

CASE REPORT

GASTROENTERITIS EXHUMED

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Nature of presenting symptoms and even signs of disease can lead to a diagnosis that seems routine but is in fact erroneous because a sufficient index of suspicion is not generated in the mind of the physician dealing with the case. A young girl of about 16 years was brought to the Casualty Department, DHQ Hospital Bannu on 12 Sep 2004 with complains of severe vomiting and diarrhoea; the casualty medical officer diagnosed her as a case of acute gastroenteritis.

INTRODUCTION

It can happen that the nature of presenting symptoms and even signs of disease can lead to a diagnosis that seems routine but is in fact erroneous because a sufficient index of suspicion is not generated in the mind of the physician dealing with the case. Such a scenario may happen in the setting of an Out Patients Department or in the ward, but is more likely to occur in a busy emergency or casualty department. We report a case which met a fatal outcome that could have been averted given the proper index of suspicion.

CASE REPORT

A young girl of about 16 years was brought to the Casualty Department, DHQ Hospital Bannu on 12 Sep 2004 with complains of severe vomiting and diarrhoea; the casualty medical officer diagnosed her as a case of acute gastroenteritis. During treatment, she remained delirious and despite all attempts at treatment, she did not survive and died after half an hour. She was buried in Bannu.

On 30 Sep 2004, the father of the deceased girl reported the matter to the police, making the statement that his daughter had not died of natural causes, but according to her statement made to him, she had been forced to drink a cup of tea by her husband and parents-in-law. They also informed the father that his daughter was seriously ill; he went to their house to inquire about her health and while she was conscious she made the above statement to her father. The father was forced to remain in custody in the house by parents-in-law, while the daughter was taken to hospital. After her death, she was buried and the parents-in-law kept the father confined to their house, so that he could not lodge any complaint. Some tribal elders later rescued the father after hearing the death of his daughter and only then did he lodge the complaint.

Accordingly, an FIR was lodged with the Police on 30 Sep 2004 under Mod No. 15. The judicial magistrate to whom the case was referred ordered the exhumation of the deceased; the department of Forensic Medicine of Khyber Medical

College Peshawar was requested to perform the exhumation. The case was subsequently referred to Gomal Medical College DI Khan.

Exhumation was performed on 7 Apr 2005. Exhumation revealed the mummified body of a young female buried only in shroud without any coffin. The body was taken to DHQ Hospital Bannu for post mortem examination. Routine samples were taken for chemical examination from the stomach, lungs, hair, liver, uterus and kidneys in different bottles; samples of soil from above, below and sides of the body were also taken. Control samples in preservative (concentrated saline solution) were also sent for analysis.

Reports from Chemical Examiner Lab, Forensic Medicine and Toxicology Department Khyber Medical College Peshawar dated 19 Apr 2005 revealed ingested poison as the cause of death; the poison in question was identified as arsenic and acute arsenic poisoning was the reason for both the presentation as acute gastroenteritis and death.

DISCUSSION

This case has some very unusual features, particularly with reference to presentation and total lack of suspicion on the part of medical officer and all other concerned people. It was only by a chance remark by deceased to her father that was taken seriously after persuaded efforts of the father that brought about exhumation and the final diagnosis.

Acute arsenic poisoning may present as acute gastroenteritis often confused with cholera. Most cases are diagnosed at autopsy, because of a velvety appearance of the gastric mucosa.

Famous Arab chemist Jaber separated Arsenic from realger in the 8th century and soon it became the ideal homicidal poison, remaining so for centuries. It was such a popular homicidal poison that it acquired many names like 'King of poison, Poison of poisons and inheritance poison'.¹ Homicidal and suicidal cases are still reported. It is widely used in industries and in a chemical hair remover in Indo-Pak subcontinent (*Ball Safa Powder*).

Arsenic is cheaply and easily available as a grey powder (locally called *Sankhia*), which is non-

poisonous as it is not soluble in water and is not absorbed from the gastrointestinal tract. However it continuously undergoes conversion to white arsenic oxide, which is tasteless and highly poisonous.

