Munchausen syndrome involving pets by proxies

In a letter in 1998 we drew attention to the pets by proxies Munchausen syndrome involving patients by proxies. Recently Munro and Thrusfield from the Royal School of Veterinary Studies, University of Edinburgh have published a paper in the Journal of Small Animal Practice, documenting the first series of reports of suspected Munchausen syndrome by proxy involving pets as proxies.1 In the study, 1000 randomly selected veterinary surgeons received a questionnaire specifically asking for details of their perceptions and experience of non-accidental injury in animals. A total of 448 cases were described, six of which were described by the respondents as possible Munchausen syndrome by proxy. Three other cases were identified by the authors as possible Munchausen syndrome by proxy. The nine cases are all described and show similarity to child proxy incidents. Common features include frequent requests for clinical review (up to four times in one day in one case), and frequent change of veterinarian ("veterinarian shopping"). In some cases the mode of clinical presentation was similar to that seen in paediatric practice, for example, presentation with haematuria or uncontrolled fitting. In one case a dog owner was insistent that a neighbour had poisoned his dog, but he was later convicted for the attempted poisoning of his child; in court it was revealed that he had previously attempted to poison two other pets treated by other veterinarians. In another case a cat owner gave an incoherent history with regard to the cause of injuries, and postoperative trauma occurred to the intramedullary pin. Repeated problems arose until the cat was admitted.

The authors conclude that their findings should not only inform the small animal practitioner about a curious syndrome but also form the basis of broader debate in comparison between the experience of the veterinary and medical professions. Communication between child protection agencies, veterinary surgeons, and the RSPCA is beginning to occur in different parts of the country. Such liaison should be welcomed by paediatricians.

H S Tucker
Paediatric Specialist Registrar, Royal United Hospital, Bath, UK
F Finlay
Consultant Community Paediatrician, Child Health Department, Bath NHS House, Newbridge Hill, Bath BA1 3GE, UK
S Guiton
Veterinary Surgeon, 12 Ruby Place, Bath BA2 4EH, UK

The prevalence of rickets among non-Caucasian children

We welcome the timely review by Shaw and Pal2 on the continuing problem of vitamin D deficiency among South East Asians living in the UK. Since our report in 1999,1 we have continued to see 8–10 non-white toddlers with florid vitamin D deficiency rickets per year, at our inner city general paediatric unit. A recent national survey showed that 20–34% of South East Asian children had biochemical evidence of vitamin D deficiency.1 However, there is no information on the prevalence of clinical rickets among non-Caucasian children in the UK.

We opportunistically studied 6–36 month old children from ethnic minority backgrounds who were brought to a child health clinic in Central Manchester between 30 May 2001 and 12 July 2000 for immunisations, weight checks, hearing tests, and developmental assessments. A structured questionnaire was used to determine if the children were receiving vitamin D supplements and whether they had been prescribed by health professionals or bought “over the counter” by the parents. Arms and legs of children were examined for deformities and swelling of the metaphyses due to rickets. Children with clinical stigmata of rickets had an x ray of their left wrist and estimation of serum calcium, phosphorus, alkaline phosphatase (ALP), parathyroid hormone (PTH), 25-hydroxyvitamin D (25OHD), and 1,25-dihydroxyvitamin D (1,25(OH)2D). Ethnicity was self determined by parents as South East Asian (Pakistani, Bangladeshi, or Indian), African, Afro-Caribbean, and Middle Eastern origin. The study was approved by the Central Manchester Research Ethics Committee.

A total of 124 children (mean age 15.4 (8.2) months) were studied. Seventy seven per cent of children were of South East Asian origin and almost 50% were of Pakistani origin. Thirty (24%) children were receiving vitamin supplements; in 13 (43%) multivitamin preparations had been bought “over the counter” by the parents. Three children had clinical stigmata of rickets. Table 1 shows biochemical results. Two (16%) of these had radiological features of moderately severe rickets (fig 1) and the third had metaphyseal sclerosis, indicating healed rickets. One child was noted to be pale; his haemoglobin was 62 g/l (normal >110 g/l).

The Department of Health’s Committee on Medical Aspects of Food Policy (COMA) recommends vitamin D supplements for all children up to 3 years, and up to 5 years in those at high risk of developing vitamin D deficiency.1 It was therefore disappointing that less than a quarter of the subjects studied were receiving vitamin D supplements. Two children (cases 1 and 2) were found to have clinical and radiological evidence of active rickets. As shown in table 1, they also had biochemical features of rickets with elevated serum ALP activity for age, low serum 25OHD (a measure of an individual’s vitamin D status), and secondary hyperparathyroidism. All three had been breast fed for periods ranging between four and nine months after birth and none had been prescribed vitamin D supplements.

