Norovirus-induced gastroenteritis presenting with reversible quadriparesis in an adult suggesting transient intramyelinic edema: A rare case report

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ABSTRACT

Neuromuscular weakness with no plausible cause other than critical illness has been labeled as an intensive care-acquired weakness. There are myriad causes of neuromuscular weakness in intensive care unit (ICU). Here, we present the case of an adult with a diagnosis of acute gastroenteritis due to *Norovirus* with acute kidney injury and sepsis presenting with acute flaccid quadriparesis which recovered fully before ICU discharge. Magnetic resonance imaging brain of this patient suggested white matter diffusion restriction with normalization of diffusion abnormality after 2 months, suggesting "transient intramyelinic edema." We highlight that *Norovirus* gastroenteritis can present as myelin sheath edema causing quadriparesis mimicking other etiologies for neuromuscular weakness in ICU and discussed the various differential diagnoses of white matter demyelination and diffusion restriction in this case report.

Key words: *Gastroenteritis, Myelin sheath edema, Norovirus, Toxic leukoencephalopathy*

Neuromuscular weakness with no plausible cause other than critical illness has been labeled as an intensive care-acquired weakness (intensive care unit [ICU]-AW). There are many differentials for neuromuscular weakness based on the site of pathology such as muscle (critical illness myopathy, polymyositis, and dermatomyositis), neuromuscular junction (myasthenia gravis and snakebite), peripheral neuropathies (acute inflammatory demyelinating polyneuropathy, porphyria, and critical illness neuropathy), anterior horn cell diseases (motor neuron disease), or spinal cord disorders (trauma, hematoma, abscess, and transverse myelitis), of which critical illness neuromyopathy (CINM) is of common occurrence [1]. In some clinical situations, mimics of CINM should also be considered.

Hereby, we present a case with acute gastroenteritis complicated by acute kidney injury (AKI) progressing to Stage 3 along with septicemia and multiorgan failure presenting to us with a clinical picture suggestive of CINM. This case is presented as it is a rare complication following viral gastroenteritis diagnosed with magnetic resonance imaging (MRI) brain and treated with steroids, globulins although few case reports had a spontaneous recovery like ours [2-4].

CASE REPORT

A 45-year-old male, a businessman by profession, presented to the department with chief complaints of loose stools around 10–15 episodes followed by 4–5 episodes of non-bilious and non-projectile vomiting for 2 days. There was no history of fever, altered sensorium, seizures, cough, breathlessness, recent trauma, or surgery. He was a non-smoker and non-alcoholic without any history of drug abuse.

The patient was initially admitted to a private hospital. The patient was conscious, oriented, and afebrile, without any focal neurological deficit but with circulatory shock in metabolic acidosis and AKI (heart rate: 130 bpm, blood pressure 110/60 mmHg on norepinephrine at the rate of 0.4 mcg/kg/min, and respiratory rate: 20 breaths per min).

Blood gas showed a pH of 7.30, paO_2 of 120 mmHg (FiO_2: 0.5), $paCO_2$ of 25 mmHg, HCO_3 of 15 meq/dl, Na: 130 meq/L, K: 3.9 meq/L, Cl: 100 meq/L, anion gap: 20, and lactate: 25 mg/dL. Other laboratory investigations were hemoglobin: 7.2 gm/dL, total leukocyte count (TLC): 15,000 cells/mm^3, platelet count: 120,000 cells/mm^3, blood urea nitrogen: 56 mg/dL, serum creatinine: 3.0 mg/dL, sodium: 135 meq/L, potassium: 3.0 meq/L, and random blood sugar: 180 mg/dL, liver function tests: Total bilirubin: 1.2 mg/dL, direct bilirubin: 0.3 mg/dL, aspartate transaminase/alanine transaminase/alkaline phosphatase: 115/150/75 IU/ml, respectively, and international normalized ratio 1.4.

Human immunodeficiency virus, hepatitis B surface antigen and hepatitis C antibody were negative. Typhoid and other tropical fever (malaria/dengue/scrub) workup were negative. Urine routine and microscopy revealed no casts or red blood cells. Stool for routine microscopy was done which revealed

no parasites or cysts or ova. Stool for the pan-virus panel (reverse transcriptase polymerase chain reaction) was positive for *Norovirus* RNA. On day 2 of admission, the patient became drowsy and developed respiratory failure requiring intubation and mechanical ventilation. As AKI progressed to Stage 3, dialysis therapy was given. After 1 week of management in that hospital, the patient was shifted to our center.

