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Case Report

A Rare Case of Compressive Cervical Myelopathy Presenting with Reversible Complete Heart Block

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ABSTRACT

We present here a rare case of an elderly male who presented with the complaints of progressively worsening gait imbalance, associated with acute onset giddiness. On evaluation, the patient had bradycardia with exaggerated upper and lower limb deep tendon reflexes and pain on neck movements. A Magnetic Resonance Imaging (MRI) showed posterior disc herniations at C6-C7 level with neural foraminal narrowing, and 12 lead Electrocardiogram showed Complete Heart Block (CHB). Decompressive surgery with temporary pacing was done, following which, the patient recovered well. Although Permanent Pacemaker (PPM) was initially planned, it was not required due to reversion to normal sinus rhythm on ECG, post-surgery. Hence, rare reversible causes of CHB should be ruled out, before opting for a long term permanent invasive treatment modality.

Keywords: Compressive Cervical Myelopathy, Complete Heart Block, Cardiac Pacemaker

INTRODUCTION

Compressive Cervical Myelopathy (CCM) is a common morbidity in the elderly population with nearly 85% of adults over the age of 60 having Magnetic Resonance Imaging (MRI) evidence of one or more cervical spinal level.[1,2] However, its presentations remain diversified, ranging from asymptomatic to severe, debilitating neurological deficits leading to limitations of activities of daily routine.[3] Here, we present a case of a rare manifestation of CCM, leading to symptomatic bradycardia.

CASE HISTORY

A 65 years old hypertensive male (well controlled on Tablet Amlodipine 5 mg once a day), presented to the hospital with progressively worsening difficulty in walking due to imbalance, associated with lower back pain over the last 3 months. His complaints had worsened over the last 10 days, with increasing difficulty in standing up and walking, without any alteration of bowel or bladder habits. However, movements were restricted since the last 10 days, due to weakness in bilateral lower limbs and associated giddiness in the form of whirling of surroundings. He also had difficulty in grasping objects with his left hand. He also had a history of experiencing giddiness upon exertion since the last 3-4 years. There was no history of fall, sustained injury, chest pain or palpitations, or loss of consciousness.

General examination showed severe bradycardia (Heart Rate: 35/min, regular), with otherwise normal vital examination (Blood Pressure: 136/82mm Hg). There was marked limitation of the movement of neck, associated with radicular pain radiating down the left hand along the C6-C7 nerve roots and positive Lhermitte's sign. Neurological examination showed power of 3/5 in bilateral knee and hip, flexors and extensors, 4/5 in elbow flexion and extension, and shoulder abduction and adduction. Biceps, Triceps, Knee and Ankle jerks were exaggerated. Babinski sign was seen. CCM was suspected, and an MRI was done (Figure-1), which showed large posterior paracentral Intervertebral Disc (IVD) herniation at the C6-C7 level, compressing upon the C7 nerve root, with bilateral neural foraminal narrowing

(left > right). Large posterior IVD herniation with osteophyte complex was also noted at L4-L5 level, with small posterior disc herniation at C3-C4 level, encroaching upon bilateral C4 nerve roots.

Evaluation of bradycardia led to a finding of Complete Heart Block (CHB) (Figure-2) with a ventricular rate of 36/min, atrial rate of 45/min, with AV dissociation on Electrocardiogram (ECG). Echocardiography showed hypertension related changes i.e., Left Ventricular Hypertrophy, LA enlargement (48 cm) and Grade 2 Diastolic Dysfunction. Blood investigations were done to rule out metabolic causes, which were all within normal limits. In view of acute worsening of complaints, the patient was planned for surgical intervention under cover of Temporary Pacemaker (TPM) with plan to convert to Permanent Pacemaker (PPM) if required post operatively. Posterior approach C5-C6 and C6-C7 level discectomy with cage fixation and L3-L4 and L4-L5 level laminectomy and foraminotomy was performed with no immediate periprocedural complications. Intraoperative heart rate was controlled with TPM. On third day post operative, the patient was taken up for PPM. However, it was noticed in the Cath lab, that the patient was able to maintain Normal Sinus Rhythm (NSR) without a pacemaker. Hence, PPM was deferred, and the coronary angiogram (CAG) was done, which revealed 80% stenosis in LAD. Percutaneous Transluminal Coronary Angioplasty was done, and stent was placed in the LAD. The rest of the hospital course was uneventful, and the patient was discharged with normal hemodynamics, and ECG showing NSR (Figure-3).