According to the 1991 census data there were approximately 4000, 6–12 month old children of ethnic minority background resident in the city of Manchester. We found that 1.6% of the children examined had rickets. If this figure were extrapolated to all 6–36

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Ethnic origin</th>
<th>Age (months)</th>
<th>Calcium (mmol/l)</th>
<th>Phosphorous (mmol/l)</th>
<th>Alkaline phosphatase (IU/l)</th>
<th>Parathyroid hormone (pg/ml)</th>
<th>25OHD (nmol/l)</th>
<th>25OHD (pg/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Pakistani</td>
<td>15</td>
<td>2.3</td>
<td>1.5</td>
<td>1859</td>
<td>192</td>
<td>10</td>
<td>103</td>
</tr>
<tr>
<td>2</td>
<td>Pakistani†</td>
<td>8</td>
<td>2.4</td>
<td>1.4</td>
<td>1377</td>
<td>288</td>
<td>16</td>
<td>Insufficient sample</td>
</tr>
<tr>
<td>3</td>
<td>Pakistani</td>
<td>9</td>
<td>2.5</td>
<td>1.6</td>
<td>697</td>
<td>48</td>
<td>13</td>
<td>145</td>
</tr>
</tbody>
</table>

The normal ranges for biochemical variables are shown in parentheses.

1 Cases 1 and 2 had radiological features of rickets; a radiograph of case 1 is shown in fig 1.
2 Parents of this child has started him on an “over the counter” purchased multivitamin preparation between being seen in the child health clinic and venepuncture, 10 days later.

Table 1. Ethnic origin, age, and biochemical results in three children with clinical stigmata of rickets.
and Pal immunisation. result in missed opportunities for care providers, mainly overly cautious on childhood immunisations. Paediatricians’ misconceptions

REFERENCES

COMA. The format of this book and layout of the information within the chapters is traditional. Each chapter provides comprehensive coverage of a specific syndrome, including the clinical aspects, differential diagnoses and genetics where known. The topics are well referenced and accompanied by many good illustrations. There are in addition chapters on chronic and overgrowth syndromes, endocrine causes of overgrowth, and non-syndromic overgrowth, although the latter is rather brief and disappointing. An introductory chapter has fascinating historical facts about overgrowth and the way it features in ancient mythology. Some tales are hard to swallow, such as the birth of the mythical giant Gargantua through his mother’s ear! A later discussion confirms that the “Elephant Man”, Joseph Merrick, had the condition of Proteus syndrome and not, as was originally thought, neurofibromatosis.

I found this book to be a very interesting read, and a superb source of references. The tables of clinical features in each chapter are also useful check lists when considering syndromic diagnoses. The molecular genetic information is reasonably up to date although it is unfortunate for the editors that one of the great mysteries of overgrowth, the cause of Sotos syndrome, was discovered just after this book was published. I am not so sure that this book would be the first choice of general paediatricians if their budget were limited, as even though the authors have provided clear explanations of many of the genetic mechanisms involved, some readers would find the more complicated genetic explanations containing a lot of molecular genetics jargon hard going. The book would also be of more relevance to those caring for children with overgrowth disorders on a day to day basis if there was more information on the general management of overgrowth syndromes. Although the issue of increased risk of neoplasia in overgrowth syndromes is raised, for example, there is no general guidance on screening affected children for tumours in childhood.

Despite these criticisms, I am sure that all paediatricians would enjoy delving into this book as they could not fail to find something of interest within its pages. From the point of view of the clinical geneticist or growth specialist the book provides a good overall review of overgrowth syndromes and will be a useful resource.

J Clayton-Smith

Handbook of Paediatric Intensive Care


George Bernard Shaw said “we have not lost faith, but we have transferred it from God to the medical profession.” Paediatric intensive care was born from the increasing technology and sophistication expected from medicine and is still a new and evolving speciality. A very small number of children will have the month old non-Caucasian children living in the Manchester area, we estimate that there would be approximately 60 children with rickets at the time of this study. Rickets is not a historical disease, as it seems to be perceived by many health professionals. However, it is an entirely preventable by use of vitamin D supplements as recommended by the COMA. We wholeheartedly agree with Shaw and Pal that a nationwide campaign, similar to the “Stop Rickets” campaign in the 1980s is needed to tackle this problem.

S Ashraf, M Z Mughal

Department of Paediatric Medicine, Saint Mary’s Hospital for Women & Children, Hathersage Road, Manchester M13 OJH, UK; zulf.mughal@man.ac.uk

REFERENCES


BOOK REVIEWS

Overgrowth Syndromes


Paediatricians are often more concerned about failure to grow rather than excessive growth. Bigger is not always better, however, and this relatively slim volume provides a wealth of information about almost all of the recognised overgrowth syndromes. Edited by some of the world’s experts on growth disorders, the focus is on multiple anomaly syndromes which have generalised or partial overgrowth as a feature. Although many of the conditions are rare, some, such as Fragile X syndrome and neurofibromatosis, are common enough for this book to be of relevance to the generalist as well as the specialist. Along with details of conditions such as Sotos syndrome and Beckwith-Wiedemann syndrome there is information on many newly delineated overgrowth syndromes, reflecting the increased interest in overgrowth and the mechanisms giving rise to it over the past few years.

