Gujjar Lung

Sir,

This recently introduced entity in the literature, is a chronic lung disease due to prolonged exposure to indoor air pollution with pine wood smoke occurring in Gujjar Community – a social and ethnic group of population residing at hilly regions of the Indian sub-continent (Jammu and Kashmir, Himachal Pradesh, Rajasthan, Pakistan, Pakistan-occupied-Kashmir and Gilgit). These people live in poorly ventilated mud houses called ‘Kothas’ and use pinewood as a fuel for cooking and heating purposes. A high oleoresin containing portion of the wood called ‘Lash’ is used as a source for lighting in the dwellings creating dense smoky atmosphere and adding further to the indoor air pollution. All family members residing in kothas are exposed to this smoky atmosphere for 12 to 16 hours daily, more during winter months.1,2

The disease is characterized clinically by progressive cough and dyspnea, usually appearing beyond fourth decade of life, varying radiological pattern of miliary mottling, reticulonodular shadows with or without features of chronic bronchitis or cor pulmonale - the picture mimicking pulmonary tuberculosis. On histopathological examination of lungs, anthracotic nodules with carbon-laden macrophages (mainly perivascular in distribution) and fibrogenic reactions are seen.2

The term ‘Gujjar Lung’ was first introduced in 1991 by Dhar and Pathania1 from Kashmir when they observed miliary and reticulonodular shadows in chest radiographs of Gujjar community who were empirically given anti-tuberculosis treatment keeping in view the high prevalence of pulmonary tuberculosis in this group of population; but the shadows remained unchanged or progressed despite adequate dosage of the drugs and duration of treatment. Pulmonary function testing revealed restrictive or obstructive pattern. Finally, lung biopsy performed in 36 of the 46 patients of their study group revealed evidence of anthracotic nodules, carbon-laden macrophages and fibrosis. Other radiodiagnostic modalities like high resolution computed tomography (HRCT) were not available. Subsequently in 2000, Raison and co workers2 observed similar radiological findings confirmed by HRCT and similar histopathological features in a Kashmiri baker working in Saudi Arabia who had significant history of exposure to pine wood smoke inhalation from early age, and, the radiograph was obtained by chance for immigration purposes. The occurrence of this lung disease was attributed to indoor air pollution with pine wood smoke,1,2 which is supported by earlier studies conducted on rabbits by Thorning and co-authors3 to see the effect of pine wood smoke on airways and the lung parenchyma. They observed predominantly large airway injury occurring beyond 24 hours of exposure to pine wood smoke in the form of disruption of surface epithelium, sloughing and necrosis with marked edema in subepithelial cells accompanied by increase in the number of extravasated erythrocytes and polymorphonuclear cells; whereas there are additional studies demonstrating injury of more distal airways and gas exchange units as well4. Pine wood on combustion gives rise to sulphurdioxide, benzopyrene, carbon monoxide, nitrogen oxides and low-molecular weight aldehydes including acrolein and albeitic acid. These gases individually or alongwith carbon are considered to be responsible for lung injury with eventual fibrogenic reaction.1,3

We are studying such cases for over eight years now, and to date have not observed any patient belonging to this entity and having history of exposure to smoke inhalation from other biomas fuels. The disease usually manifests beyond fourth decade with dyspnea and cough productive of blackish sputum. Males and females are involved almost equally and no case has been detected in the pediatric or teenage group so far. Further studies involving large samples of Gujjar population are going on and prevention of the occurrence of this disorder may need to change the living standard of these people in order to prevent exposure to pinewood smoke inhalation.

G Hassan*, GQ Khan**, W Qureshi+, RA Rashid++, T Masood***, Gazanfar Ali***

*Incharge Consultant, **Former Professor and Head, ***Senior Consultant, Department of Medicine, Government Medical College associated General Hospital, Sanat Nagar, Srinagar Kashmir; +Medical Superintendent, Government Medical College associated SMHS Hospital Srinagar, Kashmir; ++Consultant Pathologist, SKIMS Medical College, Srinagar, Kashmir.

Received : 13.1.2006; Accepted : 30.7.2007

REFERENCES


Health Professionals’ Characteristics Associated with Requisitioning Electroencephalography in Breath Holding Spells

Sir,

Breath-holding spells (BHS) are a common clinical problem affecting around 5% of children. These consist of a

670 www.japi.org © JAPI • VOL. 55 • SEPTEMBER 2007
distinctive stereotypical sequence of events beginning with a provocation to cry or tantrum and leading to noiseless expiration, color change, and loss of consciousness. The diagnosis is essentially based on the distinctive clinical sequence of events but may occasionally cause diagnostic confusion with seizure disorder when followed by brief seizure activity or terrors. We frequently see many BHS patients being treated as epilepsy, based on an abnormal electroencephalograph (EEG). Therefore, this study was conducted to study the health professionals' and patients' characteristics associated with requisitioning an EEG in BHS.

All patients (n=45) with BHS attending the pediatric OPD between 1 February and 31 October 2001 were enrolled and followed till 31 January 2003. Four children who did not return for follow-up were excluded from the final analysis. Diagnosis was based on a history of three or more episodes of typical spells as per standard definitions. The spells were classified according to skin colour change of the child during the episode into cyanotic, pallid, and mixed (when there was no clear distinction between cyanosis or pallor, or conflicting history was given by the parents). The history and medical records were reviewed to look for family history, type of spells, and presence of associated tonic-clonic movements, previous investigations and treatment.

