Paraquat Poisoning

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Abstract
Paraquat is a non selective herbicide used widely in Asia. On ingestion it can produce multi organ dysfunction and rapidly progressing pulmonary fibrosis. We report a case of delayed presentation of paraquat poisoning.

Introduction
Paraquat (N,N′-DIMETHYL-4,4′-BIPYRIDINIUM DICHLORIDE) is an organic compound with chemical formula [(C₆H₇N)₂]Cl₂. It is classified as a viologen. It is a quickly acting non selective herbicide widely used in Asia. It is highly toxic to humans and can cause multisystem involvement. No specific antidote is available for its treatment till now. Because of its toxicity, it has been forbidden in European union since 2007 and in USA, it is classified under restricted use which means it can be used only by licensed applicator.

Case Report
A 25 year old male presented to the emergency department with c/o difficulty in swallowing to solid and liquid, pooling of saliva in mouth, ulcer in tongue, yellow colouration of conjunctiva, decreased urine output and mild degree of respiratory difficulty. He was a case of paraquat poisoning, ingested 2 weeks back for which he had been treated in a local hospital symptomatically and gastric lavage was done. On examination his GCS was 15/15, pulse-50/min.BP-100/70 mm of Hg. Examination of chest, cardiovascular system, abdomen and CNS revealed no abnormality except tongue was coated and red in appearance. On investigation, TLC-14,230/cumm, Hb-12.9 gm/dl, TPC-225000, MP-negative, Urea-275 mg/dl, Cr.- 4.78 mg/dl, Sodium (Na⁺)-134, Potasium (K⁺)-3.5, Serum Total Bilirubin-2.8, AST-1000, ALT-1071, ALP-518, PT-13.3, INR-1.11, Uric Acid-11.6. Ultrasound of abdomen revealed minimal left pleural effusion. HRCT of chest showed fibrotic strands in different segments of lungs and also sub pleural bands in right lower lobe. Patient was treated symptomatically with antibiotics, nasal feeding, fluid monitoring. Gradually patient recovered and was discharged.

Discussion
Paraquat induced toxicity is a manifestation of redox cycling and subsequent generation of reactive oxygen species. Generation of highly reactive oxygen and nitrogen species results in damage to most organs but the toxicity is particularly severe in the lungs as it is taken up against a concentration gradient in lungs. In the lungs, initially it causes acute alveolitis in one to three days and subsequently progresses to rapidly progressive fibrosis. Other organs affected are kidney, liver and GI tract. The appearance of tongue is known as paraquat tongue. Paraquat can cause perforation of esophagus. The major cause of death is due to respiratory failure.

Other secondary effects of oxidative stress also play synergistic effect in the manifestation of overall clinical presentation of paraquat poisoning. These are lipid peroxidation, mitochondrial toxicity, oxidation of NADPH, activation of nulear factor kappa beta, and apoptosis.

Conclusion
Paraquat poisoning is life-threatening with multi organ failure and pulmonary fibrosis with high fatality rate. The presentation may be delayed for weeks with a symptom-free window period. Since there is no specific antidote for paraquat, if not monitored closely for few weeks, the patient may succumb to multi organ failure.

Fig. 1: Coated tongue (paraquat tongue)
Fig. 2: PA view of chest shows no abnormality
Fig. 3: (a, b, c) HRCT of chest showing fibrotic strands in different segments of lung and also sub pleural band in right lower lobe of lungs

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Cerebral Hyperperfusion Syndrome following Staged Bilateral Internal Carotid Artery Stenting

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Abstract

Cerebral Hyperperfusion syndrome is a relatively rare event following carotid revascularization. It can occur after both carotid endarterectomy and carotid artery stenting. It is characterized by focal neurodeficit, seizures and headache in the absence of ischemia. It occurs due to ipsilateral cerebral edema secondary to hyperperfusion. CT and MRI of the brain are the main modalities used for diagnosis and to rule out infarct. Prompt recognition and treatment can prevent permanent injury to the brain. We present a case of cerebral hyperperfusion syndrome in an elderly gentleman after a staged bilateral internal carotid artery stenting.

Fig. 1: Angiography showing critical stenosis of right internal carotid artery (left) and post stenting (right)

Fig. 2: Distal protection filter showing collected debris

References

1. Court of First Instance of the European Communities, Press Release No° 45/07.

Introduction

Although carotid endarterectomy (CEA) has remained a standard therapy for stroke prevention in patients with significant carotid artery stenosis, carotid artery stenting (CAS) has emerged as an alternative. Neurological complications following carotid revascularisation with either technique are well known, of which ischemic injury to the brain, due to embolisation or carotid occlusion are the most common. Cerebral hyperperfusion syndrome (CHS) is a relatively rare, but potentially devastating event known to complicate carotid revascularisation. It is defined as a clinical triad of ipsilateral headache, seizures and focal neurological deficits occurring in the absence cerebral ischemia. Awareness of this entity enables early detection and initiation of appropriate therapy to limit brain injury.

Case Report

A 72 year old gentleman, diabetic and hypertensive presented with bilateral critical internal carotid artery (ICA) stenosis of more than 70%. He had recurrent episodes of transient ischemic attacks (TIA) involving the left upper and lower limbs in the preceding 2 months. Stenting of the symptomatic artery was planned, to be followed by the contralateral vessel after four weeks. He underwent angioplasty and stenting to right ICA on 19/2/2016 using 9-7 x 40 mm X-ACT stent with a...