



Suicidal intoxication by copper sulfate

J. FONTE SANTA ; B. SILVA ; R. MATOS

Instituto Nacional de Medicina Legal – Delegação do Centro

Portugal

Introduction

Portugal is traditionally an agricultural country, consequently intoxications by pesticides are very common, especially with paraquat and organophosphorous. Copper sulfate is a fungicide used to control bacterial and fungal diseases of fruit, vegetables, nut and field crops. It can also be applied in water treatment systems to control algae. This pesticide is available as dust, wettable powder and liquid concentrate. In Portugal its major application is in vine plantation.

Copper sulfate solutions may irritate eyes, skin, respiratory and mucous membranes. Poisoning by this compound may affect the central nervous system, liver, kidneys and capillaries, causing frequently renal failure and haemolytic anaemia.

Despite Portugal's major wine production, intoxications (accidental, suicidal or homicidal) with this compound are relatively rare, as can be seen in the few cases described in forensic literature.



Case Report

An 81-year-old male, diabetic, with chronic renal failure, was admitted at the hospital with a suspicion of voluntary ingestion of copper sulfate. Four days later he died. He was admitted in the Nephrology unit with acute renal failure and anaemia, being treated with intensive hemodialysis and blood transfusions. Later on, he developed metabolic acidosis and there was a worsening of anaemia. During admittance it was detected a 1,5 mg/L copper concentration in blood.

At the autopsy it was possible to see a green colouration of the nails (Figure A), ascite and pleural effusion on the right side. Samples from heart, kidneys, lungs and liver were taken for histopathological examination. The major microscopic changes were: bilateral severe lesions of chronic pyelonephritis, renal arterioarteriolar sclerosis, toxic acute renal damage with vacuolar degeneration of proximal tubular epithelial cytoplasm and frank necrosis of distal convoluted tubules with nuclear pyknosis and desquamation of hiper eosinophilic epithelial cells (Figures B, C); acute hepatic damage with cholestasis (bilirubinostasis) and cytotoxic injury, sinusoidal lymphocytosis with spillover into nearly parenchyma and spotty, perivenular necrosis of hepatocytes (Figures D, E).

Post-mortem it wasn't possible to determine the presence of copper in blood and organs due to technical problems with the atomic absorption spectrophotometer.

