Prescribing Pattern of Stress Ulcer Prophylaxis in Acute Stroke by Neuro-physicians and General Physicians in India

Sir,

Stress ulcers are commonly encountered in critically ill patients.1 The prevalence depends on the type of practice and nature of patients e.g. head injury, post craniotomy, stroke, aneurysmal bleed. Since there is no effective management for stress ulcer; prophylaxis is commonly used for prevention of stress ulcer. In neurology literature there is paucity of studies on the management of stress ulcer. In India stroke seems to be an important cause of stress ulcer and 30% patients with ICH has been reported to have gastric hemorrhage.2 There is no information on the practice of stress ulcer prophylaxis in stroke patients. This type of study is important to know about the prevailing pattern of stress ulcer prophylaxis in our country. Such knowledge may also help in modifying or improving the clinical practice. A good neurology practice has its roots in sound general medicine knowledge. In our country generally a formal training in general medicine is necessary before starting Neurology training. Even those who have direct Neurology training have 2 years rotations in general medicine. It may however be interesting to learn bout the practice pattern between general physician and neurologists. In this communication we report the prescribing pattern of stress ulcer prophylaxis in patient with stroke by physicians and neurophysicians in India.

This study is a random sampler survey. A questionnaire (Table 1) containing 10 multiple-choice questions were sent to randomly selected 100 NP and 100 GP interested in taking care of stroke patients. The NP was randomly selected from the directory of Neurological Society of India and Indian Academy of Neurology whereas GP were selected from the directory of Association of Physician of India. These specialists were working in medical institutes, medical colleges or specialized medical centers representing the tertiary and secondary level of medical care. Family physicians, general practitioners and primary healthcare doctors were not included. Responses were collected either through postal or when responders were attending a conference. The responses to questionnaire were tabulated and answers given by NP and GP were analyzed and compared employing chi-square test.

Out of 200 physicians only 59 NP and 41GP responded. Forty six percent opined that stress ulcer in stroke patient was common whereas 54% thought it to be rare. It was thought to be common in ICH by 45% responders. Sixty two percent thought that it commonly occurred in the first week and only two thought that it could occur even after 1 week. Majority (65%) felt that it significantly affected the prognosis of the patients. Fifty-one percent thought that stress ulcers resulted in bleeding of moderate severity although a significant number felt that it could be of varying severity (23%).

The importance of contributing factors or co-morbidity was reported by all. Use of drugs e.g. NSAID, antiplatelets, anticoagulant were considered to be most important single risk factor by 42%. However, majority (54%) opined that number of risk factors in combination are most important which included systemic illness, septicemia and raised intracranial pressure. It was interesting to note that none felt that septicemia independently could contribute to increase incidence of stress ulcer.

Majority of responders reported using stress ulcer prophylaxis (84%); 71% of the users employed prophylaxis for a duration of 2-4 weeks, 18% for 4-6 weeks and only 3% used it for more than 6 weeks and one attributed the long use to aspirin or NSAID. Of the drugs used; H2 blockers (16%), sucralfate (17%) and PPI (14%) were most prominent. However antacids were used by only 7% and all were GP. Combination of drugs was used by 30% of responders. The most frequently used combination was of sucralfate and H2 blocker (11%). 63% felt that prophylaxis was useful but 37% confessed using it in spite of being convinced of its inefficacy. When questioned about the problems of stress ulcer prophylaxis, 29% did not think it produced any significant side effects; however diarrhoea or constipation (28%), drug interaction (28%), anorexia (9%) and pneumonia (6%) were regarded as important problems related to stress ulcer prophylaxis.

Comparing the practice pattern by GP and NP the significant difference was noted only in the choice of agent for prophylaxis. 17% of GP used antacids as first choice of drug where as none of the NP used it. PPI was preferred by 18.6% of NP in comparison to 7.3% of GP. Majority of NP were using combination of drugs (39%) where as only 17% of GP were using the combination. The other parameters were not significantly different between the two groups (Table 2).

