

MONITORING AND TREATMENT OF INDIVIDUALS INJURED BY WHITE PHOSPHOROUS

Pathophysiology: White phosphorus results in painful chemical burn injuries. The resultant burn typically appears as a necrotic area with a yellowish colour and characteristic garlic like odour. White phosphorus is highly lipid soluble and as such, is believed to have rapid dermal penetration once particles are embedded under the skin. Few studies have investigated the degree of tissue destruction associated with white phosphorus injuries. In the experimental animal model, most tissue destruction appears to be secondary to the heat generated by oxidation.

Systemic toxicity has been described extensively in the animal model. Pathologic changes have been documented in the liver and kidney. These changes result in the development of progressive anuria, decreased creatinine clearance, and increased blood phosphorus levels. Depression of serum calcium with an elevation in the serum phosphorus level (reversed calcium-phosphorus ratio) with electrocardiographic changes including prolongation of the QT segment, ST segment depression, T wave changes, and bradycardia also have been observed. Oral ingestion of white phosphorus in humans has been demonstrated to result in pathologic changes to the liver and kidneys. The accepted lethal dose is 1 mg/kg, although the ingestion of as little as 15 mg has resulted in death. Individuals with a history of oral ingestion have been noted to pass phosphorus-laden stool ("smoking stool syndrome").

Mortality/Morbidity: Morbidity and mortality are related directly to trauma and burns sustained from exposure.

- Burns usually are limited to areas of exposed skin (upper extremities, face). Burns frequently are second and third degree because of the rapid ignition and highly lipophilic properties of white phosphorus.
- Trauma usually is a combination of blunt and penetrating. Blunt trauma results from the percussion and force of the blast, and penetrating trauma results from projectiles produced from the explosion.

Prehospital Care: Direct prehospital management toward the evaluation and management of trauma.

- Secure the scene, since live munitions may be in the area.
- Perform ABCs of resuscitation.
- Terminate further oxidation of phosphorus by irrigation or placement of saline-soaked and/or water-soaked pads on areas of exposure.
 - Do not use oily or greasy dressing, since the element is lipid soluble and can penetrate into the tissue.
 - Remove contaminated clothing, since it may re-ignite and cause more extended and worsened burns than those sustained with white phosphorus alone.

Emergency Department Care: Continue a trauma management approach to the patient.

- Avoid contact with ignited white phosphorus. Such contact may result in a chemical burn injury to the health care provider.
- Continue irrigation; do not allow areas of exposure to dry, as this may result in re-ignition of white phosphorus.
- Grossly debride as much white phosphorus as possible. The use of a Wood lamp (ultraviolet light) results in the fluorescing of the white phosphorus and may facilitate its removal.
- Solutions of copper sulfate traditionally have been used as a neutralizing agent. Copper sulfate reacts with phosphorus to form cupric phosphate, which is black and assists in visualizing phosphorus. Stereomicroscopically, phosphorus particles have been observed to become covered with cupric phosphate, and this may facilitate their removal. This treatment has fallen out of favor because of reports of massive intravascular hemolysis associated with its use. This phenomenon is believed to be due to copper's activity as an inhibitor of several enzymes of the erythrocyte hexose monophosphate shunt.
- Take care to ensure that tetanus immunization is up to date as a standard component of burn therapy.

Consultations: Consultation with a burn team is mandatory for most patients. In addition, obtain trauma consultation for all patients with a history of significant trauma, especially those who may require surgical debridement of injuries.

MONITORING AND TREATMENT OF INDIVIDUALS EXPOSED TO DEPLETED URANIUM (DU)

- For the general population, neither civilian nor military use of DU is likely to produce exposures to DU significantly above normal background levels of uranium. Therefore, individual exposure assessments for DU will normally not be required. Exposure assessments based on environmental measurements may, however, be needed for public information and reassurance.
- When an individual is suspected of being exposed to DU at a level significantly above the normal background level, an assessment of DU exposure may be required. This is best achieved by analysis of daily urine excretion. Urine analysis can provide useful information for the prognosis of kidney pathology from uranium or DU. The proportion of DU in the urine is determined from the $^{235}\text{U}/^{238}\text{U}$ ratio, obtained using sensitive mass spectrometric techniques.
- Faecal measurement can also give useful information on DU intake. However, faecal excretion of natural uranium from the diet is considerable (on average 500 μg per day, but very variable) and this needs to be taken into account.
- External radiation measurements over the chest, using radiation monitors for determining the amount of DU in the lungs, require special facilities. This technique can measure about 10 milligrams of DU in the lungs, and (except for soluble compounds) can be useful soon after exposure.
- There are no specific means to decrease the absorption of uranium from the gastrointestinal tract or lungs. Following severe internal contamination, treatment in special hospitals consists of the slow intravenous transfusion of isotonic 1.4 % sodium bicarbonate to increase excretion of uranium. DU levels in the human, however, are not expected to reach a value that would justify intravenous treatment any more than dialysis

What are the medical procedures for people possibly exposed to high levels of DU?

- Any individual who claims to have been exposed to DU must be clinically evaluated. Physicians must collect an entire story of the possible exposure, to assess the date, time and place of exposure and the amount of dust or debris or the presence of fragments embedded in tissues. For children, ask if they were playing and possibly handling metallic fragments close to exploded bombs or other weapons, or even they are dressed with pieces of munitions, as necklaces or other.
- Perform an entire medical examination, determining blood urea and creatinine, albumin, glucose, full blood count and urine analysis. Perform a chest-x rays, if an exposure to contaminated dust can be supposed. Any patient who shows an alteration in one or more of these parameters should be tested for uranium exposure.
- In the short term the kidneys could be affected, in proportion to the amount of DU absorbed into the bloodstream. Signs of tubulopathy must be investigated. If the tubules are damaged presence of low molecular weight proteins in the urine can be demonstrated, among which beta 2 microglobulin is prevalent.
- This protein must be researched on urine collected for 24 hrs., according precisely to the recommendations of the laboratory, due to the instability of the protein. If results are positive for any alteration, it is worthwhile to perform a test to detect directly the presence of uranium in urine. And following a new positive result for uranium to test DU. Both procedures must be conducted in appropriate laboratories that will provide appropriate information about the collection of specimens.

Referral to a nephrologist is advised, particularly for a correct diagnosis on the basis of further tests and for the necessary therapy. Any way there is no treatment for asymptomatic or clinically mute acute exposure. But if there is a tubulopathy, sodium bicarbonate perfusion must be performed to alkalinize the urine, bind the uranium present in the bloodstream, facilitate its renal excretion and prevent its reabsorption in the renal tubules. Monitoring renal and liver function. It is important to remind that these methods are useful, only if applied early after exposure, because the urinary excretion of uranium is temporary. They seem to have no effect once the uranium is fixed in the kidney or in the skeleton. If dust inhalation resulted in the incorporation of significant amounts of insoluble uranium compounds, long term follow up of patients will include checks for lung tumours