

Osteomalacia induced peripheral neuropathy after obesity reduction surgery

Samhita Panda, Kapil Sharma

Department of Clinical Neurophysiology, Sir Ganga Ram Hospital, New Delhi, India

Abstract

Osteomalacia and rickets are important reversible causes of debilitating muscular weakness and bony pains in India among all socio-economic strata and at all ages. Osteomalacia after bariatric surgery is documented in literature. Most reports on osteomalacic weakness note myopathic pattern on electromyography. We present the case of a young obese girl from a good socio-economic status who developed severe muscular weakness after sleeve gastrectomy surgery. The patient was found to have osteomalacia with normal vitamin B12 and folate levels. Electrodiagnostic studies demonstrated neuropathic pattern while radiological tests confirmed osteopenia and Looser's zones. Specific vitamin D supplementation was associated with improvement though contribution of other micronutrients in diet cannot be ruled out. Relevance of vitamin D deficiency and urgent need for its correction in the population all over the world and especially in Asia is an emerging health issue. Peripheral motor neuropathy is a rare, seldom reported presentation of osteomalacia.

Key Words

Electromyography, gastric sleeve surgery, osteomalacia, peripheral neuropathy, vitamin D

For correspondence:

Dr. Samhita Panda, Department of Clinical Neurophysiology, Sir Gangaram Hospital, Rajender Nagar, New Delhi - 110 060, India. E-mail: samhitapanda@yahoo.com

Ann Indian Acad Neurol 2013;16:690-2

Introduction

Vitamin D deficiency is very common today in the Indian subcontinent despite being a tropical country with abundant sunshine.^[1] It occurs even in people from good socioeconomic strata. This often leads to the development of osteomalacia with neuromuscular manifestations.^[2,3] Moreover, with the rise of obesity globally and its management by bariatric surgery, cases of osteomalacia are being seen in this population as well. The occurrence of myopathy has been reported as the main electrophysiological finding in medical literature.^[4,5] We report a case of an obese young lady with severe limb weakness after bariatric surgery due to peripheral neuropathy secondary to osteomalacia.

Case Report

A 25-year-old lady from a good socio-economic background presented with generalised body pains, cramps in limbs,

paresthesias, and difficulty in getting up and climbing stairs for 10 days. She had undergone gastric sleeve surgery for morbid obesity 3 months before presentation. She was having recurrent vomiting and reduced oral intake after the surgery. Her weight had reduced by 21 kg over 2½ months after surgery. She had no co-morbidities. At the age of 11 years, the patient had experienced difficulty in walking and climbing stairs associated with body pains for the 1st time. She had genu valgum deformity and was chair bound for 2-3 months. At that time also, she was diagnosed to have osteomalacia. Neurophysiological evaluation had revealed a myopathic pattern. She had recovered completely with vitamin D and calcium supplementation. A similar episode a few years later also recovered with the same treatment.

At the time of current admission, her general physical examination was unremarkable except for obesity and mild pedal edema. Cardiorespiratory and abdominal examination was normal. Neurological examination revealed a waddling gait, hypotonic limbs without wasting and upper and lower limb weakness. Power was: upper limbs-3-4/5 proximally, 4-/5 distally; lower limbs-3/5 proximally, 4+/5 distally. Deep tendon reflexes were normal in upper and sluggish - Medical Research Council (MRC) grade 1 in lower limbs. There was no cranial nerve weakness and sensory examination was normal. A provisional diagnosis of limb girdle weakness of probable inflammatory or nutritional etiology was made.

Laboratory investigations including hemogram, vitamin B12 (938 pg/mL; reference range: 220-960 pg/mL), and

Access this article online

Quick Response Code:



Website:

www.annalsofian.org

DOI:

10.4103/0972-2327.120466

folate levels (5 ng/mL; reference range: 3-17 ng/mL) were unremarkable. However, serum calcium was 7.8 mg/dL, alkaline phosphatase 214 IU/L and parathormone level was raised (232 pg/mL; reference range: 14-72 pg/mL). The level of 25-hydroxy vitamin D was significantly low (2.58 ng/mL; reference range: 30-100 ng/mL). X-ray pelvis showed Looser zones in the pelvic bones [Figure 1a]. Bone densitometry testing by a Dual energy X-ray absorptiometry (DEXA) scan showed a Frisk score of 6.9 (normal: 4.7 ± 1.4) suggestive of osteoporosis [Figure 2]. The diagnosis of post-gastric sleeve surgery with osteomalacic limb girdle weakness and hypoalbuminemia was made. Electrodiagnostic test carried out 3 months after surgery revealed reduced conduction velocities in the motor nerves of the lower limbs with normal sensory potentials suggestive of symmetrical motor polyneuropathy [Table 1]. Electromyography (EMG) showed more proximal involvement with active denervation in the form of positive sharp waves and fibrillations in both vastii lateralis, high amplitude, and long duration motor unit potentials with decreased recruitment in proximal and distal muscles of both lower limbs consistent with neurogenic pattern of involvement.

