larynx clinically resemble malignancy and rarely tuberculosis as incidence of primary TB of tongue presenting as ulcer is 0.2% of all cases of TB. Apart from these, other conditions closely mimicking with histoplasmosis are sarcoidosis and blastomycosis. Cutaneous and mucocutaneous involvement in blastomycosis is far more common, while sarcoidosis lesions are ruled on clinical and epidemiological grounds.

The definitive diagnosis of disseminated disease depends on culture or biopsy identification of the organisms within the extrapulmonary tissues. Gomori’s methenamine silver stain and Wrights stain are preferred over haematoxylin and eosin (Hande) stain to detect fungi as capsule of yeast is a polysaccharide and stains poorly with Hande. Centre and base of the ulcer predominates with heavily laden parasitized macrophages as compared to the marginal areas. Microscopically histoplasmosis lesions may demonstrate tuberculoid granulomas as central caseation necrosis making it difficult to differentiate from tubercular lesions, but usually are absent in disseminated disease. If present represents mildest form of disease with low grade infection and normal immune system of the individual. Peripheral blood film and bone marrow aspiration are rarely positive in chronic disseminated form of disease.

In countries like ours where both malignancy and tuberculosis are highly prevalent, clinical diagnosis of histoplasmosis is a challenge. Clinically histoplasmatoid lesions resembles to an extent with malignancy, so a high index of suspicion must be kept for early and prompt diagnosis. Also histoplasmosis must be kept in the differential diagnosis of non healing ulcer in oral cavity.

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peripheral nerve injury cases. It usually occur secondary to fractures, sprains, and trivial soft tissue injury.1 Cardinal signs include pain, edema, stiffness, and discoloration. Demineralization of the bone and osteoporosis are among the most classic late findings.2 However, this syndrome is very rare to be seen after a pacemaker insertion. We report a case who had undergone a pacemaker insertion and later developed RSDS. The findings for diagnosis were confirmed with an X-ray of the hand suggestive of osteopenia of the bones of involved extremity.

Case Report

A 55 year old male presented with complains of pain and swelling over dorsum of right hand and small joints, and loss of sweating over right hand since two months. He was a known case of Mitral valve prolapse(MVP) with mitral regurgitation and complete heart block for which pacemaker was implanted 1 year back. He was also a known case of neurofibromatosis. On examination, pulse was 62/min, blood pressure was 130/80 mmHg. There was a pansystolic murmur in the mitral area on auscultation. There were multiple neurofibromas over the entire body. There was pain and swelling in the right wrist, metacarpophalyngeal (MCP) joints and proximal interphalyngeal (PIP) joints as well as distal interphalyngeal (DIP) joints (Fig. 1). The wrist joint was tender and movements were restricted due to pain. All the other joints were normal. His blood investigations like Haemoglobin, WBC count, liver and renal function tests and blood glucose were normal. The erythrocyte sedimentation rate (ESR) which was 50mm at the end of 1 hour. Bilateral wrist X-ray was suggestive of pronounced demineralization (osteopenia) in the right hand (Fig. 2). There were no joint erosions. His chest X-ray was normal showing the implanted pacemaker on the right side (Fig. 3).

Based on the fact that there was a pacemaker implantation done about 1 year back, blood examination being normal, the X-ray findings which were apparent only in the involved hand and there were no other episodes of trivial trauma or injury in the past 1 year following pacemaker insertion which the patient could recollect that could have triggered RSDS such as injury or stroke, a diagnosis of RSDS secondary to pacemaker implantation was entertained. As a part of treatment for RSDS he was started on oral amitryptiline 25 mg at bedtime and oral indomethacin 25 mg thrice a day to which he responded adequately. The burning pain was reduced and swelling subsided after 1 month of treatment.

Discussion

Reflex sympathetic dystrophy syndrome (RSDS, algodystrophy, complex regional pain syndrome [CRPS], Sudeck’s atrophy) usually follows minor trauma or surgery or may occur spontaneously.2 It also has been associated with various clinical conditions like diabetes mellitus and Parkinson’s disease. RSDS begins with spontaneous pain associated with vasomotor and sudomotor disturbances. Currently, no specific pathologic, histologic, or biochemical markers of this condition

Fig. 1 : Swelling in the right MCP, PIP, DIP and wrist joint and neurofibromas.

Fig. 2 : X-ray bilateral wrist joint showing osteopenia in the right hand; the joint spaces are normal. There are no erosions.

Fig. 3 : X-ray Chest Showing the implanted pacemaker.
Imidacloprid Poisoning
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Abstract
Imidacloprid is a 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 severe 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.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.2 It effectively blocks the signals induced by acetylcholine at the post-synaptic membrane, resulting in impairment of normal nerve function.2,4 Imidacloprid has a higher binding strength to insect nerve receptors than to mammalian receptors.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.

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 dyspnoeic. 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, HCO3 15mmol/L, PaCO2 29, PaO2 132) which normalized after 24 hours.

He was immediately resuscitated with endotracheal intubation and ventilated with ambu bag. Gastric lavage was

References

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