

Commentary

The article presented by Netto *et al.* is a case of pseudohypertrophy of the entire right lower extremity due to a neurogenic etiology.^[1] However, it is important for the readers to realize that there are also similar cases reported in the literature that result in neurogenic atrophy.^[2-4] Although diagnostic modalities such as EMG and imaging direct the readers to a S1 radiculopathy secondary to a sacral lipoma (manifested clinically in the distal musculature), there are likely other etiologies to explain the pseudohypertrophy witnessed in the right proximal musculature. A combination of tethered cord and radiculopathy better explains the underlying etiology. The principal etiology of tethered cord syndrome has been demonstrated to be mainly secondary to the longitudinal traction of the caudal end of the spinal cord.^[3,4-6] A sacral lipoma can be a contributing factor to a tethered cord syndrome. The pathophysiology of the neurologic deficits seen is caused by the effect of spinal cord tethering or compression produced by the presence of a lipoma. In animal models, it has been illustrated that this mechanical pull leads to changes in blood flow and to hypoxemia, with resultant reduction in oxidative metabolism that can clinically manifest as neurologic deficits.^[4] The natural history of the spinal lipoma is poorly defined, and surgical management of this lesion can be controversial.^[7]

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