She was started on a high protein, calcium, and vitamin rich diet and vitamin D and calcium supplementation. On evaluation after 1 week of treatment initiation, she had subjective improvement of 40% with respect to cramps and general well-being and had improved clinically by 10-15%. Power in the limbs had increased by one grade and mobility was better. Three months later, pain had subsided and she had only distal numbness over tips of thumb and index fingers. Power and mobility had further improved and finally she regained complete recovery of strength after 7 months of initiation of treatment. Nerve conduction parameters also normalized [Table 1]. Serum levels of albumin, calcium, alkaline phosphatase, 25-hydroxy vitamin D, and parathormone returned toward normal. X-ray pelvis on follow-up showed disappearance of the Looser's zones [Figure 1b]. Thus, this young female who had presented with severe degree of proximal weakness due to osteomalacia unmasked after bariatric surgery, finally recovered with treatment with vitamin D and calcium therapy-clinically, biochemically and neurophysiologically.

Discussion

Osteomalacia and rickets are important remediable causes of muscle weakness and generalised body pains commonly

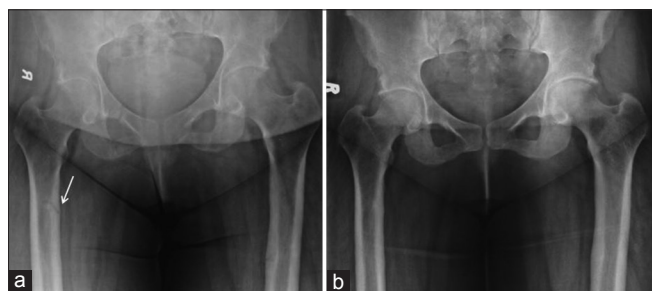


Figure 1: X-ray of pelvis showing (a) looser zones in the femur and (b) disappearance of the same after treatment

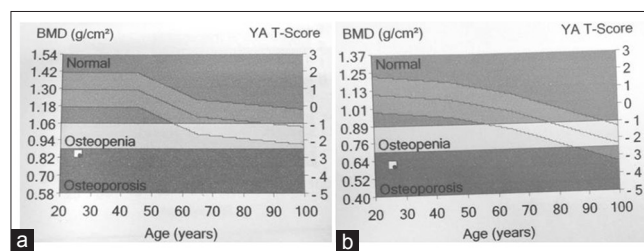


Figure 2: DEXA scan showing osteopenia at time of diagnosis in (a) lumbar spine and (b) neck of femur

Table 1: Nerve conduction parameters at presentation and 6 months after treatment

Nerves	Latency (ms)	Amplitude (mV)	Conduction velocity (m/s)	F wave (ms)
A (baseline)				
Rt Tibial- Ankle	3.2	17.3	32.6	39.8
Popliteal fossa	12.1	11.4		
Lt Tibial- Ankle	4.0	16.1	35.3	39.7
Popliteal fossa	12.5	11.5		
Rt Peroneal- Ankle	4.0	1.1	38.5	39.9
Fibular head	10.5	0.5		
Lt Peroneal- Ankle	3.2	2.0	36.0	37.7
Fibular head	10.7	0.8		
Rt Sural	4.1	11	41.9	
Lt Sural	3.2	10	54.2	
B (after 6 months of treatment)				
Rt Tibial- Ankle	2.5	9.2	41.4	42.0
Popliteal fossa	11.2	7.4		
Lt Tibial- Ankle	3.5	10.3	41.9	40.8
Popliteal fossa	12.1	8.6		
Rt Peroneal- Ankle	2.7	1.4	42.7	41.6
Fibular head	10.2	0.6		
Lt Peroneal- Ankle	3.2	1.7	42.5	41.6
Fibular head	10.5	0.8		
Rt Sural	2.0	6.1	59.9	
Lt Sural	2.6	4.0	60.0	

seen in our country and globally.^[1,4] Though previously thought to be predominant among low socio-economic strata, research has shown osteomalacia to be equally prevalent in people from good socio-economic background and seemingly well-nourished groups.^[6] Moreover, the rise of bariatric surgery for obese patients has also seen a rise in osteomalacia and other nutritional deficiencies in this set of patients.^[7] Osteomalacia is generally believed to cause proximal muscle weakness due to myopathy with EMG manifestations of short duration, small amplitude motor unit potentials with early recruitment and without denervation.^[5,8] Osteomalacia may present with marked weakness even in the presence of normal serum calcium due to secondary hyperparathyroidism. Our patient was a young obese girl who developed osteomalacia mainly due to decreased nutritional intake presenting after sleeve gastrectomy. She belonged to very good socio-economic strata and presented with severe degree of muscular weakness, which had immobilized her.

