Correspondence

Adulteration of Drugs of Alternative Systems of Medicine with Corticosteroids ........................................ 634

Importance of Ankle Jerk in Acute Flaccid Paralysis ...................................................................................... 634

Selective Serotonin Reuptake Inhibitors - A Few Pitfalls ................................................................................. 635

Falciparum Malaria Precipitating Hyperkalaemic Periodic Paralysis .............................................................. 636

Ethylene dibromide Needs to be Banned as Food Fumigant .............................................................................. 637
Adulteration of Drugs of Alternative Systems of Medicine with Corticosteroids

Sir,

The popularity of complimentary and alternative medicine (CAM) has increased in recent times. Several surveys have shown that less than 30% of Indians receive allopathic drugs. Majority (70%) rely on Ayurvedic and herbal drugs.1 The potential benefits of traditional medicine are well known, however, harm from adulteration of these medications with allopathic drugs e.g. corticosteroids must be recognized. There have been previous reports of adulteration of herbal medicines with corticosteroids.2,3

We screened 281 samples of alternative medicine for presence of corticosteroids. These samples were submitted to our laboratory by the treating physician or patients themselves because they suspected adulteration of these drugs either due to fast relief or some adverse effect such as weight gain and in some cases it was pure curiosity. The information regarding the source (registered/unregistered practitioner), dose and dosage schedule of these medications was not available to us. Majority of the samples were received from patients of unknown/unreported ailments (156), arthritis (83) and asthma (37).

The drug samples were screened chromatographically using high performance TLC plates (Merck). Out of 281 samples, 166 (59%) were ayurvedic, 28 (9.96%) were homeopathic, seven (2.5%) were Unani and 80 (28.4%) were of unknown category. One hundred and forty one (50.1%) samples out of 281 tested positive for corticosteroids. The system-wise break up is given in Table 1.

<table>
<thead>
<tr>
<th>Type of drug</th>
<th>No of samples n (%)</th>
<th>Samples +ve for steroids n (%)</th>
<th>Samples -ve for steroids n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ayurvedic</td>
<td>166 (59%)</td>
<td>92 (55.4%)</td>
<td>74 (44.5%)</td>
</tr>
<tr>
<td>Homeopathic</td>
<td>28 (9.9%)</td>
<td>1 (3.5%)</td>
<td>27 (96.4%)</td>
</tr>
<tr>
<td>Unani</td>
<td>7 (2.5%)</td>
<td>0 (0%)</td>
<td>7 (100%)</td>
</tr>
<tr>
<td>Unknown</td>
<td>80 (28.4%)</td>
<td>48 (60%)</td>
<td>32 (40%)</td>
</tr>
<tr>
<td>Total</td>
<td>281</td>
<td>141 (50.1%)</td>
<td>140 (49.9%)</td>
</tr>
</tbody>
</table>

Thirty two out of 40 ayurvedic samples, 20 out of 39 samples of unknown category and none of the four homeopathic samples from patients of arthritis tested positive for corticosteroids. Similarly, of the samples received from the patients of bronchial asthma, 17 out of 27 ayurvedic samples, one out of two homeopathic and two out of eight samples of unknown category tested positive for corticosteroids.

One patient was receiving ayurvedic preparation for increasing height, another one for slimming and two patients in ICCU were also receiving ayurvedic preparations. All these four ayurvedic preparations tested positive for steroids. Out of 156 samples being taken by the patients for unknown/unreported ailments, 39 out of 94 ayurvedic, 26 of 33 samples of unknown category tested positive for corticosteroids while none of the 22 homeopathic and seven unani samples were found to be adulterated in this category.

Thus, more than 50% samples of ayurvedic medicines (55.4%) and of unknown category (60%) were found to be adulterated with corticosteroids. The adulteration was minimal in homeopathic medicines dispensed (3.5%) and none of the unani samples tested positive for corticosteroids.