Majority of responders felt that stress ulcer bleed in ICH patients though rare, but an important complication as it is related to mortality. It commonly occurs in first week. The published literatures are also equally divergent. The 2 studies on stroke related stress ulcer have reported it as a rare complication 0.1 and 3.0%. This may be related to the spectrum of stroke patients. However both these studies have included cases of stroke rather than ICH or infarction only. Two studies
much as on the size of lesion. factors of which drugs were considered most important (42%). The importance of aspirin or NSAIDs in stress ulcer bleeding in stroke patients was highlighted earlier. The importance of underlying septicemia, resulting in abnormal coagulation may also contribute to stress ulcer. Recently in a multivariate analysis of risk factors for stress ulcer bleeding in ICH patients, size of hematoma, GCS and septicemia were found to be most significant. In a study on 51 patients, 20 had evidence of septicemia and nine had stress ulcer. It was surprising that none of the responders gave importance to systemic infections as a risk factor for stress ulcer. It may be due to lack of awareness about this modifiable risk factor. 84% used stress ulcer prophylaxis and H2 blockers, sucralfate and PPI were most commonly used. It was surprising to learn that only seven GP used antacids. The lower popularity of antacids in stress ulcer prophylaxis has been reported in earlier studies also. In a study on gastroprotection in neurosurgical patients the use of antacids was found to be declining from 36% in 1988 to 9% in 1994.8 In another study in critical care patients antacids did not figure in the choice option of physicians.9 Our responders favored the use of sucralfate (16%), H2 receptor blocker (17%) and PPI (14%). Majority of our responders used the combination (30%). This practicing pattern is different with that of Carol et al in which H2 antagonist (7%) and sucralfate (16.6%) were followed by antacid (Carroll et al 1994) however in the study of Lam et al H2 blocker (67%) followed by sucralfate (24%). For those responders who switched to another agent when H2 antagonist failed, 52% opted for omeprazole, whereas 67% opted for an H2 antagonist when sucralfate failed. It seems that introduction of a relatively new drug is favored over more established/older drugs. One reason for preference of PPI may be parenteral route of administration, probably more potent inhibition of gastric acid secretion and infrequent dosing over H2 blockers. Sucralfate and antacids have to be orally administered which may not be possible in a critically ill deeply comatose patient.

The important concern was 37% responders were using stress ulcer prophylaxis without being convinced of its efficacy. This highlights the importance of clinical practices, which tends to get influenced by trends and contemporary prescribing habits of colleagues and peers. This also underlines the need to collect or conduct scientific evidence in favor or against this practice and bring it out more clearly for practicing physicians.

Table 1: The questionnaire used for evaluating the prescribing pattern of general physician and neurophysician regarding the prophylaxis of stress ulcer in patients with stroke

Please tick (I) all the appropriate responses. You can choose more than one.

1. In your experience how common is gastric haemorrhage (GH) in stroke
   (a) Very rare <5%
   (b) Rare 5-10%
   (c) Common 10-20%
   (d) Very common >20%

2. Is it common in
   (a) Infarction
   (b) Haemorrhage
   (c) Equally common

3. When it usually occurs
   (a) < 1 week
   (b) 1-2 week
   (c) > 2 weeks

4. Does GH have significant effect on mortality
   (a) Yes
   (b) No

5. What is its severity
   (a) Mild < 10 ml
   (b) Moderate > 10 ml
   (c) Severe
   (d) All

6. Have you noted contributing risk factors
   (a) Drugs e.g. NSAID, antiplatelets, anticoagulant
   (b) Systemic illness e.g. liver disease, peptic ulcer
   (c) Septicemia
   (d) Raised intracranial pressure

7. Do you use prophylactic agents for gastric haemorrhage
   (a) Yes, then how long, (1) 2-4 weeks, (2) 4- weeks, (3) > 6 weeks
   (b) No

8. Which of the following agent
   (a) Antacids
   (b) H2 blockers
   (c) PPI, e.g. omeprazole
   (d) Sucralfate
   (e) If combination then specify what

9. The prophylactic therapy
   (a) Is it helpful
   (b) Used without any definite benefit

10. What are the problems in giving prophylaxis
    (a) Anorexia
    (b) Diarrhoea/constipation
    (c) Pneumonia
    (d) Drug interaction
    (e) None

Name: Dr. Designation Sign:

on ICH patients have revealed higher incidence of stress ulcer 30% and 50%.2-3 It may be possible that stress ulcer may not so much depend on infarct or hemorrhage as much as on the size of lesion.

