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Cerebrovascular and posttraumatic epilepsy

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Epilepsy is one of the most common neurological disorders with individual, familial, and social-economic impacts. There are 50 million people with epilepsy in the world: of these over 80% are thought to be in developing countries.[1] As the understanding of the physical and social burden of epilepsies increased, it has moved higher up the world health agenda. Any type of brain disease can cause epilepsy, but not all people with a particular disease will have epilepsy, pointing to the role of host vulnerability in generating seizures.[2] This is a brief review of cerebrovascular and posttraumatic epilepsy, two important preventable causes of epilepsy.

CEREBROVASCULAR DISEASE

Epilepsy with cerebrovascular diseases

Epilepsy with cerebrovascular diseases (ECD) is defined as onset of epilepsy after cerebrovascular accidents or stroke, and epileptic seizures caused by cerebrovascular pathological changes. ECDs are classified as early onset (< 2 weeks) and late onset (> 2 weeks) after stroke. Stroke is one of the most common causes of epilepsy in adult and elderly patients. Five thousand and ninety two cases of ECD were reported from the People's Republic of China during 1994 – 2003 giving an average incidence rate of 8.7% (3.6 – 20 %) for ECD (Unpublished observation and analysis based on http://www.cnki.net/index.htm).

Risk factors for epilepsy with cerebrovascular diseases

These include location of stroke foci, cortical versus subcortical location; carotid artery territory versus vertebrobasilar territory, and cerebral lobes are at higher risk of ECD. The size of stroke is also important, 21.5% of large lesions versus 5.2% of small lesions were associated with ECD.[3] Hemorrhagic strokes and cerebral embolism are more commonly associated with developing seizures than thrombosis.[4,5]

Seizure types of epilepsy with cerebrovascular diseases

Most of the seizures after stroke are partial. They represent 42 to 89% of seizures after stroke.[3-7] Among partial seizures, simple partial seizures are the most common. The second most common seizure type reported is the generalized tonic clonic seizures. Complex partial seizures are rarely reported in most series.[3,4,6,8,9]

Choice of anticonvulsant

In most series, seizures after stroke are usually easy to control, 88% of patients are on monotherapy, and only 12% required multiple antiepileptic drugs to control the seizures.[6]

Pathology and pathophysiology of epilepsy with cerebrovascular diseases

The pathophysiology of cerebrovascular diseases include reduction in blood flow, brain edema, subsequent cytotoxic changes; systemic metabolic changes such as acidosis, electrolyte imbalance, and altered neurotransmitter activities. The mechanism of epileptogenesis of ECD is unclear. Potential mechanisms include hypoxia, ischemia, hemorrhage with iron deposition, scar formation, excess release of excitatory amino acids as glutamate and aspartate.[4,5,10,11]

POSTTRAUMATIC EPILEPSY

Traumatic brain injury is a major cause of morbidity and mortality worldwide. Posttraumatic epilepsy (PTE) remains one of the most common complications of traumatic brain injury. PTE accounts for 5% of total epilepsies and 20% of symptomatic epilepsies (Wang JQ, Unpublished observation).

Within the first year after head trauma, the incidence of seizures exceeds 12 times than in the general population. The risk of developing PTE remains as much as 25 times higher than the normal age-matched population over the next 10 years.[12] The reported incidence of PTE varies from under 5% for head injury in general to 25 to 30 % for severe closed head injury with hematoma, and up to 51% in survivors of military penetrating head injury.[13] There were 1,266 cases of PTE reported from the People's Republic of China during 1994 – 2003 giving an average incidence rate of 8 % for PTE among patients with head

traumas (Unpublished observation and analysis based on http://www.cnki.net/index.htm).

Risk factors for posttraumatic epilepsy

The risk factors for posttraumatic epilepsy include open injury, frontoparietal and temporal lobes in location, severe injury, cortical contusion and laceration, and large total brain volume loss. Presence of intracranial hematoma, metallic fragments, depressed skull fracture; prolonged posttraumatic amnesia and children are other risk factors.[14]

Histopathological studies

Material obtained from traumatized brains show formation of axonal retraction balls, reactive gliosis, Wallerian degeneration, and microglial star formation within cystic white matter lesions. Delayed effects of acute head trauma include focal or diffuse brain edema, ischemia, necrosis, gliosis, and neuronal loss.

Prophylactic anticonvulsants

Anticonvulsants are indicated in a patient with PTE. Phenytoin, carbamazepine, or phenobarbital may be used in the first 1 to 2 weeks post injury and showed a significant protective effect to PTE.[15]

Indications for surgical treatment

The considerations for surgical treatment of PTE in our practice include an epileptogenic foci verified by CT, MRI which is concurrent with EEG findings; PTE caused by skull bone defect, ineffective treatment of antiepilepstic drugs of more than 3 years duration, epileptogenic foci in nonfunctional area, and age greater than 16 years.[16]

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