Though the samples received by us were dispensed by so called practitioners of indigenous medicine, our data indicates the presence of externally added corticosteroids in a substantially high percentage of drugs that were screened. Such indiscriminate use of corticosteroids by some practitioners of indigenous medicine could lead not only to extensive health hazards to the patients but also bring the alternative systems of medicine into disrepute. Despite the fact that the adulteration of ayurvedic medicines is being reported since more than two decades, the regulatory authorities have not initiated any measures to prevent the same. It is high time that strict quality control and regulatory systems are established to curb such malpractices.

Ashima Bhatia*, Usha Gupta**, G Tayal***

*Senior Resident; **Professor and Head; ***Ex-Director, Professor and Head; Department of Pharmacology, Maulana Azad Medical College, New Delhi.

Received : 10.8.2002; Revised : 28.4.2003; Accepted : 16.5.2003

REFERENCES


Importance of Ankle Jerk in Acute Flaccid Paralysis

Sir, Guillain Barre syndrome (GBS) is the commonest cause of acute flaccid paralysis. It usually presents with quadriparesis, generalized areflexia, and rarely involves the bulbar and respiratory muscles. Patients with respiratory paralysis need
ventilatory support in addition to immunoglobulins (IVIG) or plasmapheresis. Nerve conduction studies (NCS) and CSF examination are important diagnostic tools but may not be confirmatory early in the disease. In such situations, diagnosis and management remains largely, clinical. Here we present our observation that the preservation of ankle reflex in patients with acute severe areflexic quadriaparesis with respiratory muscle involvement is a strong pointer against the diagnosis of GBS and does not require expensive and invasive treatment protocols.

**CASE 1**

A 22 year old female presented with history of pain in abdomen, vomiting and fever seven days back followed by rapidly developing quadriaparesis and respiratory paralysis for few hours. Examination revealed grade 0/5 power in all four limbs, no neck holding, generalized areflexia except preserved ankle reflexes, single breath count (SBC) of eight and bilateral facial muscle weakness. Patient was immediately put on ventilatory support. Investigation revealed presence of porphobilinogen in the urine. She recovered in three months period following therapy with repeated blood transfusion and glucose supplementation.

**CASE 2**

A 50 years male presented with acute onset quadriaparesis for one day. He had history of acute diarrhea about a week prior to quadriaparesis. Examination revealed grade 0/5 power in lower limbs and grade 2/5 power in upper limbs, generalized areflexia except preserved ankle reflexes and single breath count of 20. He had blood pressure of 200/130 mm Hg in the right upper limb. Investigations revealed a normal nerve conduction studies (NCS) and serum potassium of 2.2 meq/L. He recovered with oral potassium supplementation.

**CASE 3**

A 30 years male was referred from nearby hospital for ventilatory support for acute flaccid quadriaparesis with respiratory paralysis after bouts of acute diarrhea and vomiting for five days. Examination revealed grade 1/5 power in all the four limbs, generalized areflexia except preserved ankle reflexes, SBC 0 and drowsy sensorium. He was immediately put on ventilatory support. At admission serum potassium of 1.45 meq/L was noted. He recovered with oral potassium supplementation.

**DISCUSSION**

All three cases presented with acute flaccid areflexic quadriaparesis mimicking acute GBS. However, preserved ankle reflexes, suggested diagnoses otherwise (two had hypokalemic paralysis and one had acute intermittent porphyria).

Universal areflexia is an essential criterion to diagnose GBS and the distal reflexes are lost early. Cases are reported otherwise typical of GBS, in which reflexes have been preserved.\(^1\) But preservation of distal reflexes throughout the course is rare. Mechanism, which explains early reflex loss in GBS chiefly, depends on dispersion and desynchronisation of high frequency afferents to trigger monosynaptic arch due to demyelination. Why the ankle jerk is preserved in porphyria and hypokalamia with other reflex loss, is not clear. Possibly both these conditions predominantly affect proximal muscles.

There are well-accepted and -defined clinical and laboratory parameters which if present suggest the diagnosis of the GBS unlikely (Table 1). To this list of parameters, we add that the preservation of ankle reflexes should cast doubt on the diagnosis of GBS. Simple tests like serum potassium and urine porphobilinogen might be helpful and can prevent unnecessary costly therapy.