At admission to our center, the patient was in shock with moderate acute respiratory distress syndrome (ARDS) and improving AKI. Antimicrobials were continued according to sensitivity reports of the previous hospital (extensively drug-resistant *Klebsiella pneumoniae* in blood cultures sensitive only to colistin). Other investigations were within normal limits except high TLC counts and procalcitonin (10 ng/ml). After 5 days of ICU care, as the shock improved, the sedation was reduced and antimicrobials were stopped.

Neurological examination revealed low power and flaccid tone in all limbs, medical research council sum score (Medical Research Council [MRC] score 1–2) in all muscle groups, along with reduced deep tendon reflexes. This low muscle power and Glasgow coma scale persisted for another 5 days. Our first suspicion is of ICUAW and we planned for electromyographic studies. However, based on his background history of AKI and dyselectronemia, osmotic demyelination syndrome (ODS) was considered. MRI Brain was done which revealed hyper intense lesions in the white matter of bilateral cerebral hemispheres in T2 weighted and T2 fluid attenuation inversion recovery (FLAIR) sequences and central pons along with diffusion restriction in the affected (Fig. 1).

Susceptibility-weighted imaging (SWI) showed multiple microhemorrhages in bilateral cerebral hemispheres. No post-contrast enhancement was noted and sinus flow voids were maintained. After viewing these changes, cerebrospinal fluid analysis was done suspecting viral encephalitis or its sequel acute disseminated encephalomyelitis (ADEM), which was within normal limits (cell count: Nil, protein: 60 mg/dL, and glucose: 100 mg/dl with serum glucose: 80 mg/dl and bacterial cultures: Sterile). The patient sensorium and muscle power improved (MRC from Grade 1 to Grade 4) progressively over 2 weeks of ICU stay after which he was discharged home.

After 1 month of discharge, he was able to do his daily routine activities. Repeat MRI brain showed complete normalization of diffusion abnormality. The pontine hyperintensity on T2/T2FLAIR also resolved; however, residual white matter hyperintensities were noted in cerebral hemispheres. Few residual microhemorrhages also noted on SWI (Fig. 2).

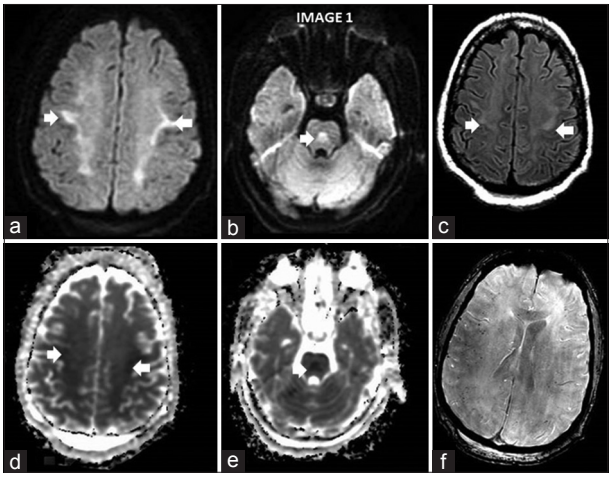

Figure 1: T2W and T2 fluid attenuation inversion recovery hyperintense lesions in white matter of bilateral cerebral hemispheres and central pons along with diffusion restriction in the affected areas (represented as bold white arrows). Susceptibility-weighted imaging showed multiple microhemorrhages in bilateral cerebral hemispheres (f)

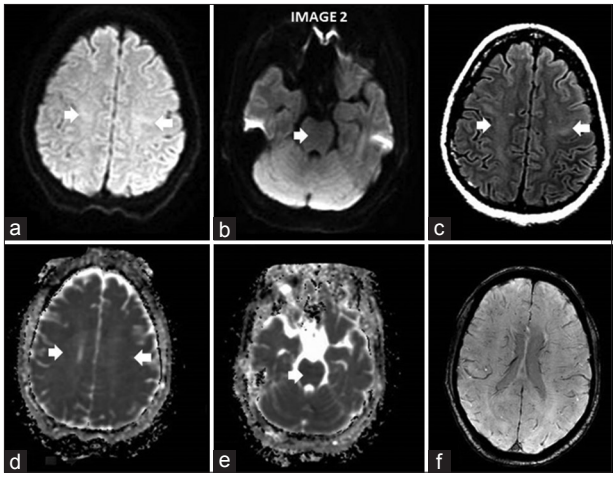

Figure 2: Complete normalization of diffusion abnormality. The pontine hyperintensity on T2 fluid attenuation inversion recovery sequence also resolved; however, residual white matter hyperintensities were noted in cerebral hemispheres (represented as bold white arrows) and few residual microhemorrhages also noted on susceptibility weighted imaging (f)