Stress ulcer though are related to parasympathetic (vagal) stimulation, raised catecholamine levels, excessive gastric acid secretion and mucosal ischemia triggering a series of activities leading to release of cytokines and oxidative stress.6,7 However the role of drugs and co-morbidity cannot be underestimated. All the responders agreed with the importance of contributing factors of which drugs were considered most important (42%). The importance of aspirin or NSAIDs in stress ulcer bleeding in stroke patients was highlighted earlier.3 The importance of underlying septicemia, resulting in abnormal coagulation may also contribute to stress ulcer. Recently in a multivariate analysis of risk factors for stress ulcer bleeding in ICH patients, size of hematoma, GCS and septicemia were found to be most significant. In a study on 51 patients, 20 had evidence of septicemia and nine had stress ulcer. It was surprising that none of the responders gave importance to systemic infections as a risk factor for stress ulcer. It may be due to lack of awareness about this modifiable risk factor. 84% used stress ulcer prophylaxis and H2 blockers, sucralfate and PPI were most commonly used. It was surprising to learn that only seven GP used antacids. The lower popularity of antacids in stress ulcer prophylaxis has been reported in earlier studies also. In a study on gastroprotection in neurosurgical patients the use of antacids was found to be declining from 36% in 1988 to 9% in 1994. In another study in critical care patients antacids did not figure in the choice option of physicians. Our responders favored the use of sucralfate (16%), H2 receptor blocker (17%) and PPI (14%). Majority of our responders used the combination (30%). This practicing pattern is different with that of Carol et al in which H2 antagonist (7%) and sucralfate (16.6%) were followed by antacid (Carroll et al 1994) however in the study of Lam et al H2 blocker (67%) followed by sucralfate (24%). For those responders who switched to another agent when H2 antagonist failed, 52% opted for omeprazole, whereas 67% opted for an H2 antagonist when sucralfate failed. It seems that introduction of a relatively new drug is favored over more established/older drugs. One reason for preference of PPI may be parenteral route of administration, probably more potent inhibition of gastric acid secretion and infrequent dosing over H2 blockers. Sucralfate and antacids have to be orally administered which may not be possible in a critically ill deeply comatose patient.

The important concern was 37% responders were using stress ulcer prophylaxis without being convinced of its efficacy. This highlights the importance of clinical practices, which tends to get influenced by trends and contemporary prescribing habits of colleagues and peers. This also underlines the need to collect or conduct scientific evidence in favor or against this practice and bring it out more clearly for practicing physicians.

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Received : 20.5.2004; Revised : 30.6.2004; Accepted : 29.12.2004

REFERENCES

Table 2: Response to the questionnaire by General physician and Neurophysician regarding the prophylaxis of stress ulcer

