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Ammonium dichromate poisoning: A case report

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ABSTRACT

Death due to ammonium dichromate has been rarely reported in medical literature. On ingestion, initially there is a relative lack of severe symptoms and signs. Hence, there is usually some delay before the affected person seeks medical attention. Ammonium dichromate is however quite toxic, and death occurs due to multi-organ failure in substantial ingestions. Anuria is often the earliest manifestation of serious systemic toxicity. Screen-printing and coloring works are the usual sources of exposure in dichromate poisoning. The diagnosis of dichromate poisoning depends mainly on the history, and clinical and postmortem findings. Chemical analysis utilizing conventional equipment may not be helpful.

Keywords: Ammonium dichromate, Anuria, Multi-organ failure

Drug Index Terms

Ammonia - Drug Toxicity; Chromium Derivative - Drug Toxicity; Dichromate Ammonium - Drug Toxicity; Unclassified Drug

Medical Index Terms

Intoxication - Diagnosis; Intoxication - Etiology; Medical Literature; Ingestion; Disease Severity; Lethality; Cause Of Death; Multiple Organ Failure; Anuria; Occupational Exposure; Anamnesis; Clinical Feature; Autopsy; Chemical Analysis; Human; Male; Case Report; School Child; Article

EMBASE Classifications

052 - Toxicology

CAS Registry Numbers

ammonia, 14798-03-9, 51847-23-5, 7664-41-7; dichromate ammonium, 7789-09-5

Introduction

Chromium compounds occur in valence forms +1, +2, +3, +4, +5, and +6, but the +3 (trivalent) and +6 (hexavalent) forms are the most important[1]. Hexavalent chromium compounds, incorrectly called chromates (unless they exist as $CrO_3^{(-2)}$ ions), usually have a yellow pigment, and include chromium trioxide (CrO_3 ; chromium VI oxide), the anhydride of chromic acid, chromates, dichromates, and polychromates.