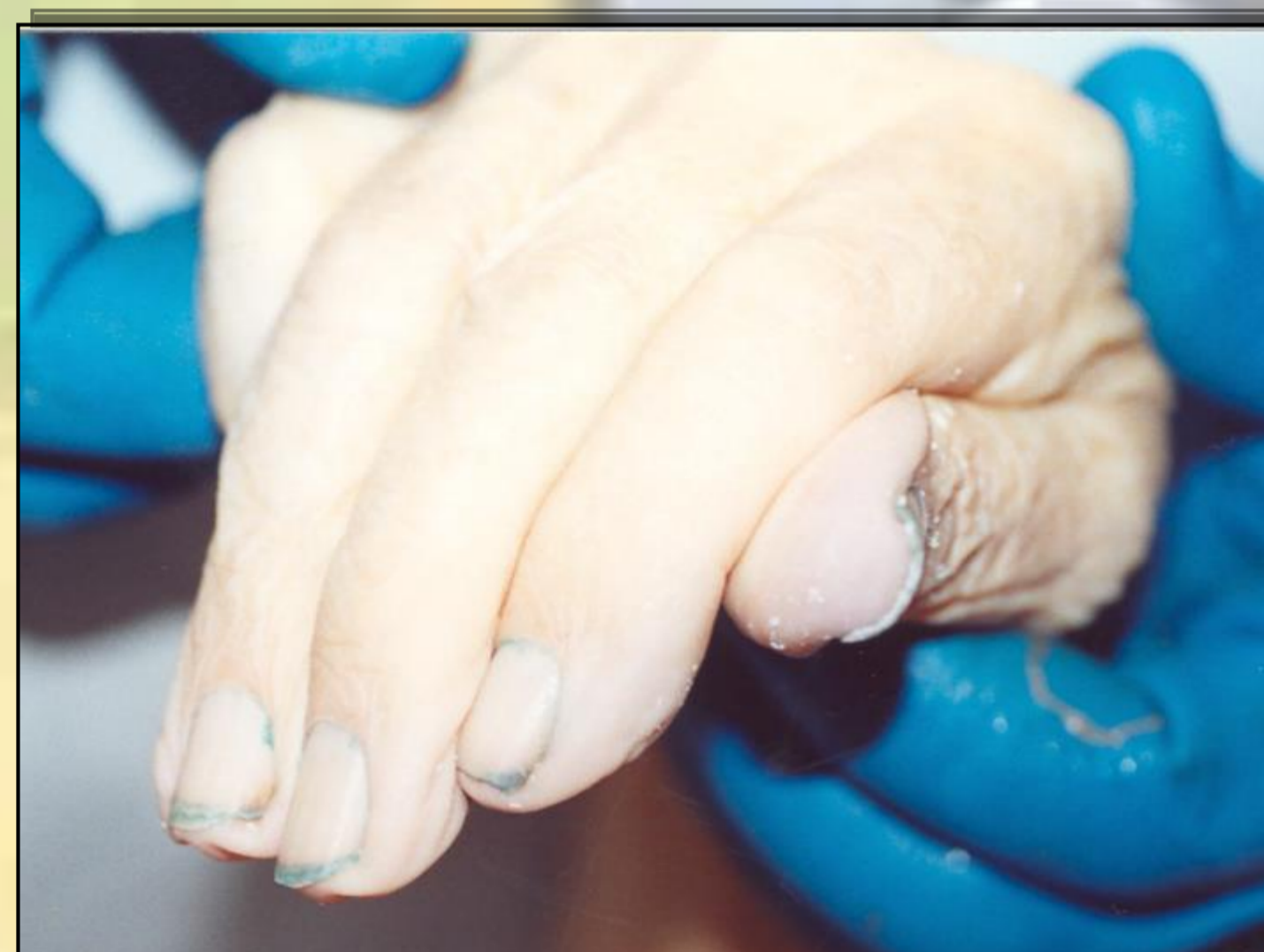


Figure A

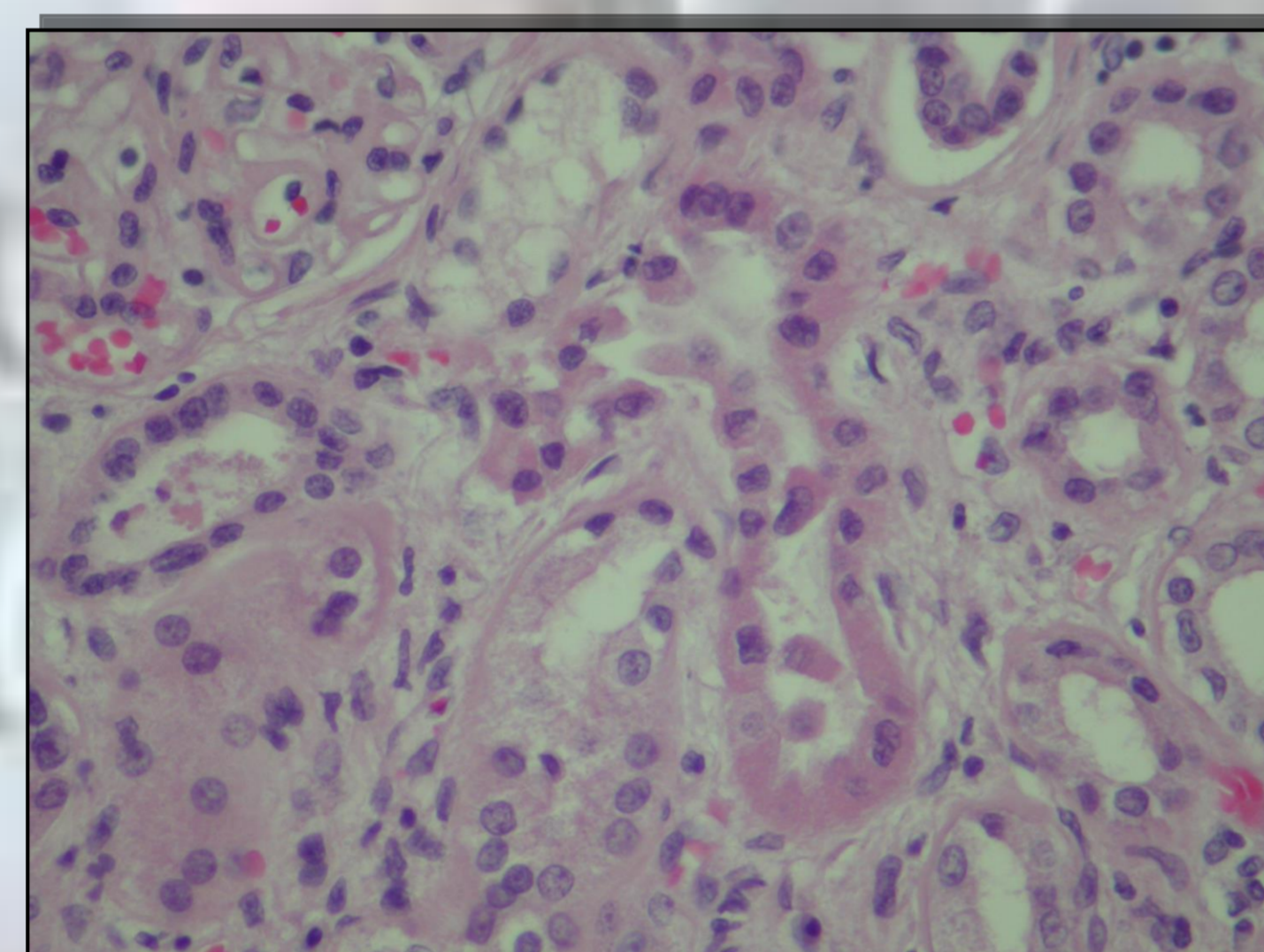


Figure B

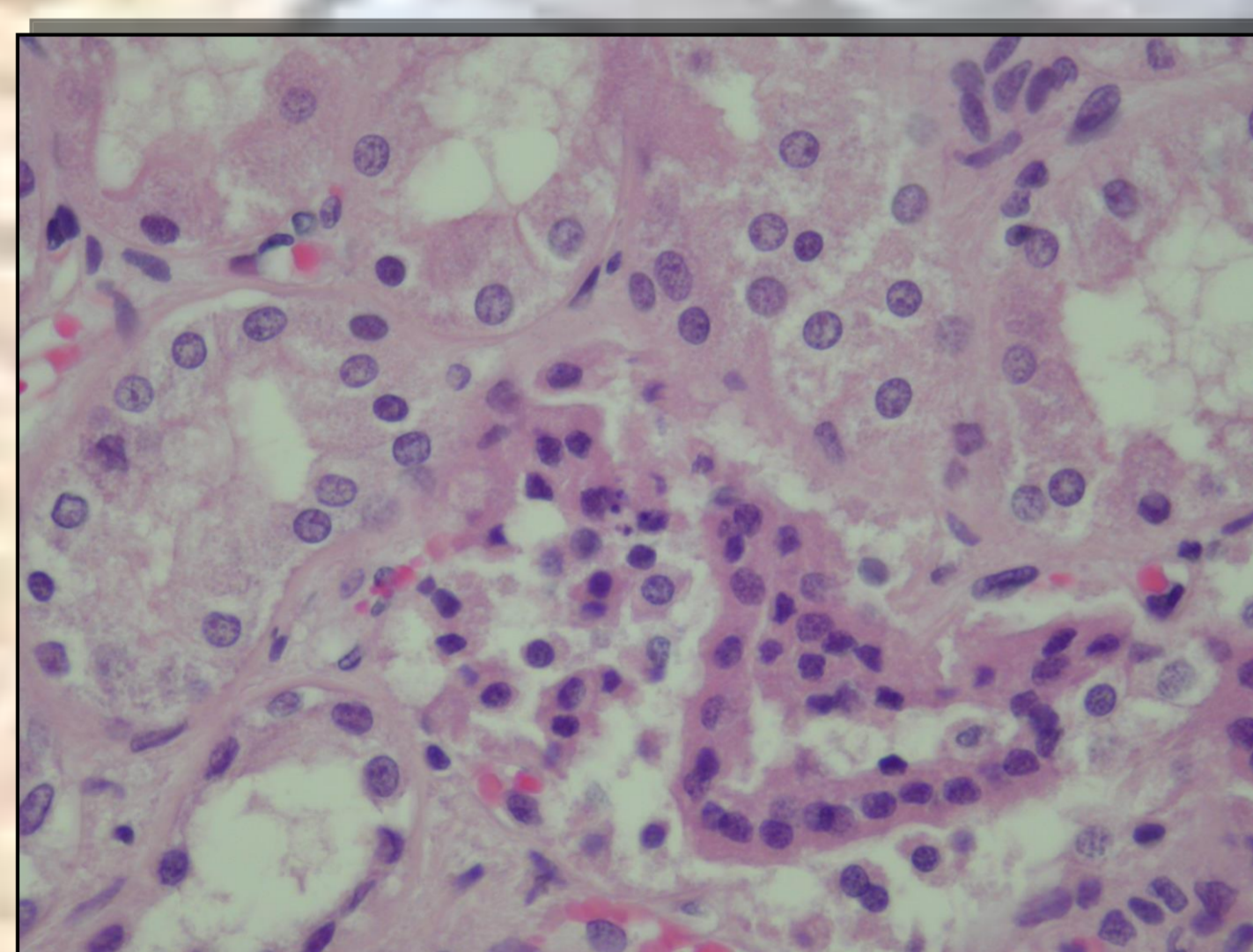


Figure C

Discussion

In this case there was suggestive clinical features of an acute intoxication by copper sulfate: renal failure, anaemia and a high copper concentration in blood (being the normal concentration of 1 mg/L). Nevertheless, autopsy findings weren't significant, the most common features like gastric and esophagic erythema or ulceration were absent. Once spectrophotometric determination of copper in autopsy material wasn't possible, the diagnosis was based on the microscopic alterations. In fact, there was evidence of renal histologic changes related with diabetes and chronic renal failure (arterioarteriolar sclerosis, chronic pyelonephritis), but also lesions compatible with copper sulfate poisoning: tubular degeneration of the renal proximal and distal convoluted tubules. Furthermore, the histopathological lesions observed in the liver were another clue to determinate the diagnosis.