<table>
<thead>
<tr>
<th>Table 1 : Features casting doubt on diagnosis of GBS(^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Marked persistent asymmetry weakness</td>
</tr>
<tr>
<td>2. Persistent bladder bowel dysfunction</td>
</tr>
<tr>
<td>3. Bladder or bowel dysfunction at onset</td>
</tr>
<tr>
<td>4. More than 50 mononuclear cells in CSF</td>
</tr>
<tr>
<td>5. Polymorphonuclear leukocytes in CSF</td>
</tr>
<tr>
<td>6. Sharp sensory level</td>
</tr>
</tbody>
</table>

**D Goel, A Singhal**

Department of Neurology, Himalayan Institute of Medical Sciences, Swami Ram Nagar, Dehradun.

Received : 12.2.2003; Revised : 8.4.2003; Accepted : 15.4.2003

**REFERENCES**


**Selective Serotonin Reuptake Inhibitors - A Few Pitfalls**

Sir,

Serotonin syndrome is a potentially fatal iatrogenic complication of psychopharmacologic therapy characterized by the triad of altered mental status (confusion, disorientation, anxiety and coma), autonomic dysfunction (diaphoresis, tachycardia, hypertension, dilated pupils, hyperthermia) and neuromuscular abnormalities (myoclonus, tremor, seizures, hyperreflexia and rigidity). The increased use of selective serotonin reuptake inhibitor (SSRI) and serotonin agonists has made this clinical syndrome increasingly frequent.\(^1\) Minor from of this syndrome is often overlooked initially, we herein present two cases of SSRI related complication who were admitted in our hospital in last six months with acute confusional state.

The first case, a 48 years male, a University Professor, was brought to our emergency department with an acute confusional state. He was being treated with alprazolam for anxiety and insomnia for last two years. Due to inadequate response to therapy, a psychiatrist was consulted. He made a diagnosis of obsessive-compulsive disorder and initiated...
fluoxetine (20 mg/day). According to his family members, one hour after ingesting his first dose of fluoxetine, the patient developed rapidly progressive agitation, uncontrolled jitters and severe diaphoresis without bowel and urinary incontinence. Attending psychiatrist further recommended a single dose of clomipramine (25 mg) to calm him down. Soon thereafter, his restlessness and tremulousness increased and he was totally confused. He had an attack of generalized tonic-clonic seizure. On presentation in our emergency department after six hours, the patient was breathing spontaneously and had a pulse oximeter reading of 100%. The skin was markedly diaphoretic. Cardiovascular examination revealed abrupt fluctuations in heart rate (range 68-116/min) and blood pressure (range 110-170/70-105 mmHg) but was otherwise normal. Pupils were large with normal reaction to light. The patient was noted to have multifocal jerks suggestive of myoclonus. Deep tendon reflexes were exaggerated with bilateral downward plantar response. Other general and systemic examination was normal. Laboratory analysis revealed a normal complete blood count, metabolic parameters and serum creatine phosphokinase (CPK) level. Peripheral blood smear and antigen test for malaria were negative. MRI scan of brain (plain and contrast), EEG, CSF study and arterial blood gas analysis were normal. He was thought to have serotonin syndrome and managed conservatively in ITU with IV fluid, oxygen, beta-blocker and cyproheptadine. He had complete recovery in next 16 hours. A repeat EEG and CT scan of brain were normal.

The second case, a 60 years female, hypertensive, on diuretic (Thiazide) was admitted in a confused state which developed over 12 hours period. One week prior to admission, she was put on fluoxetine (20 mg/day). She undertook 12 hour fasting state for religious reason just one day prior to the present illness. On presentation during admission, she was agitated, confused, diaphoretic and trembling increasingly. Her temperature was normal and a fluctuating pulse and blood pressure were also noted. Other general and systemic examination did not reveal any abnormality. Her haemogram, CPK, metabolic parameters, parasitological test and CSF analysis were normal. Serum electrolytes revealed low sodium level (114 meq/L) and normal potassium (3.5 meq/L). Neuroimaging (CT scan and MRI brain) and EEG were normal. Diuretic and fluoxetine were stopped. Sodium deficit was gradually corrected. Eventually she improved completely after about 36 hours.