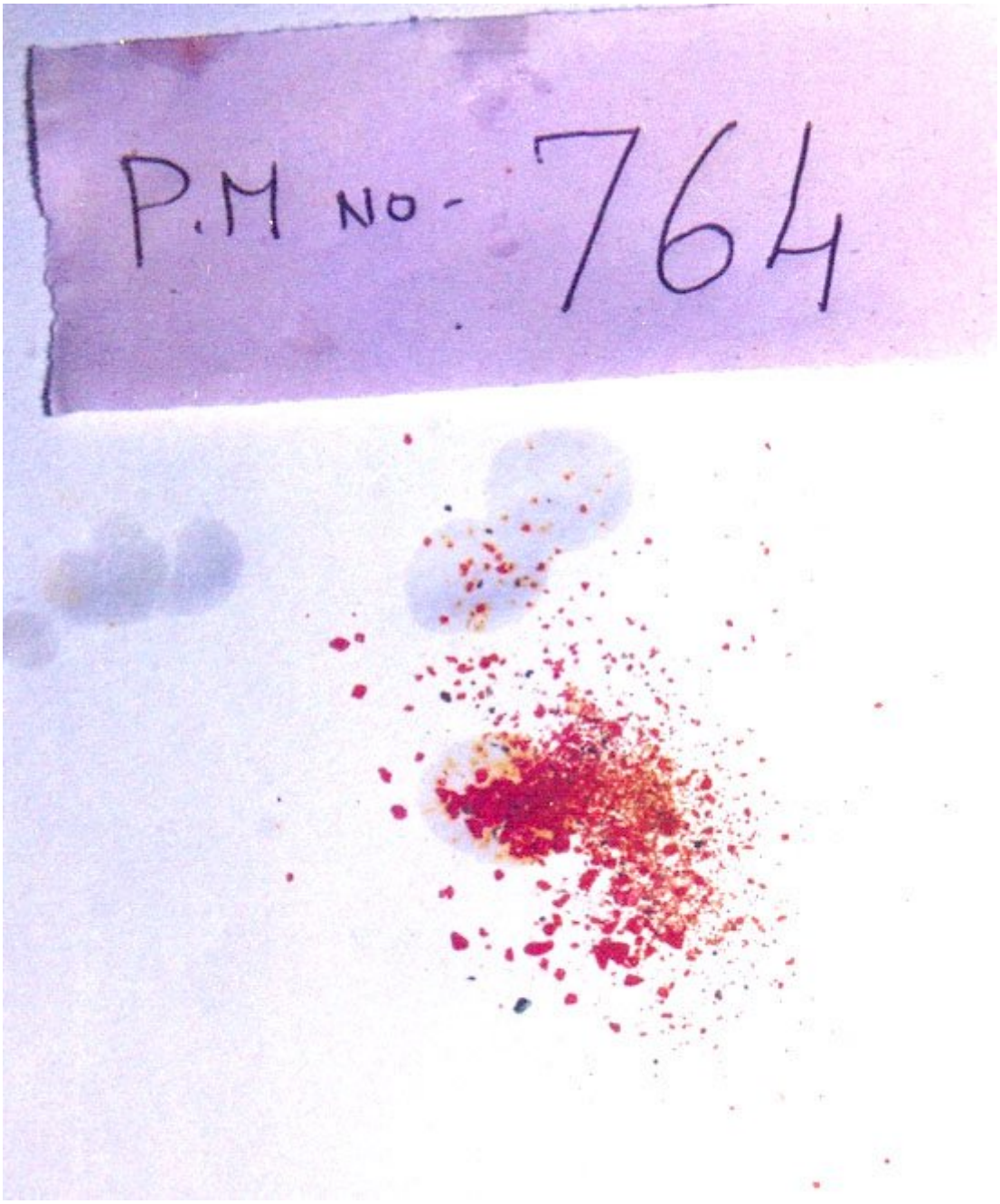


Figure 1. Ammonium dichromate crystals

These are generally the most toxic chromium compounds, particularly the water-insoluble forms. Water-soluble hexavalent chromium compounds include chromic acid, its anhydride, and monochromates and dichromates of sodium, potassium, ammonium, lithium, cesium, and rubidium. Water-insoluble hexavalent chromium compounds include zinc chromate, calcium chromate, lead chromate, barium chromate, strontium chromate, and sintered chromium trioxide.

The safety of cosmetic products is very important because of their widespread use. Today, cosmetic products find an inalienable place in the normal everyday life of most people for grooming various parts of their body such as hair, skin, and teeth. Although manufacturers of cosmetics generally take care to ensure continued safety in the use of these products, it is not possible to ensure zero risk or absolute safety for any product. Finished cosmetic products are only tested on animals if the new formulation significantly differs from previous preparations. Moreover, it is largely restricted to the investigation of effects on skin and mucous membranes.

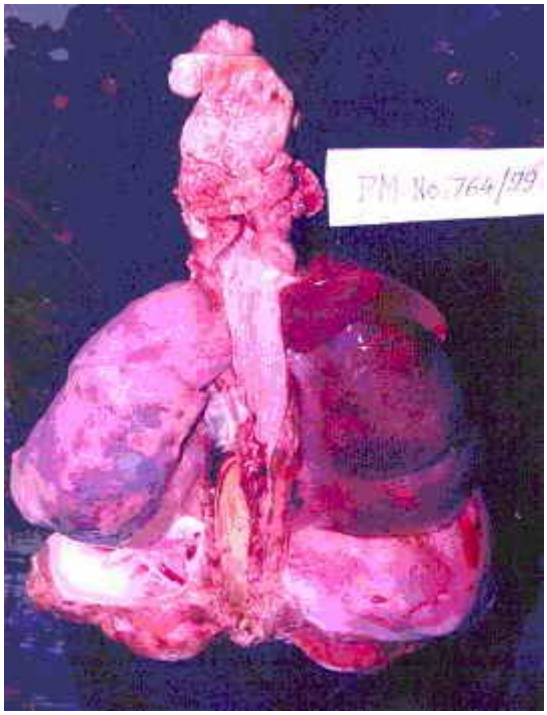


Figure 2. Corrosion of Esophagus

Acute poisoning is likely to occur through the oral route, whereas chronic poisoning is mainly from inhalation or skin contact. Death due to poisoning with ammonium dichromate is rarely reported in medical literature. Ammonium dichromate is used in printing and painting works. These works are becoming more and more popular. Hence the availability of dichromate is likely to become easier, which can increase the incidence of accidental and suicidal exposure. The problem with ammonium dichromate poisoning is that in the initial stages there are no major manifestations, and therefore the patient may delay seeking medical attention until it is too late. If such cases are not treated properly from the beginning all major organs are likely to be involved leading to multi-organ failure.

Case History

An eight-year-old boy consumed a small amount of ammonium dichromate mistaking it for "Rasna"- a soft drink concentrate (**Fig.1)** . A short time thereafter he suffered from diarrhea and vomiting, for which symptomatic treatment was given by a physician from a local hospital. However, on the 3rd day following exposure, the boy developed anuria for which he was admitted at a major private hospital. Renal failure was diagnosed and peritoneal dialysis was carried out. But the condition of the boy worsened and he died on the 4th day after ingestion of the poison.

