Bruns–Garland syndrome precipitated by anti-tuberculous therapy in a patient with type 2 diabetes mellitus

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Abstract

India has a high prevalence of tuberculosis as well as diabetes mellitus, and both contribute to a major disease burden. Diabetes mellitus increases the risk of tuberculosis. Patients with diabetes are more prone to develop cavitary lesions than non-diabetics. Tuberculosis can worsen the glycemic status of an individual and anti-tuberculous drugs like Rifampicin can interact with oral anti-diabetic drugs, further worsening the glycemic control. This can result in complications related to diabetes, like diabetic amyotrophy. We hereby report the case of a 69-year-old gentleman with diabetes mellitus, who developed uncontrolled blood sugars, leading to Bruns Garland syndrome or diabetic amyotrophy on starting anti-tuberculous therapy for sputum positive pulmonary tuberculosis.

Key words: Bruns Garland syndrome, Diabetic amyotrophy, Diabetes mellitus, Tuberculosis, Anti-tuberculous therapy

Key Messages: Tuberculosis and diabetes mellitus have a bi-directional relationship. Tuberculosis per se and the drugs used to treat tuberculosis can worsen the glycemic control, warranting adjustment in doses of oral anti-diabetic drugs.

Introduction

Tuberculosis as well as diabetes mellitus, are widely prevalent in India, contributing to a major disease burden. Diabetes mellitus increases the risk of tuberculosis and patients with diabetes are more prone to develop cavitary lesions than non-diabetics.[1] Tuberculosis also worsens the glycemic status in patients with diabetes either directly or due to the drugs used to treat tuberculosis.[2] This can lead to the development of complications related to hyperglycemia, like diabetic amyotrophy. We hereby report the case of a 69-year-old gentleman with diabetes mellitus, who developed uncontrolled blood sugars, leading to Bruns Garland syndrome or diabetic amyotrophy on starting anti-tuberculous therapy for sputum positive pulmonary tuberculosis.

Case report

A 69-year-old gentleman, with history of type 2 diabetes mellitus, for the past 16 years, was on Metformin 1 gm/day, Glimepiride 4 mg/day and injection human insulin 40 units/day, with adequate blood sugar control. He started developing cough 3 months back, there was no hemoptysis. He complained of loss of appetite, fatigue and lost around 8 kg weight over 1 month. He was evaluated, chest X- ray showed a cavitary lesion in the right lung apex. Sputum acid fast bacilli (AFB) was positive. Hence he was started on 4 drug regimen of anti-tuberculous therapy (ATT), including Rifampicin, Isoniazid, Pyrazinamide and Ethambutol. His baseline glycated haemoglobin (HbA1c) was 6.5%. Around one month later, he started complaining of severe left thigh pain, with difficulty in climbing up and down the stairs and difficulty in getting up from squatting position. He

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developed buckling of the left knee. He also complained of paraesthesias over bilateral soles. On examination, he had tenderness and wasting of the left thigh. Examination of motor power revealed MRC 4/5 of left hip flexion and knee extension. The left knee jerk was absent; all other deep tendon reflexes were preserved. Pain sensation (tested by a pin prick test) was normal. There was impaired sense of vibration over bilateral feet. The rest of his physical examination was normal. The possibility of a diabetic amyotrophy (Bruns–Garland syndrome) involving the left side, along with a diabetic sensory polyneuropathy was considered. Blood tests indicated a fasting plasma glucose level of 250 mg/dL and HbA1c of 10. Nerve conduction study showed significant reduction in compound muscle action potential involving the left femoral, peroneal and tibial nerves, with normal distal latency. It also showed severe sensory axonal neuropathy involving bilateral lower limb nerves. Electrophysiology was suggestive of left lumbosacral plexopathy, with sensory axonal neuropathy. MRI of the lumbar spine and plexus was normal. His oral antidiabetic drugs (OAD) and insulin were optimized, leading to adequate blood sugar control over the next one week. He was initiated on intravenous immunoglobulin (IVIg) for diabetic amyotrophy, and was given for 5 days at a total dose of 2g/kg. On the fifth day of starting IVIg, the symptoms of weakness dramatically improved. Electrophysiology was not repeated.

Discussion

Diabetes and tuberculosis contribute to a major disease burden in developing countries, and they often co-exist. Diabetes is reported to increase the risk of developing active tuberculosis. [1] Tuberculosis also affects diabetes by causing hyperglycemia and causing impaired glucose tolerance, as a result of stress response to infection or associated tuberculous pancreatitis. The drugs used to treat tuberculosis, interact with oral anti-diabetic drugs and may lead to suboptimal glycemic control, leading to complications related to diabetes, like diabetic amyotrophy. [2] Diabetic amyotrophy, was first described Bruns and Garland defined the term diabetic amyotrophy. [3] It has been found in less than 1% of patients with diabetes mellitus, and affects males more frequently than females. [4] It is also known as diabetic lumbosacral radiculo-plexo-neuropathy (DLRPN) and is often unilateral. The diagnosis of diabetic amyotrophy can be made clinically. It is associated with severe pain, followed by weakness and wasting. The weakness of the lower limb is often proximal, although distal muscle groups can also be affected. Other symptoms include paraesthesia, burning sensation, allodynia and numbness. It is an immune mediated inflammatory disorder, resulting in vasculitis and ischemic injury to the plexus. Hence immunotherapy has an important role in management of diabetic amyotrophy. It is often precipitated by an episode of hyperglycemia, as was seen in this patient. Our patient had optimal control of blood sugar, before staring on ATT. In studies, Rifampicin has shown to modestly reduce the plasma concentrations of glimepiride, probably by inducing the CYP2C9-mediated metabolism of glimepiride.[5]This must have been responsible for hyperglycemia in our patient, precipitating diabetic amyotrophy.

Conclusion

Diabetes and tuberculosis often co-exist in our country and are known to adversely affect each other. Anti-tuberculous drugs can induce the metabolism of OAD's, thereby worsening glycemic control, leading to complications related to diabetes. A thorough knowledge of pharmacokinetic interactions of anti-TB drugs with OAD's will always enable a clinician to treat patients with more safety.

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