Serotonin syndrome was suspected in both the cases in view of SSRI uptake followed by development of confusional state, fluctuating cardiovascular responses, absence of any infection and normal imaging. However the differential diagnosis was broad and included most potential causes of delirium. In the first case, the final diagnosis of serotonin syndrome was based on the exclusion of other possibilities such as infection, metabolic disturbances and other toxic causes. With the growing trend of increased use of SSRI in general, serotonin syndrome needs to be considered in any patient presenting with any combination of altered cognition, autonomic dysfunction and neuromuscular abnormalities and though rare, this may occur as in the first case, following exposure to a single dose of SSRI.2 Closest differential diagnosis is neuroleptic malignant syndrome in which serum CPK is usually very high and commonly follows after antipsychotic medication. In the first case serotonin syndrome developed following a single dose of fluoxetine, which was further exacerbated, by a single dose of clomipramine.

Hyponatremia is an uncommon but widely reported complication of SSRI, especially in the elderly.3 The presentation is usually that of SIADH, but the underlying mechanism leading to the syndrome is poorly understood. This illustrates the need for periodic measurement of serum sodium in elderly patients soon after they start taking any SSRI, especially during the first 2-4 weeks of treatment, as it has been highlighted by the second case.

To conclude, the early recognition of serotonin syndrome will lead to timely discontinuation of SSRI and mortality and morbidity due to this iatrogenic complication may be avoided.

**REFERENCES**


**Falciparum Malaria Precipitating Hyperkalaemic Periodic Paralysis**

Sir,

Hyperkalaemic periodic paralysis (HyperKPP) is a rare autosomal dominant disorder characterized by acute and episodic muscular weakness.1 It is uncommon to find the weakness precipitated by malaria paroxysm. Here we report a patient of HyperKPP who had paralysis, precipitated by falciparum malaria due to its rarity.

A 39 years man presented with history of fever for two days and weakness of four limbs for one day. The fever was high grade with chill and rigor and subsided with sweating. On second day, after the paroxysm of fever the patient noticed weakness of four limbs without bladder and bowel involvement. There was no history of difficulty in deglutition, vomiting and diarrhea. He had not received any antimalarial. One year ago he had quadruparesis without sphincter affection which improved with treatment and was diagnosed
as a case of HyperKPP on the basis of raised serum potassium (6.7 mEq/L), myotonic pattern of EMG, and presence of vacuoles on muscle biopsy. Physical examination revealed average built with fair nutrition, temperature-100°F, pulse-110 per minute, BP - 100/70 mm of Hg, respiration rate-20 per minute. Hepatic span was 15 cm. Spleen was firm, non-tender and palpable 4 cm below costal margin. Patient was conscious, well oriented without any cranial nerve deficit. Power was grade III around all joints with normal tendon reflexes and flexor plantar. There was no muscular tenderness, myotonia, respiratory muscle involvement, and sensory deficit. Other systems were clinically normal.

Investigation showed Hb - 8 gm/dl, TLC - 8200/cumm, DLC:N - 78%; L - 12%; E - 10%, ESR - 10 mm first hour, random blood glucose - 90 mg/dl, blood urea - 20 mg/dl, s. creatinine - 0.2 mg/dl, s. bilirubin - 0.6 mg/dl, AST - 40 IU/L, ALT - 32 IU/L, s. sodium - 132 mEq/L, s. potassium - 6.2 mEq/L. Giemsa stained peripheral blood smear showed \( P. falciparum \) rings with the count of 3280/µL. ECG showed absence of p wave and tall T wave in chest leads (Fig. 1). ICT test was positive for \( P. falciparum \) but negative for \( P. vivax \) malaria.

