenal ulcers as well as for prevention of stress ulcerations of the gastric mucosa, particularly in patients under intensive care treatment for burns, multitrauma, neurosurgery, and more. It is administered orally, typically via a gastric tube, and will exert its effect by binding to the mucosa, where it stimulates factors that enhance the gastric defense barrier, thereby protecting against ulcerations. The many aluminum groups on this large sugar molecule are supposed to be crucial for the cytoprotective effect, similar to other antacids. Aluminium sucralphate has been shown not to enter the blood circulation to any significant amount upon oral administration.

An 83-year-old man underwent surgery for aortic valvular stenosis. After the surgery, bleeding from the gastric tube was noticed. Gastroscopy revealed erosive gastritis and it was decided he should receive tranexamic acid and omeprazol as well as four units of erythrocyte concentrate. In addition, aluminium sucralphate (brand name Andapsin®) was ordered in a dose of 2g (10mL)x3. His condition remained stable until the second day, when he suddenly developed bradychardia and experienced shortness of breath. He was taken to the intensive care unit, where he lost consciousness. Pericardiocentesis revealed no blood in the pericadial sac. Shortly thereafter, he became pulseless and stopped breathing. Resuscitation was unsuccessful. Later, a nurse who was new at the clinic and not experienced with all routines, reported that she was unsure if she had administered sucralphate into the gastric tube or into the central venous catheter just before the patient developed bradychardia.

The case was reported to the police because of suspicion of malpractice and a forensic autopsy was performed. Gross macroscopic findings included heart enlargement and heavy lungs, but no blood in the thoracic or abdominal cavities. Samples were taken for toxicology, including a sample from the left jugular vein/superior vena cava at the position of the intravenous catheter. Analysis for sucralphate was requested. The forensic toxicology laboratory explained that this drug would not be possible to identify by their screening method and that no known method existed for verification analysis of such a large molecule as aluminium sucralphate phosphate. Given the large number of aluminium groups present on the drug molecule, analysis for aluminium was considered a possibility since no or very little amount of this drug should be expected to be found in the blood if the drug was given orally. The department of inorganic chemistry at Umea University, Sweden, was contacted and agreed to analyze aluminium levels in the samples. To this end, both femoral and jugular blood samples were submitted from this patient, as well as blood samples from an additional two patients who later died at the same intensive care unit, although by known causes. One of them had also been administered sucaralphate. An empty test tube was also sent for analysis. An accredited method for analysis of aluminium using graphite-oven atomic absorption spectroscopy was performed. The results are shown in the table below.

Case	Al conc (mcg/L)	Sucralphate dose
Present case, jugular blood	8800	2g x 3
Present case, femoral blood	2100	2g x 3
Control patient #1	30	1g x 4
Control patient #2	10	No
Empty test tube	2	No

Microscopy included immunohistochemistry with an anti-fibrinogen antibody, 1:500, and with Vulcan Red as a chromogen. Widespread, intravascular, well-developed networks of fibrin, capturing large numbers of polymorphonuclear cells, typical of extensive micro-embolism, were seen in samples from the lungs and kidneys.

Conclusion: The intravascular microthrombi are an expected finding upon binding of sucralphate to fibrinogen, promoting its aggregation. The extreme increase in aluminum levels in the blood of the patient along with the presence of microthrombi strongly supported the notion that sucralphate indeed had been given intravenously.

Antacid, Intravascular Coagulation, Malpractice