Surgical decompression of the mass was performed by postero-lateral approch. The tumor was encapsuled and adherent to the periosteum of the radius [Figure 3]. The tumor was totally removed without damage to the PIN branches [Figure 4]. The histological examination of the mass confirmed the diagnosis of lipoma. Follow-up at four months showed no motor deficits and no local recurrence of tumors.

Non traumatic palsy of the PIN is rare and PIN palsy caused by lipoma commonly occurs at the level of elbow.[1] The lipoma may be intramuscular or parosteal.[2-5] Only a few cases of paralysis of PIN secondary to parosteal lipoma of proximal radius have been reported.[4,5] The parosteal lipoma may cause palsy of PIN because of the near anatomical relationship of the nerve in this location.[5] The other mass lesions that can cause PIN palsy include: ganglion,[6] and soft tissue chondroma of the elbow.[7] The parosteal lipoma is extremely rare and account for 0.3% of all lipoma. These lesions are often solitary, slow growing, and adherent to periosteum. The histological features are similar to any superficial lipoma.[1] Typically, patients with PIN palsy due to compressive lesion often present with insidious onset symptoms including weakness of digital extension and deep forearm proximal swelling. As the level of nerve compression is distal to the radial nerve division into PIN and superficial branches. Pre-operative MRI is very useful as it can exactly localize the tumor and evaluate its relationship with the neighboring structures, especially the PIN. Presence of muscle involvement suggests the diagnostic possibility of liposarcoma.[8] Early surgical excision of parosteal lipoma is recommended to ensure good recovery of the nerve paralysis.[4,5] Surgical access for the decompression of the PIN can be posterolateral or anterior. Using the anterior approach, the dissection is easy and allows a direct vision of parosteal lipoma and minimizing the risk of damage of main nerve and also muscular branches.[4]

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Blepharospasm and vitamin B12 deficiency

Sir,

Blepharospasm is a form of focal dystonia characterized by involuntary contractions of the orbicularis oculi muscles resulting in bilateral closure of the eyes and is of two types: primary (unknown cause) and secondary (a known cause). The primary form has a gradual onset and affects mainly women in the middle age, whereas the secondary forms are associated with lesions in the basal ganglia, brainstem and thalamus. The pathophysiology is unknown.[1]

Vitamin B12 deficiency can result in various neurological syndromes: neuropathy, myelopathy, dementia, cerebellar ataxia, optic atrophy and mood disturbances.[2] Movement disorders are not usually the features of B12 deficiency in the adults[3] Whereas infants,with B12 deficiency can present with chorea, tremor, twitches, myoclonus and other abnormal movements.[4] Blepharospasm as a manifestation of B12 deficiency in adults has not been reported. We describe a patient with severe blepharospasm in whom investigations revealed low vitamin B12 levels, elevated homocysteine levels as well as other laboratory features supporting B12 deficiency.

A 51-year-old male presented with three months history of difficulty to keep eyes open. There was no history of any drug exposure. His medical history was otherwise normal. The symptoms progressed in severity but with no functional blindness. Physical examination was normal, including blood pressure. Neurological examination revealed bilateral blepharospasm grade 3 (severity, frequency) on the 0–4 Jankovic Rating Scale. He had in addition length dependent loss of vibration in both the lower and upper limbs. Computer tomography (CT) of the brain and electroencephalogram were essentially normal. A cerebrospinal fluid analysis,

including neurofilament and tau protein was normal. Laboratory evaluations revealed: macrocytic anemia; MCV of 125 fL (normal range, 80–100); hemoglobin of 88 g/L (normal range, 115–150); and plasma level vitamin B12 value of 16 pmol/L (normal range, 150–650). Plasma homocysteine was 43 µmol/L (normal range <15 µmol/L) and serum folate was 24 nmol/L (normal range 7–40 nmol/L). Serum methylmalonate and holotranscobalamine were not measured. The bone marrow was hypercellular, which is consistent with B12 deficiency. Tests for malabsorption were normal. Anti-intrinsic factor antibody serology was positive and antral biopsy was not done. Nerve conduction studies were normal. Other investigations for other causes of movement disorders were essential normal or within normal range.

A clinical diagnosis of blepharospasm secondary to B12 deficiency was made and he was initiated on intramuscular cyanocobalamin 1 mg daily for 10 days and then weekly, no other medications were prescribed. Following the treatment tests at eight weeks revealed: B12 >1500 pmoll/L, homocysteine 9.7, MCV 90 fL, hemoglobin 128 g/L and folate 21 nmol/L. At three months follow-up the blepharospasm had improved significanlty (grade 1). At 9 moth follow-up he was totally free of blepharospasm. The vibration sense in the hands and feet had also improved. He is on life-time maintenance dose of vitamin B12 therapy and he felt well at further follow-up after 3 years.

Extrapyramidal involvement due to vitamin B12 deficiency in adults is rare. In adults with vitamin B12 deficiency, focal dystonia, chorea, parkinsonism, myoclonus and even ataxia have been documented mostly as single case reports.[5-8] There are no previous reports of blepharospasm in adults associated with vitamin B12 deficiency. The mechanism of extrapyramidal involvement in vitamin B12 deficiency is poorly understood. The following facts may contribute to our understanding. Vitamin B12 deficiency is the most common cause of hyperhomocysteinemia and homocysteine is required for the methylation of methionine. Homocystein has N-methyl-D-aspartate agonist action and homocystein by its action on the thalamocortical pathway may cause excitatory activity in the basal ganglia thus resulting in dystonia and chorea.[9] In addition, the excess methyl levels in B12 deficiency increase the levels of methyltetra hydrofolate, which has kainic acid agonistic action. In experimental animals, kainate produces damage similar to that seen in Huntington's disease.[10] Also, methylmalonic acidemia, an inborn error of metabolism, usually presents with acute extrapyramidal syndrome in infants, and some of these children respond to vitamin B12 therapy.[11] Müller *et al*. found elevated plasma levels of homocysteine in patients with dystonia.[12] The authors suggest that high level of homocysteine may be a contributing factor for the onset and severity of dystonia and they also recommend routine testing of plasma homocysteine and treating these patients for hyperhomocysteinemia.

It remains unclear whether cobalamin deficiency and blepharospasm in this single case is just coincidental or causally related. Both cobalamin deficiency and blepharospasm are fairly common in older patients. However, the reversibility of the syndrome with only cyanocobalamin supplementation is striking suggesting a possible causal relation between the two. In conclusion, blepharospasm can be a rare manifestation of vitamin B12 deficiency, which is reversible with therapy. Vitamin B12 levels and homocysteine levels should be tested in patients with blepharospasm in whom there is no obvious cause for blepharospasm.

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