INTRODUCTION

Convulsive (tonic—clonic) status epilepticus (CSE) is currently defined as a generalised convulsion lasting 30 min or longer, or repeated tonic—clonic convulsions recurring over 30 min without recovery of consciousness between each convulsion. It is the most common neurological emergency in childhood and is life-threatening with risk of neurological sequelae. (Hussain et al, 2007).

Epidemiological studies have suggested that four to eight children per 1000 may be expected to experience an episode of CSE before the age of 15 years and in children with first seizures, 12% present with CSE as their first unprovoked seizure. A recent systematic review of the epidemiology of status epilepticus suggested an incidence of 18—20/10,0000 children per year. Approximately 10—25% of children with epilepsy will have at least 1 episode of CSE. Mortality and morbidity due to CSE have declined during the last two decades (**Hussain et al, 2007**).

SE can be classified as generalized, partial, nonconvulsive and neonatal Generalized tonic clonic status is the most common form and carries the highest mortality and morbidity. It can also be differentiated on the basis of the underlying cause. A primary central nervous system (CNS) disorder or a metabolic abnormality may result in an episode of SE. Some such important conditions are trauma, infections such as meningitis or encephalitis, hypoxic-ischaemic. (Behera et al, 2005).

Status epilepticus (SE) is a life-threatening emergency that requires prompt treatment, including basic neuroresuscitation principles (the ABCs), antiepileptic drugs to stop the seizure and identification of etiology. It results from an inability to normally abort an isolated seizure either due to ineffective inhibition, or due to abnormally persistent excessive excitation. Symptomatic SE is more common in younger children and the likely etiology depends on the age of the child. Treating the precipitating cause may prevent ongoing neurologic injury and facilitates seizure control. Benzodiapenes, phenytoin and phenobarbital form the mainstay of treatment. A systematic treatment regimen, planned in advance, is needed, including one for refractory status epilepticus (RSE). Patient education and home management of seizures is important to reduce the morbidity and mortality associated with SE. (Behera et al , 2005).

Seizures in PICU have different clinical characteristics from those in adults. Recognizing the common seizure etiologies in PICU is likely to lead to a more prompt and effective treatment. Antiepileptic drug prophylaxis may be useful in post-craniotomy patients. A neurological consultation and EEG evaluation are of the utmost importance to help rule in or out epileptic disorders in the PICU. (Valencia et al, 2006).

Infantile SE occurred more frequently in children with pre-existing epilepsy or neurological disorder. Acute febrile illness and infection were the most common precipitating causes. Early recognition and treatment of fever and infection in conjunction with prompt and appropriate termination of seizure in epileptic children may prevent the occurrence of SE and its morbidity (**Visudtibhan et al , 2002**).

Outcome in convulsive status epilepticus is influenced by cause, duration, age, the occurrence of medical complications and quality of treatment. Outcome in nonconvulsive status epilepticus is good and does not seem to be influenced by the treatment strategy. The use of a therapy protocol may prevent unnecessary delay and contribute to a better outcome. (Arzimanoglou, 2007).

Prolonged seizure lasting over 5-10 minutes are unlikely to stop spontaneously and should be treated as CSE .Prehospital administration of benzodiazepines is safe and simplifies subsequent management of CSE in the hospital setting .Treatment includes resuscitation measures ,identification and treatment of causal factors and early antiepileptic treatment following local guidelines that may be based on national guidelines (Chaure and Scott 2007).

Management of CSE includes identification and treatment of the underlying cause. In the absence of an obvious aetiology, blood glucose, blood gases and electrolytes (including sodium, calcium and magnesium) should be tested and any metabolic derangement should be corrected. However, the diagnostic assessment should not delay treatment and clinicians should be able to diagnose CSE on clinical grounds alone (**Prasad and Seshia, 2006**).

In general, an EEG, neuroimaging or other laboratory studies are not needed before the initiation of anticonvulsant therapy. If available, indications for emergency EEG (**Prasad and Seshia**, 2006).