Postmortem Findings

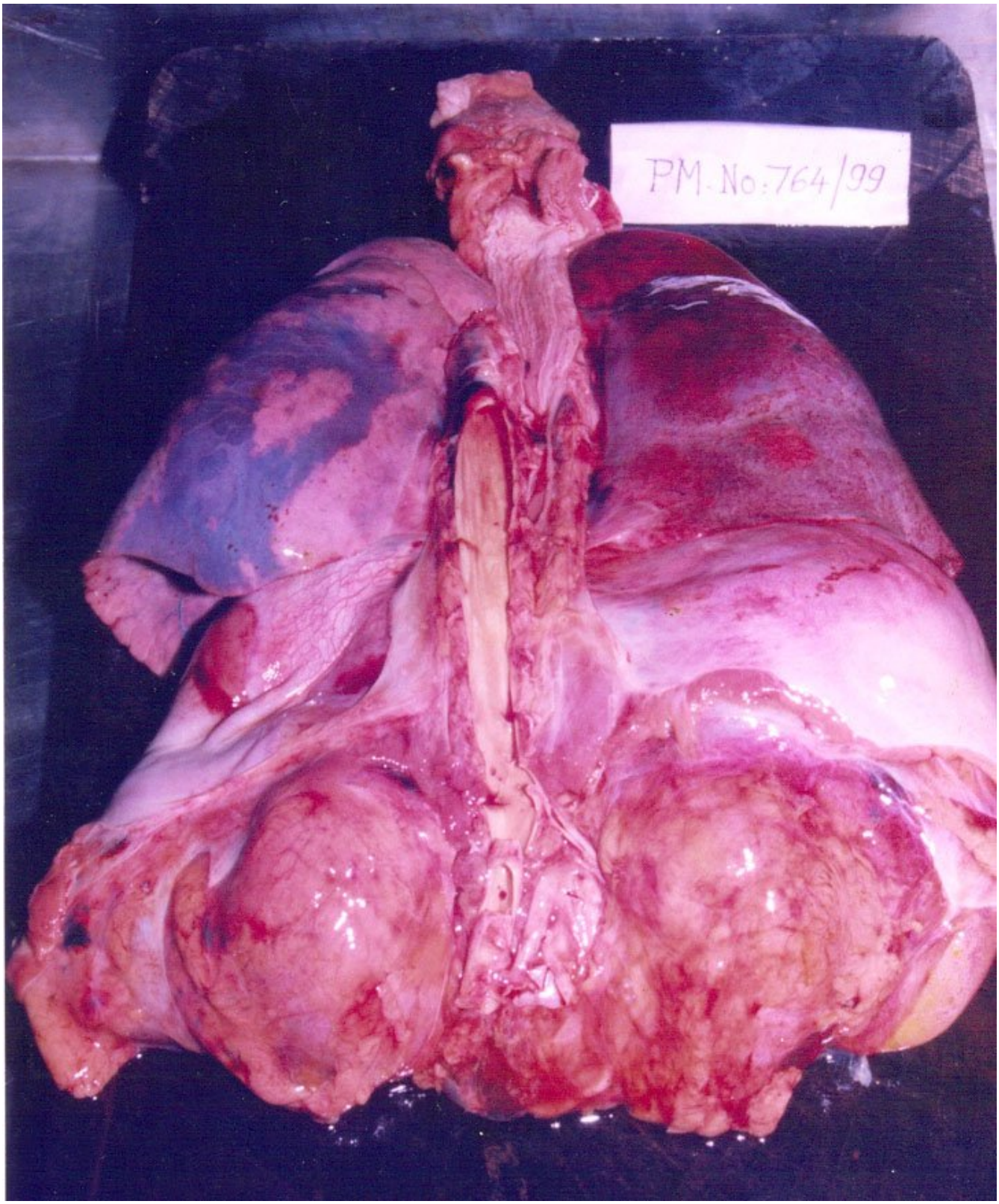


Figure 3. Petechiae and ecchymoses over lungs

On postmortem examination the entire body appeared edematous externally. Internal examination revealed the brain to be congested with mild edema. The esophagus showed corrosion (**Fig.2**). Pleural cavities contained bloodstained fluid. Multiple petechial hemorrhages with ecchymoses were seen on both lung surfaces; they were however more intense over the right lung (**Fig.3**). Both

lungs were congested and edematous. Petechial hemorrhages were also present on the epicardial surface of the heart. Liver appeared yellowish (**Fig.4**). Kidneys were enlarged and pale with red streaks in the narrowed cortex (**Fig.5**). Stomach contained reddish viscous fluid without any unusual smell (**Fig.6**). The mucosa was quite congested.

Histopathological examination of the lungs revealed pneumonic changes (**Fig.7 & Fig.8**). Kidneys showed acute tubular necrosis mainly involving the proximal convoluted tubules (**Fig.9**), while the liver demonstrated evidence of fatty change with peripheral necrosis (**Fig.10**). Other organs were congested, but otherwise normal.

Selected viscera including stomach, liver, and kidneys, and blood were sent for chemical analysis. But no chemicals or toxins were detected.