The median age at presentation was 25 months (M:F=3:1:1) and the spells were cyanotic, pallid and mixed in 69%, 22% and 9% children, respectively. Twenty-eight children (68.5%) had previously consulted one (18, 64.3%) or more health professionals before coming to the hospital. A total of 41 consultations had been taken (10 pediatricians, 6 non-pediatric postgraduates, 14 medical graduates and 11 Registered Medical Practitioners). None had consulted Ayurvedic/Unani physicians, although three others had had interaction with traditional faith healers (not included in analysis). Twenty-two patients (54.7%) had undergone an EEG prior to attending the hospital. Five EEGs had been reported as abnormal, of which only one record was available for review and was normal. Five children (3 reportedly abnormal EEG; 2 reportedly normal EEG) were receiving AED (antiepileptic drugs) (4, phenobarbitone; 1, phenytoin). The various patient and health-professional characteristics associated with an EEG examination are tabulated in Table 1.

More than three-fourth (22/28, 78.6%) of the patients who had consulted a health professional before attending the hospital had been advised an EEG, specially in those with onset in infancy, with family history of seizure disorder, attending unqualified health professionals, having associated tonic-clonic movements, and mixed BHS. This shows that many medical practitioners, particularly those who are unqualified or those without pediatric qualifications apparently are not familiar with the syndrome and had requisitioned an EEG. This is often done either because of a misdiagnosis of epilepsy or the physician’s belief that these drugs prevent or reduce the frequency of spells. BHS, in fact, is a benign condition with an expected natural history.

The convulsive movements seen during BHS are reflex anoxic seizures, which are not epileptic and do not require antiepileptic medication. A few authors have reported patients with prolonged seizures or status epileptics following BHS. Emery reported two children with status epilepticus following BHS with a normal interictal EEG and, no effect of prophylactic anticonvulsants on the frequency of BHS or the associated convulsions. Moorjani et al reported having seizure activity following the spells in 55.5% patients in their series with prolonged seizures in five. Treatment with AED prevented further prolonged seizures. These reports underscore the non-epileptic nature of the seizures in this disorder. Probably these patients have a lower seizure threshold and hypoxia-ischemia triggers the seizures. The term ‘anoxic-epileptic seizures’ has been used for these events.

More than three decades back, Livingstone observed that many physicians and particularly those with a primarily adult practice were unfamiliar with BHS. The situation still seems to be similar, at least in India. This study suggests that there is a need to create awareness about BHS to primary physicians and training them to recognize and diagnose it correctly, so as to prevent misdiagnosis and ensure proper management.

<table>
<thead>
<tr>
<th>Table 1: Patient and health-professional characteristics associated with EEG examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Characteristics</td>
</tr>
<tr>
<td>---------------------------------</td>
</tr>
<tr>
<td>Patient Characteristics (n=28)</td>
</tr>
<tr>
<td>Onset&lt;1 year</td>
</tr>
<tr>
<td>Family h/o seizures</td>
</tr>
<tr>
<td>Family h/o BHS</td>
</tr>
<tr>
<td>Type of BHS</td>
</tr>
<tr>
<td>Cyanotic</td>
</tr>
<tr>
<td>Pallid</td>
</tr>
<tr>
<td>Mixed</td>
</tr>
<tr>
<td>Associated T.C. movement</td>
</tr>
<tr>
<td>Physician Characteristics (n=41)</td>
</tr>
<tr>
<td>RMPs</td>
</tr>
<tr>
<td>Qualified</td>
</tr>
<tr>
<td>Graduates</td>
</tr>
<tr>
<td>Physicians</td>
</tr>
<tr>
<td>Non-Pediatricists</td>
</tr>
</tbody>
</table>

*p<0.05, Pediatrician vs non-pediatrician

REFERENCES

3. Moorjani BI, Rothner AD, Katagal P. Breath-holding spells...


Cardiogenic Unilateral Pulmonary Edema

Sir,

A 35 years man presented with severe dyspnea for two days. Fine crackles were present at the left infrascapular area. ECG showed left ventricular hypertrophy and left axis deviation. X-ray chest showed unilateral infiltrates with 'bat-wing' appearance in left lung field (Fig. 1). Echocardiography revealed dilated and poorly contractile left ventricle. Treatment with diuretics resulted in rapid improvement of symptoms and disappearance of infiltrates within 12 hours.

Unilateral pulmonary edema (UPE) due to left heart failure is a distinctly uncommon condition. It mostly affects the right lung. The exact cause is not known but various hypotheses put forward include the effect of gravity and posture, variation in pulmonary venous pressure and disturbances of neurogenic control of capillaries and their permeability. Cardiogenic origin of the infiltrates is supported by their rapid clearing after diuretic therapy. The awareness of this unusual condition helps in early initiation of proper therapy. This case is of particular importance because of the rarity cardiogenic UPE in left lung.

SP Singh*, Ragini Singh+, D Gautam**, Ravikant**, R Bassi#, SD Bassi##

*Professor of Medicine (Cardiology Division); +Assistant Professor, Department of Pediatrics; **Assistant Professor, #Junior Resident, ###Professor and Head, Department of Medicine; SGRR Institute of Medical and Health Sciences, Patel Nagar, Dehradun - 248 001, Uttaranchal.

Received : 21.5.2007; Accepted : 26.6.2007

REFERENCES

1. Hassan W, El Shaer F, Fawzy ME, Al Helaly S, Hegazy H, Akhras N. Cardiac unilateral pulmonary edema: is it really a rare presentation?

Announcement

TOXOC4N-4


Organized by : The Indian Society of Toxicology

For further details contact : Dr. VV Pillay, Organizing Secretary, Houseboat Conference (TOXOC4N-4), Dept. of Analytical Toxicology, Amrita Institute of Medical Sciences, Cochin 682 026.

Phones : 0484-4008056 (direct), 0484-4001234, Extn. 8056, 6034
Email : toxicology@aims.amrita.edu; poisonunit@aims.amrita.edu