McLeod syndrome and acanthocytosis

Sir,
We read with interest the article titled "McLeod syndrome: Report of an Indian family with phenotypic heterogeneity" by Chakravarty A, *et al*.[1]

The authors describe three brothers diagnosed as having McLeod syndrome with phenotypic variations. The proband had confirmed diagnosis of MLS by hematological evidence of acanthocytosis, immunohematological, and molecular genetic studies. The authors state that this appears to be the first report of MLS from India.

We wish to share and bring to the notice of authors that a case report of McLeod syndrome (a variant of neuroacanthocytosis) was published in the documentation section of JAPI.[2] Some of the details of case reported were as follows:

A 40-year-old male patient presented with abnormal involuntary generalized choreiform movements since 8 years. The patient also had involuntary movements involving the orofacial region, especially the tongue, lips, and other facial muscles with grimacing and difficulty in swallowing because of constant movement of the tongue. He also developed generalized tonic clonic seizures 6 years back, which were controlled by using Carbamazepine. The family history was negative upto the preceding two generations. Peripheral blood smear, done on two occasions, was positive for acanthocytes (more than 5%). Serum creatine phosphokinase (CPK) was 1042 IU/L. Kell antigen analysis revealed a negative high-frequency Kell antigen. Electrophysiological studies showed motor axonal degeneration in both lower limbs. Sural nerve biopsy was suggestive of chronic axonal neuropathy with regenerative activity. MRI of the brain revealed enlargement of frontal horn of lateral ventricles, atrophy of the caudate nucleus with hypointensity signals in the basal ganglia on T_1-weighted images. On T_2-weighted images, hyperintense lesions were seen in the basal ganglia.

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