Discussion

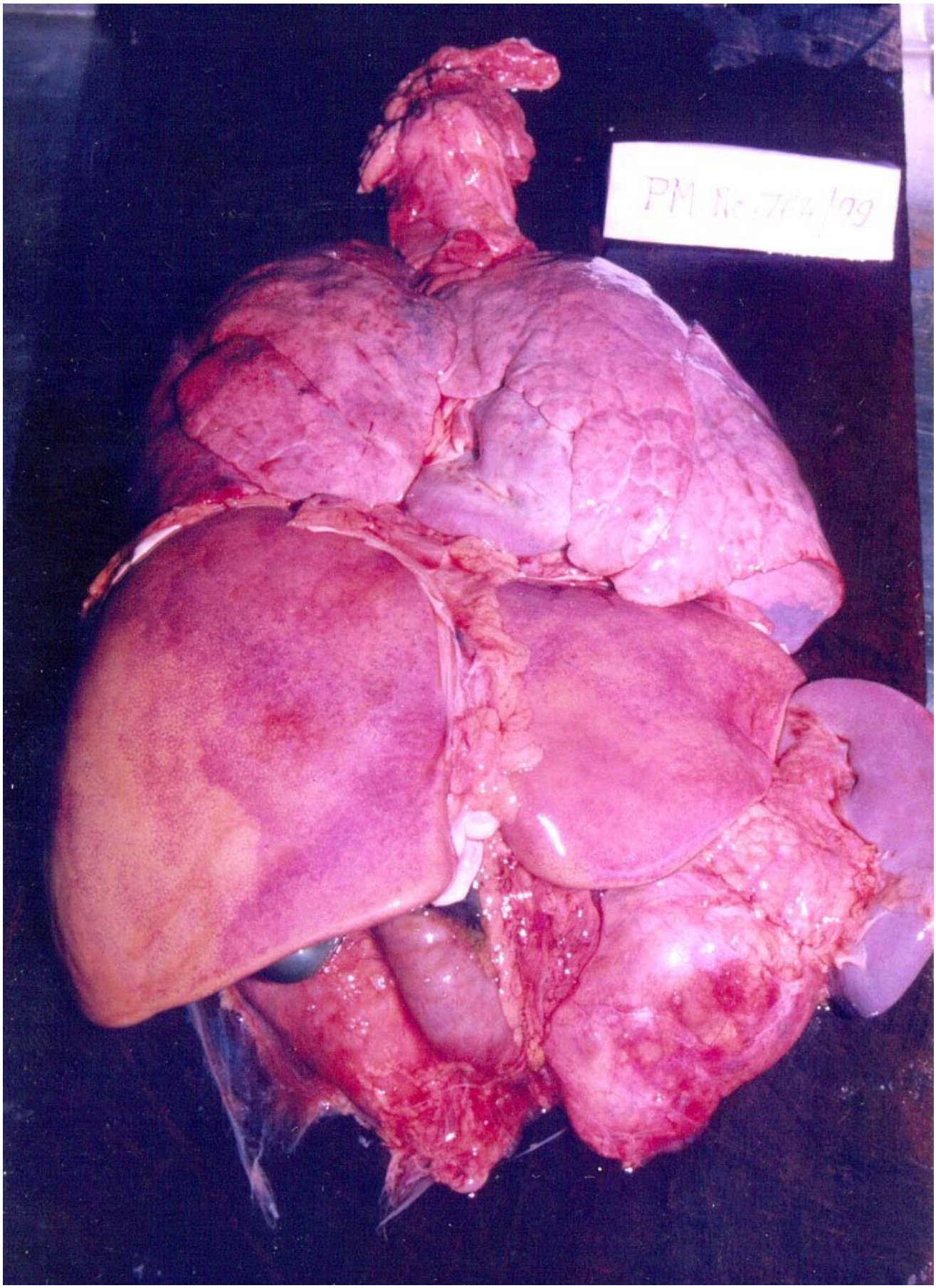


Figure 4. Yellowish discoloration of liver

Ammonium dichromate [NH_4 (Cr_2 O_7)] is a rarely encountered chemical in clinical toxicology. It is a red crystalline solid, and is commonly used as a pigment or dye. In undiluted form, ammonium dichromate acts as a corrosive and systemic poison. Oral intake is reported to cause intense gastrointestinal irritation or ulceration and corrosion, epigastric pain, nausea, vomiting, diarrhea, vertigo, fever, muscle cramps, hemorrhagic diathesis, toxic nephritis, renal failure, intravascular hemolysis, circulatory collapse, peripheral vascular collapse, liver damage, acute multisystem shock, coma, and even death, depending on the dose. In this particular case, there is evidence of initial gastrointestinal irritation in the form of vomiting and diarrhea, but subsequently, the manifestations were mainly renal in nature.

It is known that acute poisoning by ammonium dichromate and other soluble hexavalent salts usually results in local tissue necrosis and severe kidney damage. Acute toxicity after ingestion is a result of GI bleed more so than of systemic poisoning. Violent gastroenteritis with rice-water stools, yellow-green or coffee-ground emesis, corrosive burns of the mouth, esophagus, and gastrointestinal tract, and hemorrhage have occurred shortly after oral ingestion of chromates[2-5].