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<th></th>
<th>NP (59)</th>
<th>GP (41)</th>
<th>X²</th>
<th>P</th>
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<tr>
<td>1. In your experience how common is GH in stroke?</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Very rare &lt;5%</td>
<td>12</td>
<td>10</td>
<td>.9</td>
<td>.82</td>
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<tr>
<td>Rare 5-10%</td>
<td>18</td>
<td>14</td>
<td>4.56</td>
<td>1.02</td>
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<tr>
<td>Common 10-20%</td>
<td>27</td>
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<td></td>
<td></td>
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<tr>
<td>Very common &gt;20%</td>
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<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Is it common in Infarction?</td>
<td>4</td>
<td>8</td>
<td></td>
<td></td>
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<tr>
<td>Haemorrhage</td>
<td>26</td>
<td>19</td>
<td></td>
<td></td>
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<tr>
<td>Equally common</td>
<td>29</td>
<td>14</td>
<td></td>
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<td>3. When it usually occurs</td>
<td></td>
<td></td>
<td>1.03</td>
<td>.59</td>
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<tr>
<td>&lt;1 week</td>
<td>39</td>
<td>23</td>
<td></td>
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<tr>
<td>1-2 week</td>
<td>19</td>
<td>17</td>
<td></td>
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<tr>
<td>&gt;2 weeks</td>
<td>1</td>
<td>1</td>
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<tr>
<td>4. Does GH has significant effect on mortality?</td>
<td></td>
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<td>1.80</td>
<td>.17</td>
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<td>Yes</td>
<td>42</td>
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<tr>
<td>No</td>
<td>17</td>
<td>18</td>
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<td>5. What is its severity?</td>
<td></td>
<td></td>
<td>2.9</td>
<td>.40</td>
</tr>
<tr>
<td>Mild&lt;10 ml</td>
<td>8</td>
<td>9</td>
<td></td>
<td></td>
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<tr>
<td>Moderate&gt;10ml</td>
<td>31</td>
<td>20</td>
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<tr>
<td>Severe</td>
<td>4</td>
<td>5</td>
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<tr>
<td>All</td>
<td>16</td>
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<td>6. Have you noted contributing risk factors?</td>
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<td>0.01</td>
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<tr>
<td>Drugs</td>
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<td>Systemic illness</td>
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<td>Septicemia</td>
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<td></td>
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<tr>
<td>Raised ICP</td>
<td>0</td>
<td>0</td>
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<tr>
<td>Combination</td>
<td>35</td>
<td>19</td>
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<tr>
<td>7. Do you use prophylactic agents for GH?</td>
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<td></td>
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<tr>
<td>Yes</td>
<td>50</td>
<td>34</td>
<td>18.02</td>
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<tr>
<td>2-4 weeks</td>
<td>37</td>
<td>26</td>
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<td>4-6 weeks</td>
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<tr>
<td>&gt;6 weeks</td>
<td>3</td>
<td>0</td>
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<td></td>
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<tr>
<td>NO</td>
<td>9</td>
<td>7</td>
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<tr>
<td>8. Which of the following agents you choose?</td>
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<tr>
<td>antacids</td>
<td>0</td>
<td>7</td>
<td>.019</td>
<td>.88</td>
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<tr>
<td>H2 antagonists</td>
<td>7</td>
<td>9</td>
<td></td>
<td></td>
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<tr>
<td>PPI</td>
<td>9</td>
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<tr>
<td>Sucralfate</td>
<td>11</td>
<td>3</td>
<td></td>
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</tr>
<tr>
<td>Combination</td>
<td>23</td>
<td>7</td>
<td></td>
<td></td>
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<tr>
<td>9. The prophylactic therapy?</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Is it helpful?</td>
<td>38</td>
<td>25</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Used without any definite benefit</td>
<td>21</td>
<td>16</td>
<td></td>
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<td>10. What are the problems in giving prophylaxis?</td>
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<td>2.48</td>
<td>.64</td>
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<tr>
<td>Anorexia</td>
<td>6</td>
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<tr>
<td>Diarrhoea/constipation</td>
<td>15</td>
<td>13</td>
<td></td>
<td></td>
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<tr>
<td>Pneumonia</td>
<td>5</td>
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<td>Drug interaction</td>
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<tr>
<td>none</td>
<td>18</td>
<td>11</td>
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</table>

GH: Gastric haemorrhage.