In a rare electrophysiological presentation of osteomalacic weakness, there was evidence of neuropathy with chronic neurogenic changes along with active denervation that reverted after treatment. Clinical examination also suggested probable neuropathy in view of diminished deep tendon reflexes in contrast to the brisk reflexes seen in osteomalacic myopathy.^[4] There are not many illustrations in medical literature, which reveal neurogenic involvement of muscles in osteomalacia.^[2,3] Although most studies demonstrate a myopathic pattern, Mallette *et al.* did demonstrate neurogenic involvement in four patients with osteomalacia as demonstrated by EMG as well as muscle biopsy and attributed the muscular weakness in these cases to be secondary to a neuropathic rather than myopathic process.^[2] Except in a single patient, no denervation such as fibrillations or positive sharp waves were found. Our case had similar features with neurogenic involvement of proximal and distal muscles of the lower limbs. Nerve conduction showed motor polyneuropathy in the lower limbs. EMG demonstrated proximal denervation, high amplitude potentials of decreased number on voluntary effort. Despite having severe weakness secondary to osteomalacia, no myopathic changes were recorded on EMG.

This is in contrast to the EMG findings by Irani who found absence of denervation, presence of normal motor nerve conduction velocity and myopathic potentials in the proximal muscles in osteomalacia.^[8] Skaria *et al.* has also reported the presence of reduced nerve conduction velocity in most patients of the series though there were myopathic potentials without denervation on EMG and proposed a lack of correlation between osteomalacia and neuropathy.^[3] Our patient's weakness resolved completely with vitamin D and calcium supplementation with resolution of biochemical abnormalities and nerve conduction parameters. Thus, it is evident from this case that the neuromuscular syndrome associated with osteomalacia does not always result in myopathy and neuropathic mechanism for the weakness should be considered.

Laparoscopic sleeve gastrectomy has recently been proposed as a simple technique to tackle morbid obesity and is being increasingly performed.^[7] The nervous system may be involved at the central and peripheral level. Mononeuropathy, polyneuropathy, and radiculopathy have been reported after

bariatric surgery.^[9] Malnutrition and long-term nutritional consequences of such procedures may be responsible. Among the multiple micronutrient deficiencies, the most likely candidates found are vitamin B12, folate, zinc, thiamine, copper, vitamin A, and vitamin E deficiencies.^[10] Concomitant nutritional deficiency of vitamin B12 and folate were duly ruled out in our patient and osteomalacia with vitamin D deficiency associated with bone changes and peripheral neuropathy were found with documented improvement after adequate supplements. In a study on nutritional imbalances after bariatric surgery, 39% had vitamin D deficiency despite supplements. Changes in bone metabolism and long-term risk of osteoporosis should be considered due to lack of vitamin D, secondary hyperparathyroidism and low albumin levels in the post resection state as seen in our patient. Inadvertent supplementation of other vitamins and minerals in the diet after diagnosis may have contributed to added improvement in the nutritional status and symptoms of the patient and is the limitation of this report.

Conclusion

This report highlights the rare presentation of osteomalacia with peripheral neuropathy, which responds favorably to adequate supplementation. It also emphasizes the need for meticulous evaluation of the nutritional status of patients prior to and periodically after obesity reduction surgeries like sleeve gastrectomy. This is more important in the Indian subcontinent where nutritional bone disease has been found to be more frequent than previously thought.

References

1. Teotia SP, Teotia M. Nutritional bone disease in Indian population. *Indian J Med Res* 2008;127:219-28.
2. Mallette LE, Patten BM, Engel WK. Neuromuscular disease in secondary hyperparathyroidism. *Ann Intern Med* 1975;82:474-83.
3. Skaria J, Katiyar BC, Srivastava TP, Dube B. Myopathy and neuropathy associated with osteomalacia. *Acta Neurol Scand* 1975;51:37-58.
4. Smith R, Stern G. Myopathy, osteomalacia and hyperparathyroidism. *Brain* 1967;90:593-602.
5. Prineas JW, Mason AS, Henson RA. Myopathy in metabolic bone disease. *Br Med J* 1965;1:1034-6.
6. Goswami R, Gupta N, Goswami D, Marwaha RK, Tandon N, Kochupillai N. Prevalence and significance of low 25-hydroxyvitamin D concentrations in healthy subjects in Delhi. *Am J Clin Nutr* 2000;72:472-5.
7. Aarts EO, Janssen IM, Berends FJ. The gastric sleeve: Losing weight as fast as micronutrients? *Obes Surg* 2011;21:207-11.
8. Irani PF. Electromyography in nutritional osteomalacic myopathy. *J Neurol Neurosurg Psychiatry* 1976;39:686-93.
9. Thaisethawatkul P, Collazo-Clavell ML, Sarr MG, Norell JE, Dyck PJ. A controlled study of peripheral neuropathy after bariatric surgery. *Neurology* 2004;63:1462-70.
10. Rudnicki SA. Prevention and treatment of peripheral neuropathy after bariatric surgery. *Curr Treat Options Neurol* 2010;12:29-36.

How to cite this article: Panda S, Sharma K. Osteomalacia induced peripheral neuropathy after obesity reduction surgery. *Ann Indian Acad Neurol* 2013;16:690-2.

Received: 19-02-13, **Revised:** 08-03-13, **Accepted:** 02-04-13

Source of Support: Nil, **Conflict of Interest:** Nil