Figure 5. Degenerative changes in the kidneys (cut section)

Large doses of chromates induce albuminuria with desquamated cells, hyperemia, fatty degeneration, and necrosis in the kidney. While circulatory collapse and shock are frequently reported following overdose6, they were not evident in this case. Respiratory distress syndrome (ARDS) may be noted with significant oral ingestion[7], but was again not significant in this patient. Hepatic encephalopathy and cerebral edema were also not features noted by the physicians attending to the case, but postmortem examination revealed evidence of both brain and liver damage, apart from non-specific signs of lung injury. Meert et al (1994) report the death of a child following a 1gram ingestion of ammonium dichromate[8]. At autopsy, the brain appeared edematous with scattered areas of neuronal shrinkage and pyknosis. There was also ulceration and hemorrhage of the esophagus, stomach, duodenum, and jejunum mucosal areas, while in the case being reported there was only gastric congestion. The esophagus however did demonstrate frank evidence of corrosion.



Figure 6. Appearance and contents of stomach

Since ammonium dichromate is a highly dissociable compound, it is not easily demonstrable by chemical analysis of viscera or body fluids. In this case, the chemical analysis report was negative.

Conclusion

Screen-printing and coloring work is becoming more and more popular in India of late, and hence the availability of dichromate pigments such as ammonium dichromate is likely to get

progressively easier. So the incidence of poisoning is expected to rise in the years to come. In the case being reported, there was a time lag in beginning proper treatment because the attending doctors were not aware about the exact toxic profile of the chemical. It is imperative to heighten awareness of medical professionals about the toxicity of chromates in general, and ammonium dichromate in particular so that fatalities can be minimized. This is all the more important when one considers the fact that diagnosis of chromate poisoning depends mainly on the history and clinical and postmortem findings. Chemical analysis is usually not helpful.

