Severe Bradycardia and Hypotension Possibly Induced by Ranitidine

Nausea, vomiting, diarrhea, constipation and rash are the more frequently encountered adverse reactions of ranitidine – a selective histamine H2 receptor antagonist. Only a few cases of cardiovascular side effects have been reported [1-3].

A 10-year-old boy was admitted to our hospital for percutaneous endoscopic gastrostomy (PEG). He had had neurological disability because of neonatal bilirubin encephalopathy. He was receiving diazepam and baclofen dystonia for last six years. Laboratory examination, including whole blood count, blood chemistry results, and thyroid function tests were normal. Percutaneous endoscopic gastrostomy (PEG) tube was inserted. Ranitidine was injected at a dose of 4 mg/kg/day in four divided doses by intravenous route starting from 24 hour after the procedure. After four hours of the first dose of ranitidine, the child was noted to have bradycardia (HR 60/min) and the blood pressure fell to 80/50 mmHg 16 hours after the first dose of ranitidine. At the third day of ranitidine treatment, the heart rate was detected to be 36/min and there was common voltage drop on electrocardiography. Echocardiography was normal. Physical examination revealed no additional findings except bradycardia and hypotension. Ranitidine treatment was stopped. No treatment was given for bradycardia and hypotension because of the good general condition of the patient. Heart rate and blood pressure improved after 12 hours of discontinuation of ranitidine.

H2 receptors are reported to be present in sinus node, atrial and ventricular myocardium as well as gastric mucosa [1]. Cimetidine and ranitidine have been reported to cause significant hypotension in critically ill patients. Though gastric interventions such as PEG insertion may lead to increased vagal tone causing bradycardia, it was not seen until the first dose of ranitidine treatment in present case. Moreover, it resolved following cessation of ranitidine treatment.

Clinicians should always be aware of the possibility of rare but potentially serious cardiovascular adverse events of ranitidine, especially in sick children.

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Subacute Sclerosing Panencephalitis: A Disease Not to be Forgotten

Evaluation a child with encephalitis is difficult due to the similarities in the clinical, imaging and laboratory findings of many forms of autoimmune and infectious encephalitis. Presentation of autoimmune encephalitis in childhood is often subacute, with varied clinical manifestation [1]. However, as it takes time to get the results of antibody tests for autoimmune encephalitis, immunosuppression is often started with a presumed diagnosis of AE. Due to increasing awareness of AE, many primary-care physicians are diagnosing it and starting immunomodulation, which may be detrimental at times.

In past few months, two children presented to us in vegetative state. Both children were diagnosed as AE based on their presentation with fever, behavioral changes and myoclonic jerks/focal seizures. Pulse methylprednisolone was administered to the children with presumed diagnosis of autoimmune encephalitis. There was no improvement on immunotherapy and children deteriorated to vegetative state in next 2-3 weeks. There was no history of measles in these children, and they were vaccinated (one dose of measles vaccine at 9 months). Fundus examination showed hyperemic disc, large whitish subretinal patch over posterior-pole with satellite lesions, and magnetic resonance imaging (MRI) of brain showed subtle asymmetrical hyper-intensities in peri-ventricular white-matter. Based on these findings, Subacute sclerosing panencephalitis (SSPE) was suspected, and subsequently confirmed by raised (1:625)