9. Niemman-Pick disease is a rare familial autosomal recessive genetic defect in which phospholipids are deposited in reticulo-endothelial cells and nervous system due to two distinct metabolic derangements. It is three times more common in Jews than in other races and five times more common in girls than boys.
10. In type A and B of Niemman-Pick disease, there is a deficiency of sphingomyelinase, an enzyme that hydrolyzes sphingomyelin to yield ceramide and phosphorylcholine, therefore, this excessive sphingomyelin accumulate in many organs and tissues. In addition type C of Niemman-Pick disease is also associated with massive lysosomal accumulation of unesterified cholesterol due to an incomplete intracellular utilization of cholesterol and thus accumulation of sphingomyelin is modest and the main defect is in storage of unesterified cholesterol.
11. Most common type, Niemman-Pick type A, begins shortly after birth with hepatosplenomegaly, failure to thrive, neurological impairment and fundus shows cherry red spots but seizures and hypersplenism are rare. Type B of Niemman-Pick disease is a relatively benign disorder with hepatosplenomegaly and sometime with pulmonary infiltrates but without any neurological involvement. A rare variant of Niemman-Pick disease type C characterized by organomegaly and gradual progression neurological deterioration. Majority of them become symptomatic in childhood, such as, seizure (in one third of patients); extrapyramydial dysfunction (dystonia, tremor and choreoathetosis) and pyramidal tract dysfunction (hyper-reflexia and spasticity). Progressive cognitive impairment, dysarthria and dysphasia eventually develop. The clinical triad of vertical gaze paresis, ataxia and foam cells in bone marrow virtually establishes the clinical diagnosis and confirmatory test, is the determination of the rate of cholesterol esterfication in cultured skin fibroblast or lymphocytes. Choking, aspiration and recurrent pulmonary infections are generally the terminal events in teens or early twenties.
A 23 years old Muslim girl, bedridden, presented with progressive ataxia, mental impairment for the last four years; dysarthria and abnormal movements of limbs for the last six months. Intermittently, she used to have vague abdominal pain without any localization. Bowel and bladder habits were normal. She had seizure at 11 years of age and since then on regular multiple anticonvulsant drugs. She had been operated for gallstones six years back. On examination: moderately build with precocious physique, moderately anemic with gingival hypertrophy and macroglossia without any lymphadenopathy; abdominal examination revealed hepatosplenomegaly; on neurological examination, she was conscious, irritable with impaired cognitive functions, bilateral vertical gaze palsies, dysarthric with generalized spasticity and gait ataxia. Fundus examination was normal. Other systemic examination was within normal limits. Ultrasonography of abdomen showed hepatosplenomegaly and lipidogram showed hypercholesterolemia (S. Cholesterol 306 mgm%). Neither facilities for bone marrow aspiration were available at our center nor patient had agreed for higher institution.

The clinical features of precocious physique and organomegaly; progressive neurological involvement in the form of seizure, extrapyramidal and pyramidal features (vertical gaze paresis and ataxia) and mental impairment; cholelithiasis in early teens and vague abdominal visceral pain, all are suggestive of a rare variant of Niemmm-Pick disease Type C. Since, there is no specific therapy available, seizures can be controlled with anticonvulsants and finally death usually occurs in the teens or early twenties from aspiration and frequent pulmonary infections.

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Received : 17.3.2004; Accepted : 28.12.2004

REFERENCES

Isolated Trigeminal Sensory Neuropathy Complicating End Stage Renal Disease

Sir,

Isolated facial numbness usually indicates involvement of the trigeminal sensory system which may be primary, unassociated with other neurological disorders or secondary to a host of conditions. End stage renal disease (ESRD) can cause peripheral neuropathy but is not known to give rise to cranial mononeuropathy. A case of trigeminal sensory neuropathy involving maxillary and mandibular divisions on both sides which predated the appearance of peripheral neuropathy in an elderly male suffering from azotemic obstructive uropathy is presented.

A 72 years old male suffering from urinary obstruction due to prostatic hypertrophy was referred to Medical out patient department for raised blood urea and creatinine. Patient had no uremic symptoms and was on terazosin and finasteride therapy. He gave a history of numbness and hypoaesthesia, involving the lower face on both sides including the oral cavity, tongue, hard palate, started 4 weeks back without any progression or fluctuation. There was no such symptom in the upper part of face or scalp; neither the patient had any difficulty in chewing, eating, closing eyes or opening mouth. His past, personal and family history was non-contributory.

On examination, patient had loss of pain, crude touch and temperature sensation over the distribution of maxillary and mandibular divisions of trigeminal nerve on both sides, sparing an area over the angle of jaw. Corneal reflexes and taste sensation were preserved. Jaw jerk was not elicitable. Examinations of rest of the nervous system including other cranial nerves and all other systems were unrevealing.