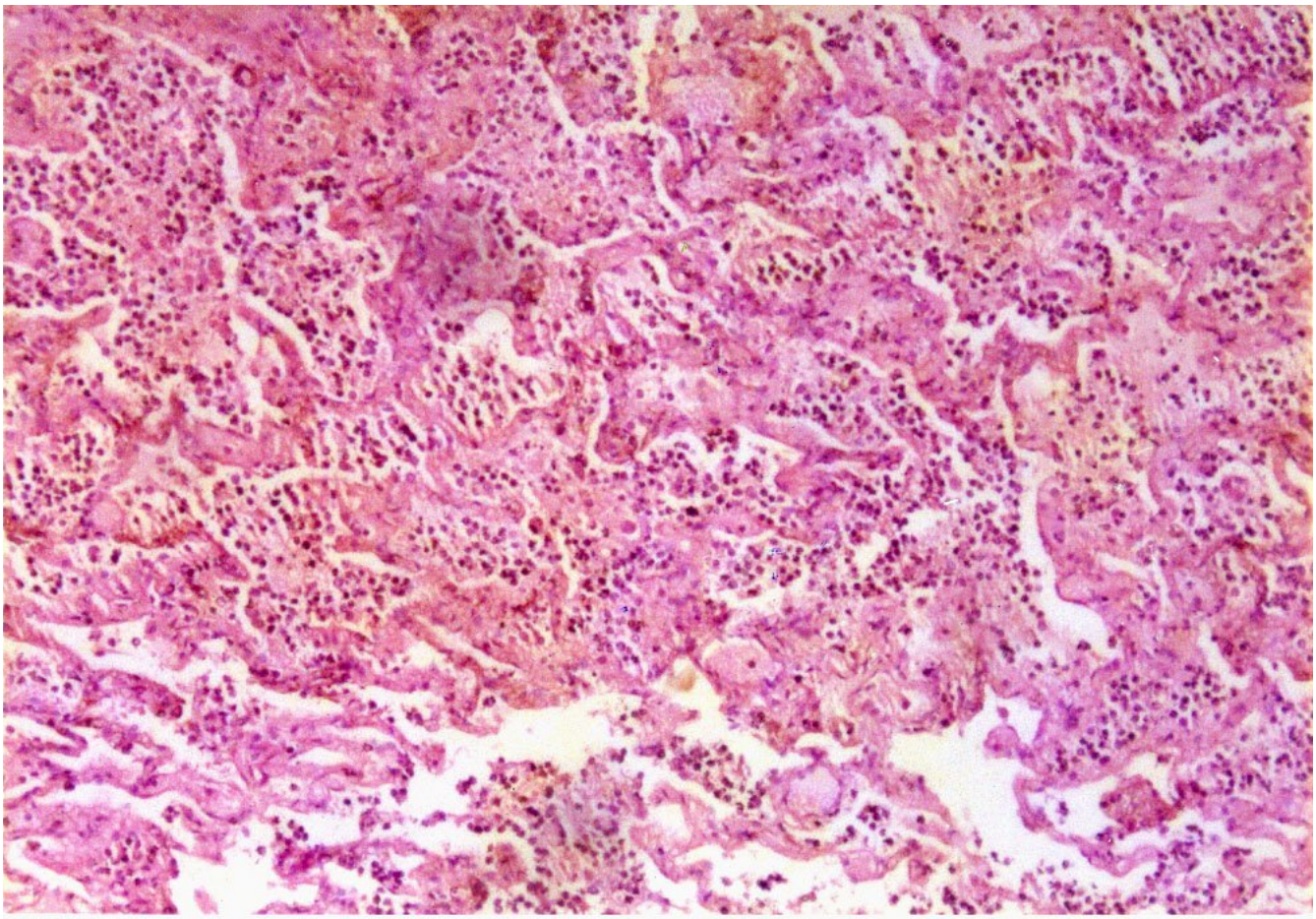


Figure 7. Low power photomicrograph of Pneumonic Lung

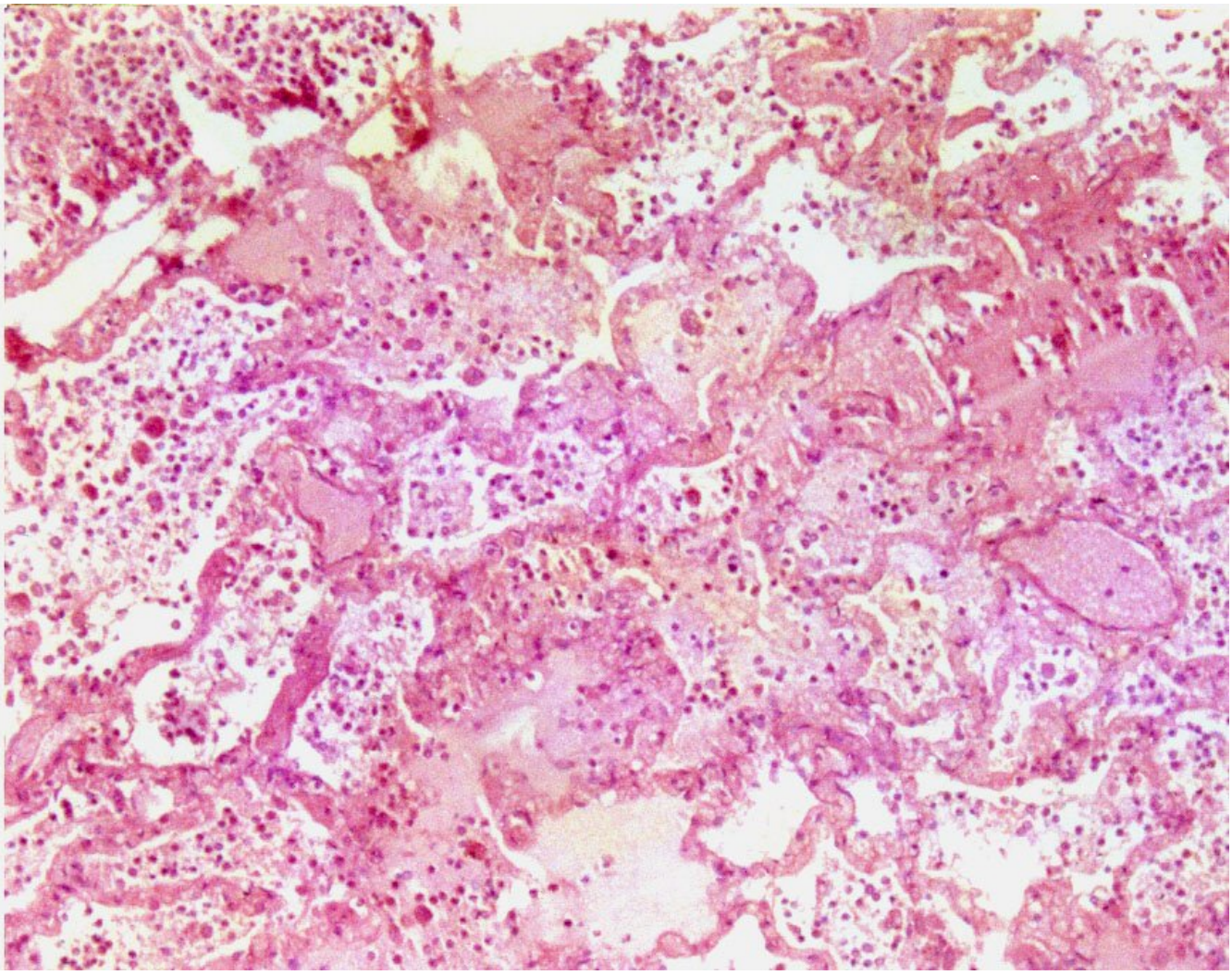


Figure 8. High power photomicrograph of Pneumonic Lung

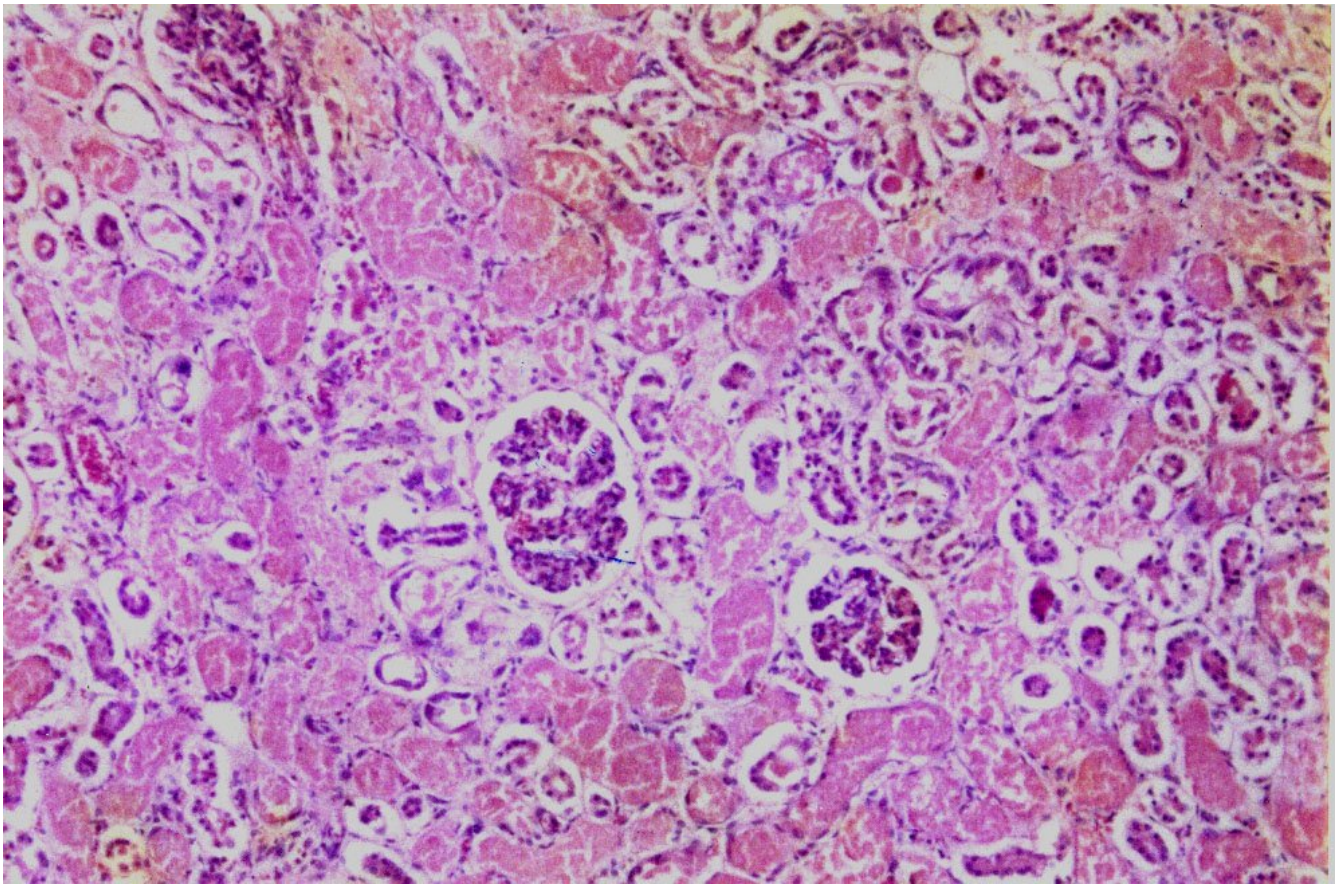


Figure 9. Photomicrograph showing Renal Tubular Necrosis

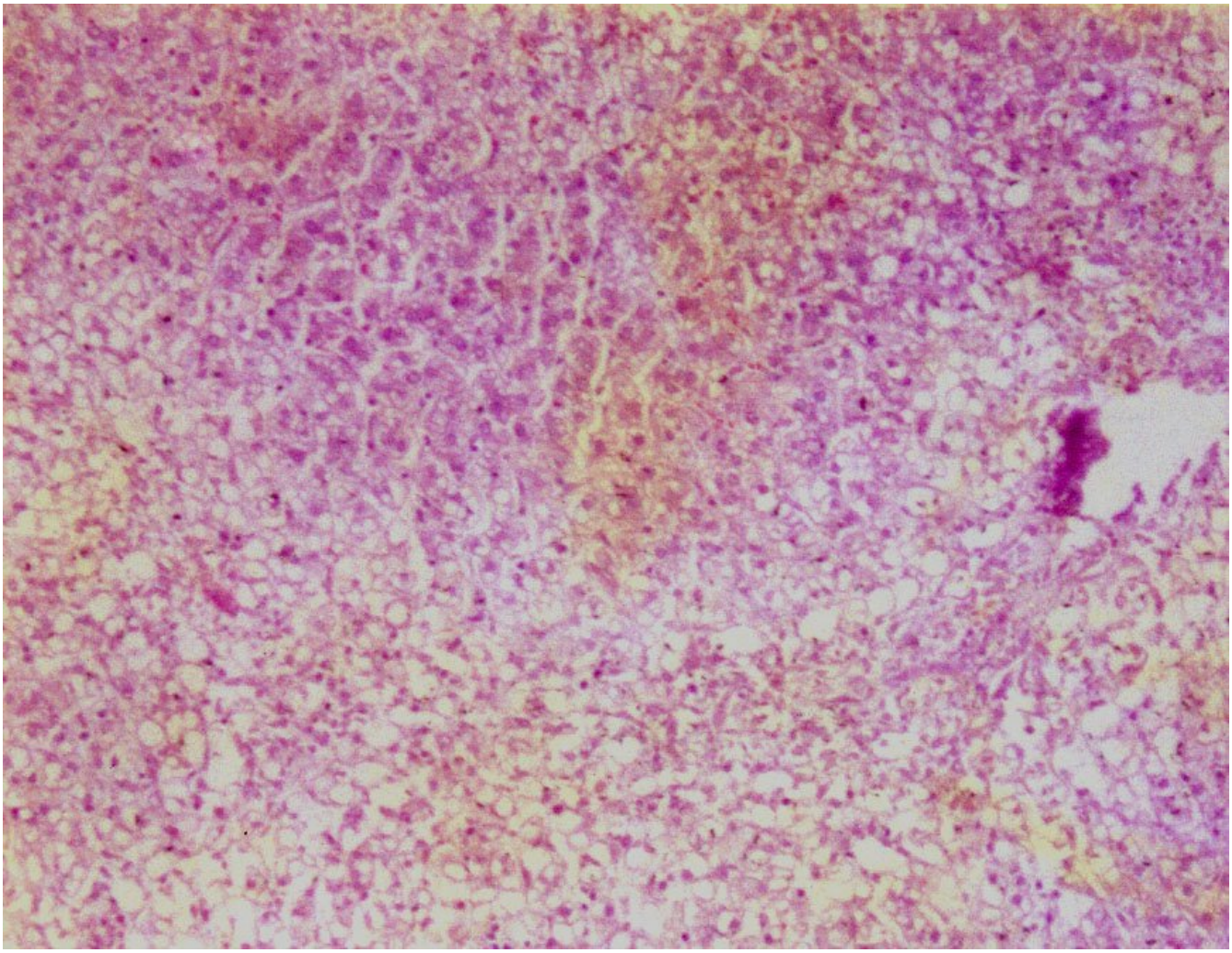


Figure 10. Photomicrograph of Liver with fatty change and necrosis

References

1. Clayton GD, Clayton FE. Patty's Industrial Hygiene and Toxicology, Vol 2C, Toxicology, 4th ed, John Wiley & Sons, New York, NY, 1994