DISCUSSION

Diffusion restriction of the brain in MRI is a characteristic feature of acute ischemic stroke-induced cytotoxic edema. However, diffusion restriction can also be seen in non-ischemic conditions such as genetic, metabolic disorders, drug toxicity, and trauma [5-7]. The cause of reduced diffusion in the white matter may arise from intramyelinic edema and resultant myelin vacuolation, cytotoxicity through capillary endothelial injury, or direct toxic demyelination. Intramyelinic edema, also called "myelin sheath edema," occurs in intramyelinic clefts along the white matter or within the myelin sheath itself. Such transient reversible pathology has been found in viral infections caused by rotavirus and *Norovirus*-induced gastroenteritis [2-4]. Although exact pathogenesis is not known, the release of cytokines from injured microglia, excessive brain dehydration, and metabolic compromise mediated by fluid balance system has also been implicated as possible etiology.

Other differentials of the condition were CINM (never reported with MRI brain changes), ODS (pontine, pyramidal tracts, and cerebellar peduncles hyperintensities, rarely shows any diffusion restriction) [8], ADEM (multiple areas of brain

usually bilateral subcortical white matter along with variable contrast enhancement) [9], hypoxic-ischemic encephalopathy (gray matter involvement predominantly) [10], and posterior reversible encephalopathy syndrome (reversible symmetrical subcortical vasogenic edema of posterior parietal or occipital lobes) [11]. In all likelihood based on imaging and recovery, our patient developed cytotoxic edema manifesting as "transient intramyelinic edema" mimicking other etiologies of neuromuscular weakness in ICU. MRI brain can be both diagnostic and of prognostic value in such rare cases. Virus-related encephalopathy has been previously reported with reversible restriction in diffusion-weighted sequences in splenium of corpus callosum and as abnormal signals in the cortex of opercular part, insula on FLAIR images which recovered either spontaneously overtime or with steroid pulse therapy with immunoglobulins in few cases [2-4].

Based on the imaging and recovery pattern, our patient could have developed cytotoxic edema manifesting as transient intramyelinic edema following infective gastroenteritis due to *Norovirus* and had a spontaneous recovery.

CONCLUSION

Myelin sheath edema can present after *Norovirus* gastroenteritis manifesting as quadriparesis mimicking other etiologies for neuromuscular weakness in ICU. MRI brain can help in the diagnosis of this condition which reveals normalization of restricted diffusion after some time with full recovery.

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REFERENCES

1. Maramattom BV, Wijdicks EF. Acute neuromuscular weakness in the intensive care unit. Crit Care Med 2006;34:2835-41.
2. Karampatsas K, Spyridou C, Morrison IR, Tong CY, Prendergast AJ. Rotavirus-associated mild encephalopathy with a reversible splenial lesion (MERS)-case report and review of the literature. BMC Infect Dis 2015;15:446.
3. Kimura E, Goto H, Migita A, Harada S, Yamashita S, Hirano T, *et al*. An adult norovirus-related encephalitis/encephalopathy with mild clinical manifestation. BMJ Case Rep 2010;2010:Bcr0320102784.
4. Liu WM, Lin CH. A reversible stroke-like splenial lesion in viral encephalopathy. Acta Neurol Taiwan 2013;22:117-21.
5. Moritani T, Kim J, Kirby P. Intramyelinic edema on diffusion-weighted imaging correlated with pathology and pathophysiology. e-Poster Presented at European Congress of Radiology. Iowa City, United States of America; 2013.
6. Citton V, Burlina A, Baracchini C, Gallucci M, Catalucci A, Dal Pos S, *et al*. Apparent diffusion coefficient restriction in the white matter: Going beyond acute brain territorial ischemia. Insights Imaging 2012;3:155-64.
7. Cornelius LP, Kannan B, Saravanan V, Venkatesan EP. Intramyelinic edema in maple syrup urine disease. Ann Indian Acad Neurol 2014;17:211-3.
8. King JD, Rosner MH. Osmotic demyelination syndrome. Am J Med Sci 2010;339:561-7.
9. Lin CH, Jeng JS, Hsieh ST, Yip PK, Wu RM. Acute disseminated encephalomyelitis: A follow-up study in taiwan. J Neurol Neurosurg Psychiatry 2007;78:162-7.
10. Chalela JA, Wolf RL, Maldjian JA, Kasner SE. MRI identification of early white matter injury in anoxic-ischemic encephalopathy. Neurology 2001;56:481-5.
11. Hobson EV, Craven I, Blank SC. Posterior reversible encephalopathy syndrome: A truly treatable neurologic illness. Perit Dial Int 2012;32:590-4.

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