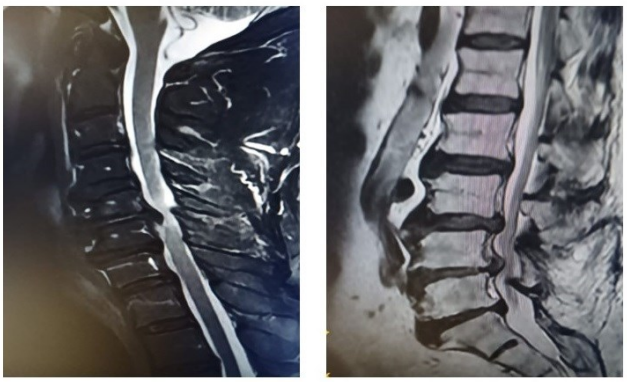

Figure-1: MRI Cervical and Lumbar Spine showing posterior disc herniations

DISCUSSION

Although multiple presentations of CCM, such as reduced hand dexterity, neck pain and limitation of range of neck movements, and quadriplegia amongst others have been noted, presentations of bradycardia are rare.[4] Moreover, CCM presenting with symptomatic CHB, is further rare. The parasympathetic outflow from the spinal cord comes from the craniosacral segments i.e., cranial parasympathetic outflow and the pelvic splanchnic parasympathetic outflow. The Vagus Nerve, via the superior and inferior cardiac nerves, causes bradycardia. These branches are given off superior to the mid-cervical trunk and functional stimulation can cause bradycardia.[5] Since these branches lie in close anatomical proximity to the cervical spine, it is likely that in the current case, the cervical IVD herniations impinging and irritating these branches, could have led to bradycardia. Although multiple neurological deficits secondary to CCM have been documented, those causing autonomic disturbances are less frequent. Similarly, the sympathetic autonomic outflow, coming from the thoracolumbar segments via the stellate ganglion, cause tachycardia.[6] Hence, for thoracolumbar spinal lesions to cause bradycardia, would have to be interruptive lesions.[7] That is, interruption of the supraspinal vasomotor control leading to unopposed vagal stimulation, can lead to persistent bradycardia. However, the current case did not have any obvious thoracolumbar spinal lesions. Hence, it is likely, that the former hypothesis caused symptomatic bradycardia and CHB in our patient. Moreover, the CHB converted to NSR post-surgery. To the best of the knowledge of the authors, this is the first case to report CCM presenting with CHB which resolved with surgical intervention of CCM.

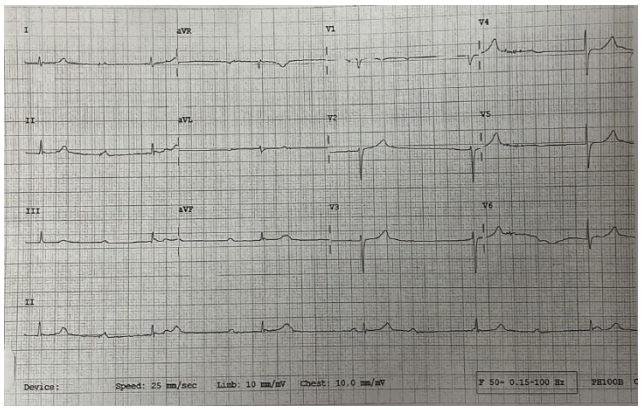

Figure-2: ECG on presentation showing CHB

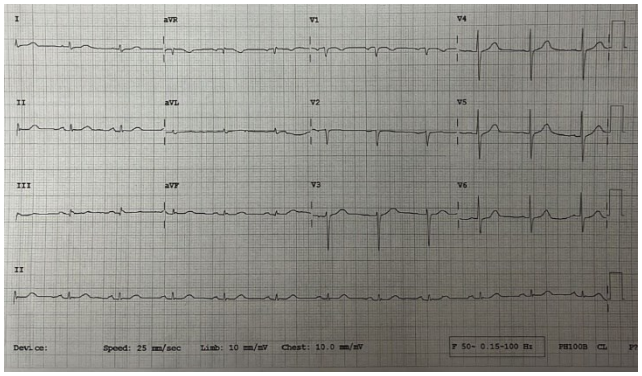

Figure-3: ECG on discharge showing NSR

CONCLUSIONS AND IMPLICATIONS

In the current era of availability of efficient interventions, it is necessary for clinicians to rule out likely rare reversible causes of presenting complaints. This case highlights the importance of clinical examination to approach a correct diagnosis. Although rare, autonomic complications of CCM could be potentially fatal, unless operated upon immediately.

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