Letters to Editor

The abrupt onset of CNS symptoms in SLE patients presents a diagnostic and therapeutic challenge. As reported on a recent review of 323 SLE patients, the most common CNS presentations in descending order of frequency were headache, cerebrovascular disease, mood disorders, cognitive dysfunction, seizures, psychosis, anxiety disorder, and acute confusional state. [2] These symptoms may be either due to direct immune mediated injury of the CNS or secondary events (i.e., related to complication of SLE or its treatment).

PRES is an acute or subacute, progressive reversible neurologic syndrome which can mimic neuropsychiatric SLE presentations. Hence, in SLE patients with acute neurologic symptoms in the setting of hypertension, renal insufficiency and immunosuppressive treatment, PRES should be considered as the cause of CNS abnormalities. [3]

A total of 30 cases of PRES in SLE patients have been reported in the literature but the true prevalence is unclear. [4] In recent years, another MR technique, echo-planar DWI findings are useful in distinguishing PRES from neuropsychiatric SLE presentations. Regions with vasogenic edema show marked hyperintensity on ADC and mostly iso or hypointensity on DWI. [5]

Although the lesions of our patients challenge with this knowledge, according to Ay et al., an increase in T2 signal within regions of vasogenic edema (T2 shine-through) could cause slight DWI hyperintensity. [5]

In conclusion, PRES should be recognized in patients with SLE presenting with CNS findings. It is extremely important to distinguish this syndrome from other causes, since it is reversible and readily treated by controlling blood pressure, discontinuing the offending immunosuppressive agent or decreasing the dose and controlling of seizure activity.

Necioglu Orken Dilek, Kenangil Gulay, Ur Emel, H. Forta
Department of Neurology, Sisli Etfal Education and Research Hospital, Istanbul.
E-mail: dilek.necioglu@gmail.com

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References

Midbrain infarct presenting as isolated medial rectus palsy

Sir,
A 30-year-old man, labourer by occupation, presented with sudden onset of giddiness followed by diplopia with horizontal separation of images on looking to
Letters to Editor

Oculomotor nerve has two motor nuclei, the main motor nucleus and the accessory parasympathetic nucleus. The main oculomotor nucleus which is situated in the anterior part of the gray matter surrounding the cerebral aqueduct of the midbrain at the level of superior colliculus supplies all the extrinsic muscles of the eye except the superior oblique and lateral rectus muscle. It consists of one unpaired central caudal nucleus for bilateral levator palpebrae superiors and four paired subnuclei.[1] The subnucleus for superior rectus muscle is situated dorsomedially and decussation of the fibers to the superior rectus takes place within the oculomotor nuclear complex; thus, lesions affecting the nucleus may simultaneously involve ipsilateral superior rectus subnuclei as well as crossing fibers resulting in bilateral superior rectus muscle palsy.[2] Ventral to the superior rectus subnuclei lies the subnuclei for ipsilateral inferior rectus followed by intermediate subnuclei for ipsilateral inferior oblique and the most ventrally situated subnuclei is for ipsilateral medial rectus.[3] Third nerve nuclear lesions cause weakness of bilateral superior rectus muscle ipsilateral inferior oblique, inferior rectus, medial rectus, with bilateral partial ptosis (due to involvement of central caudal subnuclei supplying both levator palpebrae superiors). This is a case of nuclear oculomotor nerve palsy with an unusual presentation due to strategic location of infarct in the rostral midbrain involving the medial rectus subnuclei which is situated most ventrally and can be diagnosed with diffusion weighted imaging (DWI).[3] Unilateral ocular palsy is commonly seen with lesions in the orbit or from muscular diseases and rarely from a third nerve nuclear lesion, though inferior oblique muscle palsy caused by involvement of intermediate subnuclei[4] and isolated inferior rectus palsy due to ipsilateral involvement of dorsally situated subnuclei or fascicular lesions have been described.[5] With use of DWI and other multimodality MRI the probability of picking up midbrain infarcts causing isolated oculomotor palsies have increased.

Simerpreet Bal, Vivek Lal, Dheeraj Khurana, Sudesh Prabhakar
Department of Neurology, Postgraduate Institute of Medical Education and Research, Chandigarh - 160 012, India.
E-mail: vivekl44@yahoo.com
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A case of sporadic periodic hypokalemic paralysis with atypical features: Recurrent differential right brachial weakness and cognitive dysfunction

Sir,

We present a case of 28-year-old man admitted with an episode of fall due to weakness in all four limbs, with differential weakness of right upper limb that was associated with cognitive dysfunction. He had experienced multiple episodes of such attacks in the past 10 years. The relatives also noticed that after such an attack the patient remained confused for few hours. He did not give history of palpitations, hand tremors, or heat intolerance. His computed tomography scan of head and electroencephalogram suggested no abnormality. MRI of the brain was not done. A short review of systems was otherwise noncontributory. Neurological examination revealed the overall power in limbs was about 4/5, but in the right upper limb it was found to be 1/5. There was generalized hyporeflexia with a flexor plantars. His score on mini mental state examination (MMSE) was 21/30. The remainder of the physical examinations was noncontributory. Serum electrolyte: Potassium 1.7 mEq/L (normal range: 3.5-5.3 mEq/L). He was investigated for renal loss of potassium which was normal. His thyroid stimulating hormone level (TSH) was reduced to 0.05 ng/dL (normal range: 0.45-4.5 ng/dL) indicating a hyperthyroid state.

He was given potassium supplementation after which his symptoms recovered completely along with improvement of cognitive functions without any residual weakness. His nerve conduction velocity (NCV) done during a nonparalyzed state was normal.

Periodic hypokalemic paralysis is often unrecognized when first encountered because of its relative rarity. Atypical presentations of severe hypokalemia may be in the form of total paralysis including respiratory, bulbar, and cranial musculature. Some patients complain of muscular weakness, especially of the lower extremities, while marked and generalized weakness of skeletal muscles is common with more severe potassium depletion, but they may present as weakness of specific group of muscles. Sudden deaths from respiratory failure and arrhythmia like ventricular tachycardia and fibrillation have been reported. Our case was typical in presence of marked generalized weakness with differential weakness of right upper limb, reason of which cannot be explained. The sensations and level of consciousness are generally unaffected. Our patient had history of cognitive disturbance during the attack with hyporeflexia in all four limbs. Cognitive disturbances with hypokalemia have not been reported in literature. Both of the problems improved completely after potassium replacement which is a prerequisite for diagnosis of this disease.

A. N. Joshi, A. P. Jain, A. D. Bhatt, S. Kumar
Department of Medicine, Mahatma Gandhi Institute of Medical Sciences, Sewagram, Wardha, Maharashtra, India.

E-mail: dr.anjalijoshi@gmail.com

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