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Perioperative Management of a Patient with Leprosy- A Case Report

Dr. SAFIYA I. SHAIKH	Professor and Head, Department Of Anaesthesiology, Karnataka Institute Of Medical Sciences, Hubli , Karnataka
Dr. ROHINI DATTATRI	Post-Graduate Student, Department Of Anaesthesiology, Karnataka Institute Of Medical Sciences, Hubli , Karnataka
Dr.SARALA B. M	Post-Graduate Student, Department of Anaesthesiology, Karnataka Institute Of Medical Sciences, Hubli , Karnataka

ABSTRACT

Leprosy is a granulomatous disease affecting the skin and nerves caused by mycobacterium leprae. It continues to be a significant public health problem and remains a stigmatizing disease. The disease causes skin lesions and neuropathy. Complications secondary to neuropathy can result in deformity and disability. Here, we share our experience in successful management of a lepromatous leprosy patient posted for correction of deformity.

Introduction:
Leprosy, also called Hansens disease, is a mildly infectious disease that attacks skin and peripheral nerves. The infection can damage nerves in the face and body leading to a loss of sensation and paralysis. Because sensation is lost, everyday activities are fraught with danger as wounds go unnoticed and may lead to serious harm when left unattended. Patient may lose fingers, feet and eyesight[1]. This report sums up the perioperative management of leprosy patients posted for corrective surgery.

Case report:
A 16 year old girl presented with claw deformity of medial two fingers of right hand. She was diagnosed as having lepromatous leprosy and was on multi drug treatment. She was posted for elective correction of the deformity.

On examination, she had multiple hypopigmented patches over the right forearm and right thigh. Sensation to heat, cold and pain to pinprick was decreased over these areas. This neurological deficit was documented and a general anaesthetic technique was chosen for this patient.

Routine pre operative investigations like complete hemogram, coagulation profile, renal and liver function tests were normal. Chest x ray and ECG were also within normal limits.

In the operating room, iv line was secured with 18 g cannula over the left side. Monitoring included pulse oximetry, NIBP, ECG, ETCO2 and temperature. Injection Ranitidine 50mg and ondansetron 4mg was given intravenously. Premedication was given in the form of glycopyrrolate 0.2mg, midazolam 2mg and fentanyl 100mcg intravenously. Preoxygenation was done with 100% oxygen for 3 minutes and anaesthesia was induced with injection propofol 100mg and atracurium 25mg was given to facilitate tracheal intubation. Trachea was intubated with 7mm cuffed endotracheal tube and anaesthesia was maintained with nitrous oxide in oxygen in the ratio 5:3 and intermittent boluses of atracurium for relaxation. Intra operative vitals remained stable throughout the procedure. At the end of the procedure, patient was reversed with neostigmine 2.5 mg and glycopyrrolate 0.4mg intravenously and the trachea was extubated.

Discussion:
Dr Armauer Hansen first discovered the leprosy bacillus in Norway in 1873[1]. Though seldom lethal, the disease causes appreciable morbidity principally through its effect on the skin, peripheral nerves and nasal mucosa[2]. Globally 244,796 and in India 133,717 new cases were diagnosed and reported to World health organization in 2009. Nepal eliminated leprosy as a public health problem during the last quarter of 2009 [3].

Lepromatous leprosy is characterized by early, widespread, ill defined erythematous or hypopigmented papules and plaques. Later findings include facial infiltration (leonine facies), ocular effects, neurological involvement, saddle nose deformity and hypogonadism. Severe peripheral neuropathy in our patient resulted in peripheral anaesthesia, distal digit resorption and claw hand. Hence general anesthesia was planned.

Autonomic neuropathy widely occurs in leprosy as in diabetes mellitus. There is involvement of both divisions of autonomic nervous system. Sympathetic system involvement is always associated with parasympathetic system and severity of neuropathy is found to be high in leprosy of longer duration[4].

Ischaemic heart disease is a rare occurrence in leprosy since elevation of high density lipoprotein occurs in lepromatous leprosy which is a protective factor against ischaemic heart disease[4].

Laryngeal lesions in leprosy have been a frequent finding causing husky voice, hoarseness, cough and dyspnoea. In the larynx, the mycobacteria infiltrate through the mucosa and reach small branches of sensory nerves or affect a motor nerve, thus causing paralysis or paresis of the vocal folds and possible alteration of the sensation of the mucosa. The sensory or motor impairment may cause aspiration and bronchopneumonia[5]. These patients have respiratory dysautonomia with prolongation of breath holding time and impaired cough reflexes which is due to involvement of the afferent vagal fibres. Spontaneous respiratory arrest and unexplained sudden death may also occur[6]. Hence patients should be referred for otolaryngological examination when they have the symptoms and during anaesthesia, aspiration prophylaxis should be considered.

Lepromatous leprosy usually begins in the form of chronic rhinitis. The nasal mucosa becomes ulcerated and form scabs which can bleed. Later it may lead to perforation of the septum or spread to maxilla[7] Hence great care should be taken while introducing the nasogastric catheter. Atrophy of the anterior nasal spine and maxillary alveolar process are seen in patients with advanced lepromatous leprosy. These lead to nasal collapse and loosening of the upper central incisor teeth forming part of the changes described as “facies leprosa”[8] These changes may cause a difficulty in nasal intubation.

Patients with leprosy show a dimorphic picture of hemolytic and megaloblastic anemia.

These patients may present with renal damage in the form of amyloidosis, glomerulonephritis, interstitial nephritis and pyelonephritis. Commonly observed abnormalities are proteinuria and hematuria[8].

Significantly elevated levels of gamma glutamyl transpeptidase, indicating liver damage has also been shown. Impaired drug metabolism in the form of decrease in drug clearance and drug metabolizing enzymes has also been reported. Hepatitis B viral infection is frequently associated with leprosy[8].

To avoid sequelae, early recognition and treatment are necessary. Multi drug therapy regimen have been devised and recommended by the WHO and are based on paucibacillary or multibacillary status. These medications include rifampicin, clofazimine and dapsone and are given for six months to two years depending on the clinical findings[2].

Patients of lepromatous leprosy are also believed to have natural immunity to tetanus. Probably perineural and intraneural fibrosis in these patients prevents the tetanus exotoxin from spreading proximally along the peripheral nerves[8].

These patients should be assessed preoperatively for –

1. Cardiovascular autonomic dysfunction like abnormal response in heart rate and blood pressure response to standing, baroreceptor dysfunction.
2. Respiratory autonomic dysfunction, nasal obstruction, vocal cord involvement.

A complete motor and sensory neurological assessment should be carried out to ensure that nerve function is not deteriorating, especially as this can be asymptomatic.

Researchers have speculated that there may be a possible genetic link between pseudocholinesterase deficiency and susceptibility to leprosy. Hence succinyl choline should be used cautiously in these patients which can lead to prolonged apnoea[9,10]. So for our patient we preferred atracurium for endotracheal intubation.

Spinal and epidural anaesthesia should be used cautiously in these patients. Autonomic nerve involvement may cause bradycardia and hypotension[11,12].

These patients can have a delay in postoperative recovery because of impaired metabolism of drugs and decreased renal clearance of drugs. [11,13].

As our patient was not having any abnormality in LFT and RFT, intraoperative course and postoperative recovery was smooth.

Leprosy continues to be an important health problem in India and worldwide. These patients may require anesthesiologist services for surgical interventions and we must be prepared to provide good treatment.

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