2. Michie CA, Hayhurst M, Knobel GJ, Stokol JM, Hensley B. Poisoning with a traditional remedy containing potassium dichromate. Hum Exp Toxicol. 1991 Mar;10(2):129-31 PubMed
3. Stift A, Friedl J, Laengle F. Liver transplantation for potassium dichromate poisoning (letter). N Eng J Med 1998; 338:766-767 PubMed
4. Kolacinski Z, Kostrzewski P, Kruszewska S, Razniewska G, Mielczarska J. Acute potassium dichromate poisoning: a toxicokinetic case study. J Toxicol Clin Toxicol. 1999;37(6):785-91 PubMed
5. Stift A, Friedl J, Langle F, Berlakovich G, Steininger R, Muhlbacher F. Successful treatment of a patient suffering from severe acute potassium dichromate poisoning with liver transplantation. Transplantation 2000; 69:2454-2455 PubMed
6. Loubieres Y, de Lassence A, Bernier M, Vieillard-Baron A, Schmitt JM, Page B et al. Acute, fatal, oral chromic acid poisoning. J Toxicol Clin Toxicol. 1999;37(3):333-6 PubMed
7. Hay E, Derazon H, Eisenberg Y, Natalia B. Suicide by ingestion of a CCA wood preservative. J Emerg Med. 2000;19(2):159-163 PubMed
8. Meert KL, Ellis J, Aronow R, Perrin E. Acute ammonium dichromate poisoning. Ann Emerg Med. 1994;24(4):748-750 PubMed