exist.\textsuperscript{4,5} Although controversial, the presence of sympathetically mediated pain is an accepted etiology for many regional pain problems.

The treatment of RSDS consists of physical therapies like active and passive range of motion exercises, transcutaneous electrical nerve stimulation (TENS), desensitization techniques, and sensory re-education of the extremity.\textsuperscript{6} Pharmacologic therapy consists of analgesics, antidepressants, anticonvulsants, membrane-stabilizing agents, adrenergic compounds, calcium channel blockers, corticosteroids, bisphosphonates and newer agents like neurotropin.

Till date very few cases of RSDS have been reported to occur after a pacemaker implant.\textsuperscript{2,4} Our patient had symptoms which began after 10 months of the pacemaker implantation which was very similar to the patient reported by Okada et al.\textsuperscript{3} The patient described by Okada et al had a pacemaker insertion in January 2000 and was diagnosed to have RSDS in September 2000.\textsuperscript{4} She was treated with methylprednisolone and neurotropin.\textsuperscript{2} Our patient responded to indomethacin and Amitryphline and there was reversal of the demineralisation and osteopenic changes of the bone.

Imidacloprid Poisoning

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Abstract

Imidacloprid is newer systemic insecticide, a nicotine analogue, acts on the nervous system. Patient can present with variable manifestations like irritability, labored breathing, emaciation, twitching and delirium. Here we report a case presented with sever neuropsychiatric symptoms with respiratory failure following self ingestion of poison. Patient recovered with supportive and symptomatic treatment.

Introduction

Imidacloprid is a relatively new insecticide in the chloronicotinyl nitroguanidine class. It was first registered for use as a pesticide in the U.S. in 1994 and was the first insecticide in its chemical class to be developed for commercial use.\textsuperscript{1} Imidacloprid has a wide variety of uses; it is used on cotton and vegetable crops, turf grass and ornamental plant products, in indoor and outdoor cockroach control products and in termite control products. It is also used in products for pets, home lawn and garden use including some, like potting soil, which may not always be easily recognized as pesticides.

Imidacloprid acts as a competitive inhibitor at nicotinic acetylcholine receptors in the nervous system.\textsuperscript{2} It effectively blocks the signals induced by acetylcholine at the post-synaptic membrane, resulting in impairment of normal nerve function.\textsuperscript{2,4} Imidacloprid has a higher binding strength to insect nerve receptors than to mammalian receptors.\textsuperscript{2}

Poisoning with imidacloprid has been reported to have very low toxicity. We are reporting a case of self poisoning with imidacloprid poisoning leading to severe psychiatric symptoms and respiratory failure.

References


Case Report

A forty one year male patient was brought to emergency with alleged history of self ingestion of 75ml 70% imidacloprid three hours before admission. He had developed nausea, vomiting, abdominal cramps, muscle twitching and difficulty in breathing within 30 minutes of ingestion of poison. He had no significant co-morbid medical illness or any addiction. On arrival in emergency room he was found to be drowsy and dyspnecic. On physical examination his temperature was 98°F with heart rate 115/min, blood pressure 150/90 mmHg, respiratory rate 45/min and oxygen saturation of 60%. Muscle twitching was present. There was no pallor, cyanosis or injury marks. There were scattered coarse crepitations on chest auscultation. On neurological examination he was drowsy with Glasgow Coma Scale (GCS) of 12/15 (E5, M6, V1) with no focal neurological deficit. Rest of the systemic examination was unremarkable.

Investigations showed that he had mild leucocytosis with normal hemoglobin level, RBC and platelet count. Serum electrolytes, random blood sugar, renal, liver and thyroid function was found normal. His serum cholinesterase and CPK level was normal. Chest x ray and ECG did not reveal any abnormality. Initial arterial blood gas showed metabolic acidosis (pH 7.2, HCO\textsubscript{3} 15mmol/L, PaCO\textsubscript{2} 29, PaO\textsubscript{2} 132) which normalized after 24 hours.

He was immediately resuscitated with endotracheal intubation and ventilated with ambu bag. Gastric lavage was
Nephropathy in Association with Annular Psoriasis

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Abstract
Occurrence of glomerular diseases in psoriasis is rare, although the number of reports is increasing in recent years. Different types of glomerular involvement have been reported but mesangio-proliferative glomerulonephritis with IgA deposits, AA amyloidosis and membranous nephropathy are relatively common in association with psoriasis. The term 'psoriatic nephropathy' has been introduced recently. We contribute a case to the ongoing discussion regarding psoriatic nephropathy. Our patient had mesangio-proliferative glomerulonephritis (with IgG and C3 deposition) in association with annular psoriasis (rare variety of chronic plaque psoriasis). Presence of mesangio-proliferative glomerulonephritis with IgG deposition is rare in association with psoriasis. The patient responded well to weekly methotrexate (15 mg) injection. Methotrexate has not been tried previously in psoriatic nephropathy or reported to be effective in it.

Introduction
Psoriasis is an immune-mediated chronic inflammatory disorder of the skin with distinct microvascular changes. It can involve joints but involvement of internal organs is uncommon. Psoriatic nephropathy is a recently described clinical disorder. Nephropathy in association with psoriasis is increasingly being reported in recent years.

Case Report
A 32 years old Hindu male from rural West Bengal presented with wide spread psoriasis with hair and nail involvement for last two years, which was aggravated since last four months. The patient presented to us with complaints of swelling of face and both lower limbs for last six days, along with decreased urine output for same duration. There was no history of fever, sore throat, and joint pain or swelling. He had no history of haematuria. He was previously non-diabetic and normo-tensive and had no past history of nephropathy.

On examination patient was conscious, oriented, and...