It is crystalline at room temperature,² odorless,³ colourless and solubility is 20 g/L in water at room temperature.^{4,5} It is still in use for homicidal as well as suicidal purposes.^{6,7}

Absorption of trivalent and pentavalent forms is about 96.5% and 94% respectively¹ as compared to the less soluble forms, which is only 20-30%^{8,9} as indicated by the absence of urinary excretion of arsenic selenide^{10,11} after an oral dose.^{5,12-15}

It is mainly distributed in liver, kidneys, lungs, spleen and skin and excreted through kidneys.^{16,17} Clearance from these organs except skin is rapid.⁵ Its elimination through kidneys is up to 80% in 61 hours after oral dose.¹⁸ It is also extensively deposited in hair and nails¹⁸ but this is a biologically non available pool.^{1,17}

Though the lethal dose range is 120-200 mg (2 mg/Kg),¹⁶ some deaths have occurred only after taking amounts as much as 2 gm.¹⁹

Trivalent arsenic interacts with sulfhydryl group of proteins and enzymes; pentavalent arsenic substitutes for phosphorylation.²⁰ It can also directly damage DNA.²¹ Affinity of sulfhydryl group is used as chelating therapy.²² Its toxicity is related to its combining power with sulfhydryl enzymes thus interfering with cell metabolism; it also irritates mucous membrane and remotely depresses the central nervous system.

The fatal period is from 2 to 3 hours. Gastrointestinal presentation causes death within 12-48 hours.

Soluble compounds produce more toxicity than the less soluble ones due to their increased absorption.²² Arsenic trioxide is a major source of intoxication.^{23,24} The first manifestation of acute poisoning occurs within 15-30 minutes, but may be delayed if taken with food.

Acute arsenic toxicity usually leads to nausea, vomiting, diarrhea,^{16,23,24} anorexia, epigastric pain mimicking cholera and acute gastroenteritis, muscle cramps, dermatitis (exfoliative and erythroderma), cardiac abnormalities, hepatotoxicity, bone marrow depression, haematological abnormalities (anaemia), vascular lesions, peripheral neuropathies (motor dysfunction and parasthesias).^{5,12,25-28} The usual symptoms are nausea, burning pain in oesophagus, stomach and epigastrium followed by persistent continuous vomiting. The vomitus initially contains stomach contents, later on bile and mucous mixed with altered blood (coffee ground appearance). The main effect however is

diarrhoea accompanied by tenesmus and anal irritation. Stools are tinged with blood, similar to rice water stools of cholera and contain shreds of mucous membrane and traces of poison in 3-4 hours. There is intense thirst, but drinking water increases vomiting. Severe dehydration occurs with cramping pain in legs and suppression of urinary output.

Gastrointestinal haemorrhage may lead to cardiovascular collapse due to direct effect of arsenic on capillaries via sulfhydryl group binding.²⁹ Pronounced gastrointestinal symptoms are due to dilatation of splanchnic blood vessels resulting in sub mucosal vesicle formation; so the diarrhoea may be bloody; a garlic odour may be present in breath and in stools.³⁰

Collapse sets in with cold clammy skin, pale anxious face, sunken eyes, dilated pupils, rapid feeble pulse and sighing respirations. Death may be preceded by convulsions or coma.

Autopsy findings are mostly related to the gastrointestinal tract with mucosal hyperaemia, erosions, and haemorrhages and even sloughing; gastric contents emit a garlicky odour. Systemic effects may be found such as haemorrhages, degenerations and necrosis in liver and petechial haemorrhages in pericardium and also in the subendocardium.

Peripheral sensory neuropathy and/or motor neuropathy may be associated with dermal manifestations in the survivors of acute intoxication.^{15,31} Severe exposure can result in acute encephalopathy, CCF, convulsions, paralysis, coma and death.²⁵ Acute intoxication also produces hepatotoxicity^{6,19} and nephrotoxicity;^{19,29} rhabdomyolysis may precipitate renal failure.⁶

Tachycardia is typical after ingestion;^{19,32} ventricular arrhythmias,³³ typical Torsade de pointes,³⁰ prolongation of QT interval and non-specific T-waves⁶ and seizures may be present in acute and heavy intoxication.^{32,33}

It is a major constituent of weed killers, rodenticides and insecticides²² used in agriculture and forestry; not to mention its use to a lesser extent in the glass & ceramic industries. It is also used in food, homeopathic and herbal medicines.³⁴ It is a major product of combustion;³⁰ fumes of arsenic trioxide may be found in fires.³⁵

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