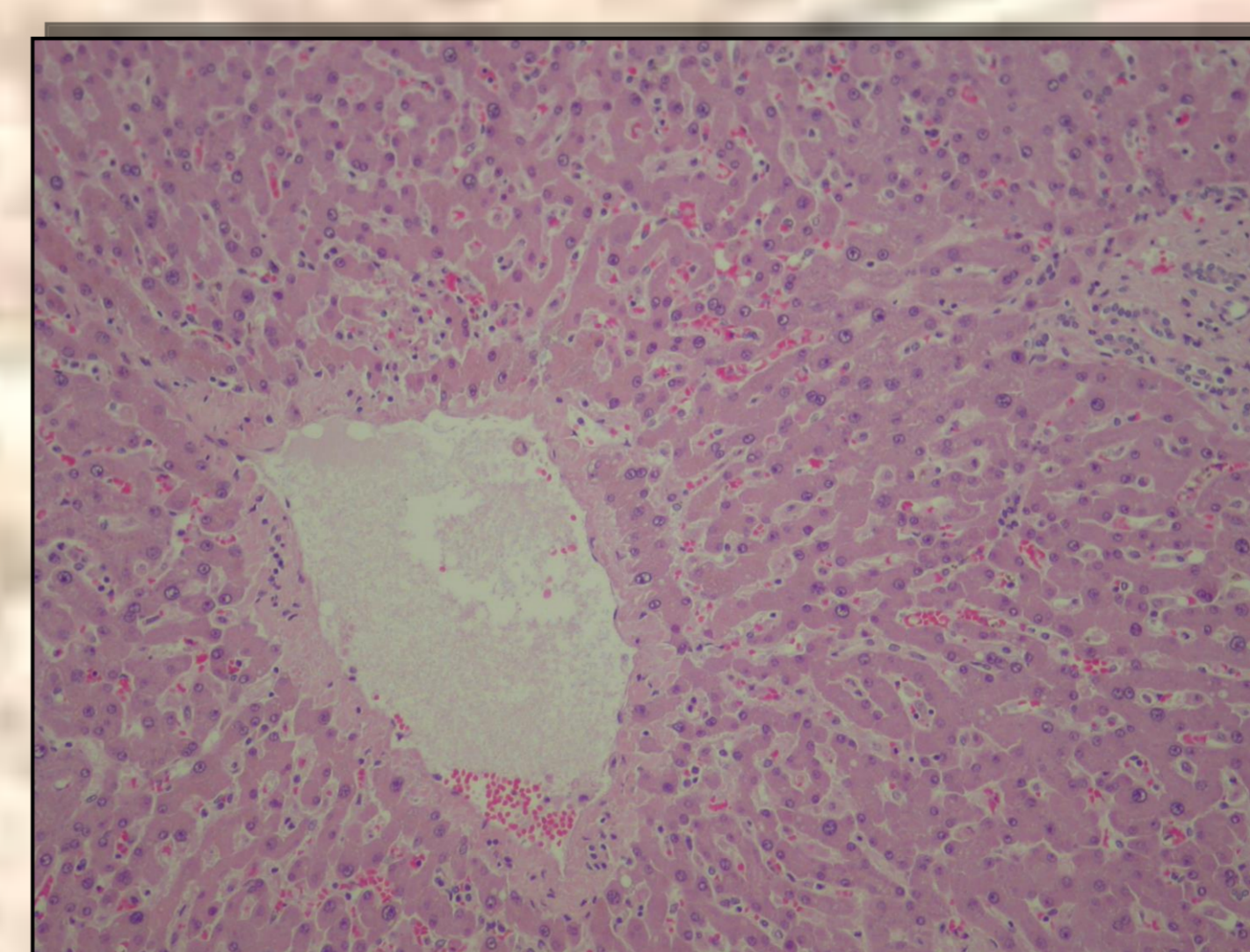


Figure D

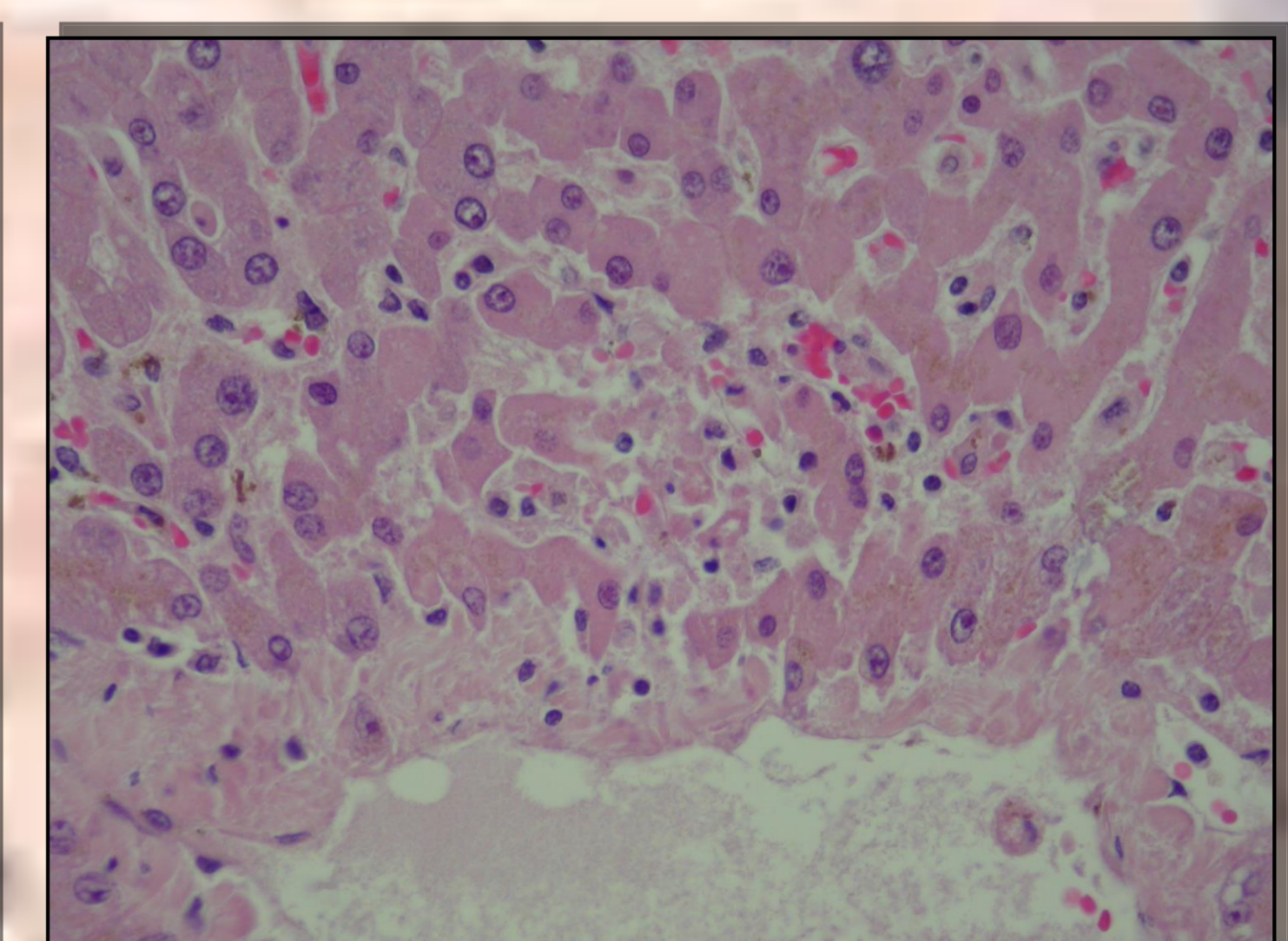


Figure E

References:

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