Here, in a diagnosed patient of HyperKPP, as the episode occurred after chill and rigor, it was reasonable to think that the weakness had been precipitated by malarial paroxysm. He was treated with oral chloroquine. Signs of improvement was marked after eight hours. ECG returned to normal after 12 hours. He recovered completely after 24 hours and was discharged from the hospital on fifth day.

Periodic paralysis complicating falciparum malaria is a rare entity. Only three such cases had been reported from Sri Lanka, of which two had mixed infection (\( P. vivax \) and \( P. falciparum \)) and one had \( P. falcipaum \) infection. All the patients were treated with chloroquine successfully. Unlike hypokalaemic periodic paralysis, in patients of HyperKPP, paralysis is precipitated by potassium administration or stimuli that lead to hyperkalaemia. Exercise and haemolysis can induce transient hyperkalaemia. Also occurred during febrile episode of malaria, and has been ascribed to intense muscular contraction during rigor and lysis of red blood cells. Hence, hyperkalaemia induced by malaria precipitated paralysis in this patients.

**MK Mohapatra*, SP Das**

*Assistant Professor; **Professor; Department of Medicine, MKCG Medical College, Berhampur - 760 004, Orissa, India.

Received : 16.1.2002; Revised : 5.4.2003; Accepted : 15.4.2003

**REFERENCES**


**Ethylenedibromide Needs to be Banned as Food Fumigant**

Sir,

An article - Ethylenedibromide Poisoning by Garg *et al* (2002) appears to be timely review for Indian clinicians. The most common cause of poisoning in our experience during year end has been organo-phosphorus (45%) followed by sedative overdose (25%) and aluminium phosphide (20%). EDB accounted for 10% of cases of poisoning.

Proportion of females has been more than males and the age ranged from 23 to 52 years for both the groups. Mortality was 9/12 in year 2001 and 6/9 in 2002. The few cases who survived had either low ingestion dose or had used expired EDB. The death usually occurred after 2-3 weeks of ingestion and clinical course usually was hepato-renal involvement following respiratory/cardiac failure despite the intensive care support.
The main purpose of this communication is to highlight the suicidal use of EDB by virtually all cases in this series.

The drug is widely used as fumigant for grains in our state and this offers an easy access for the drug. As pointed out by Garg et al (2002), the use is banned after 1984 in USA for the fumigation of food since significant residues of it could be found in food products by US environmental agency. Looking at its usage as suicidal drug, it is our earnest appeal to ban its use as fumigant for food before many more die of it. Other industrial applications of EDB should be also under strict vigilance so as to restrict the access the reduce accidents.

WHO estimates that over 500000 people died from the use of pesticides for self harm in Southeast Asia in the year 2000 and a minimum pesticides list availability is the approach by

WHO.2 Physicians have already requested for the ban of aluminium phosphide in India3 and the same should be followed for EDB.

Ravikant*, S Geed**, DS Chitnis+
*Medical Director; **Medical Officer, ICU In-charge; +Head and Prof in Microbiology and Immunology; Choithram Hospital and Research Centre, Indore.
Received : 20.9.2002; Revised : 13.3.2003; Accepted : 19.3.2002

REFERENCES

Announcement

Indian Stroke Association

Members of Association of Physician of India and affiliated associations are eligible to enroll as member of "Indian Stroke Association" by quoting their membership no. with Biodata.

For further details, please contact : Dr. G Arjandas, President - Indian Stroke Association, 36, Pantheon Road, Egmore, Chennai 600 008.
E_mail : arjandas@satyam.net.in

Sd/-
Prof. PM Dalal
Vice president, Indian Stroke Association
Past President, API

31st Annual Scientific Meeting of the Research Society for the Study of Diabetes in India at Jaipur on October 10,11 and 12, 2003. RSSDI 2003 Secretariat.
Web site : www.rssdi2003.com
For further details contact : Dr. Anant Nigam, Organising Chairman, E_mail : dranigam@hotmail.com
Dr. Arvind Gupta, Organising Secretary, E_mail : arvindneelam@hotmail.com;
Tel. 0141-2708808; Fax : 2214796

Sd/-
Dr. Arvind Gupta