Routine investigations revealed normal hematolody (except Hb 8.9 gm %), raised urea (162 mg %) and creatinine (8.3 mg %), normal liver biochemistry, serum electrolytes and chest radiography. Electrocardiography and echocardiography showed left ventricular hypertrophy. Blink reflex study was normal. Trigeminal sensory evoked responses showed mild prolongation of latency. Nerve conduction study and electromyogram of all four limbs were normal. Sural nerve biopsy carried out as a part of diagnostic work up and magnetic resonance imaging (MRI) of the brain, orbit and upper cervical spine was normal. CSF study for cell type, cell count, total protein, albumin, glucose, chloride was within normal range. Serum ACE level was not done. A diagnosis of trigeminal pure sensory neuropathy (involving maxillary and mandibular divisions) complicating ESRD was made.

Facial numbness occurring alone usually indicates involvement of the trigeminal sensory system: either one or more of the three divisions of the trigeminal nerves, the trigeminal (Gasserian) ganglion, the sensory root, or the spinal trigeminal or main sensory nuclei. Objectively there are deficits of one or more of the usual methods of facial sensory testing: pinprick, temperature or light touch. If all three are involved the trigeminus is affected outside the brain stem, whereas dissociation between pinprick and temperature and light touch sensation indicates that the lesion lies within the brain stem.3 Trigeminal involvement may be primary, or secondary
to tumours, multiple sclerosis, vertebro basilar vascular disease, collagen vascular disease, dental or facial trauma, toxic chemical and drugs, and so forth. Studies of blink reflexes are abnormal (if the ophthalmic division is involved), majority showing an afferent defect with modest prolongation of the latency. Trigeminal sensory evoked responses show mild prolongation of latencies.

Neuromuscular manifestations in ESRD may present as alteration of higher mental functions, movement disorders, neuromuscular irritability and peripheral neuropathy. Cranial neuropathies in uremia are not described in the literature. However there are reports of facial paraesthesia resembling restless leg syndrome in patients on hemodialysis.

Our patient had facial numbness complicating ESRD. Even after thorough investigations, no cause could be established for the isolated trigeminal neuropathy. The neuropathy did not subside in due course; rather it was persistent even after institution of dialysis which we started after one month. The patient died from uremic encephalopathy nine months after the initial presentation and by that time had developed distal sensory neuropathy characteristic of ESRD.

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REFERENCES

Post-Malarial Neuropsychiatric Syndrome

Sir,

Most of the complications attended with malaria are attributed to infection with plasmodium falciparum species. Cases with falciparum malaria have registered a significant rise in recent times. As a result, more and more uncommon systemic complications, hitherto unreported are being highlighted. Post-malarial neurological syndrome is diagnosed in patients with recent symptomatic infection by P. falciparum after parasites have been cleared from blood. In cases with cerebral malaria, the syndrome is diagnosed when patients regain full consciousness but manifest neuropsychiatric symptoms within two months of onset of acute illness.

The case in point is a 50 year old male from endemic zone, admitted in the ward in deep coma that followed high grade fever of 3 days duration. History of seizures or previous psychiatric illness was denied. Drug history was unremarkable as was family history for any mental illness. Icterus was absent and urine output was normal. Pallor was present and spleen was mildly enlarged. Peripheral blood smear was teeming with trophozoites and gametocytes of P. falciparum. QBC score was more than 300 pf/QBC field. Renal and liver function tests were normal. The patient was treated with injection quinine and oral doxycycline, in required doses. He recovered consciousness within 3 days and was afebrile and showed normal cognitive function. However after a week, he started showing behavioural changes, hallucinations and persecutory delusions. The peripheral blood smear this time was normal. Patient showed good clinical recovery of mental function after administration of antipsychotic drug (Risperidone).

Post-malarial neuropsychiatric syndrome is an entity itself and should be suspected in situation where patients regain consciousness but later show unusual behavioural symptoms. Malarial psychosis manifesting as paranoid and maniac syndromes in the acute stage whereas depression is a late sequela. In clinical practice it is taken to be a part of acute encephalitic syndrome associated with malaria and left untreated. Brief reactive psychosis some times develop as patient recovers. The condition is treatable and fully reversible.

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REFERENCES