The format of this book and layout of the information within the chapters is traditional. Each chapter provides comprehensive coverage of a specific syndrome, including the clinical aspects, differential diagnoses and genetics where known. The topics are well referenced and accompanied by many good illustrations. There are in addition chapters on chronic and overgrowth syndromes, endocrine causes of overgrowth, and non-syndromic overgrowth, although the latter is rather brief and disappointing. An introductory chapter has fascinating historical facts about overgrowth and the way it features in ancient mythology. Some tales are hard to swallow, such as the birth of the mythical giant Gargantua through his mother’s ear! A later discussion confirms that the “Elephant Man”, Joseph Merrick, had the condition of Proteus syndrome and not, as was originally thought, neurofibromatosis.

I found this book to be a very interesting read, and a superb source of references. The tables of clinical features in each chapter are also useful check lists when considering syndromic diagnoses. The molecular genetic information is reasonably up to date although it is unfortunate for the editors that one of the great mysteries of overgrowth, the cause of Sotos syndrome, was discovered just after this book was published. I am not so sure that this book would be the first choice of general paediatricians if their budget were limited, as even though the authors have provided clear explanations of many of the genetic mechanisms involved, some readers would find the more complicated genetic explanations containing a lot of molecular genetics jargon hard going. The book would also be of more relevance to those caring for children with overgrowth disorders on a day to day basis if there was more information on the general management of overgrowth syndromes. Although the issue of increased risk of neoplasia in overgrowth syndromes is raised, for example, there is no general guidance on screening affected children for tumours in childhood.

Despite these criticisms, I am sure that all paediatricians would enjoy delving into this book as they could not fail to find something of interest within its pages. From the point of view of the clinical geneticist or growth specialist the book provides a good overall review of overgrowth syndromes and will be a useful resource.

J Clayton-Smith

Handbook of Paediatric Intensive Care


George Bernard Shaw said “we have not lost faith, but we have transferred it from God to the medical profession.” Paediatric intensive care was born from the increasing technology and sophistication expected from medicine and is still a new and evolving speciality. A very small number of children will have the
misfortune to need to be treated in a paediatric intensive care unit (PICU). It’s an expensive business and often a very emotional one; but very real paediatrics. There are few PICUs in the UK and only six centres that are fully recognised for formal PICU training. Trainees may feel that mercifully few of them will rotate through PICUs as part of their training and that perhaps a book like this might not be for them. If that describes you I would encourage you to think again. Most PICUs are staffed with juniors who are paediatricians in training. Even if they decide that other areas in paediatrics interest them more, I believe most find their time in PICU valuable, varied and exciting. You won’t forget it. You won’t regret it. This book might suit you and there are not that many like it in the bookshops.

Gale Pearson is undoubtedly well qualified to write this book as clinical director in one of the largest units in the UK. Some of his passion for the subject comes through the text even when discussing “developing a Bayesian approach to PICU”. In a slim textbook a great breadth of the subject is covered including respiratory physiology, audit, congenital heart disease, and nutrition for example. Despite the breadth this book appears to me to have some depth and may be less of a handbook and more of an introductory textbook to PICU. There are valuable management suggestions and algorithms but much of the book is carefully selected background to the problems encountered in clinical practice and requires time to read and take it in. From a personal point of view there were a few areas that perhaps needed more coverage. If the intention was a bias to include common causes of admission to PICU, I think bronchiolitis deserved more than the 10 lines it got but maybe that’s my bias.

The book ends with a concise but detailed chapter: “Issues surrounding death on the PICU”. Public expectation is very high. Despite the public’s faith we are not the deities they see on the television and certainly not as good looking (not in the units I’ve worked in). We do our best—sometimes we fail. Samuel Beckett said: “Ever tried. Ever failed. No matter. Try Again. Fail again. Fail better.”

R O’Donnell

Practical Endocrinology and Diabetes in Children

This concise textbook has proved extremely useful in general paediatric ward and clinic work. Problem based chapters give an overview of the relevant physiology and a practical guide to examination, investigation, and management of a broad range of childhood endocrine disorders. Each chapter includes an interesting reference to controversial points in each field, future developments and example case history problems. There is also guidance on when to involve a specialist centre.

The authors have achieved consistency in the depth and approach of all chapters. The layout is modern, clear, and well illustrated which makes the book extremely readable. The more unusual clinical problems such as intersex are included with remarkable depth and clarity for such a short text. The chapters on the common problems have been well thought out to cover practical questions. For example, the guidance on management of diabetes includes problems of long haul travel, surgery, alcohol use, and contraception in addition to practical aspects of ketoacidosis and cerebral oedema treatment.

The management guidance points are not individually referenced for level of evidence but represent established current practice. General references and key papers are given at the end of each chapter. Appendices list UK patient support group contacts and several growth charts, although height velocity and decimal age charts are not included.

The book is aimed at paediatricians in training and general paediatricians. In my department the book has also been used by nursing staff and doctors training in adult endocrinology. The authors achieve the difficult task of providing a text that is a good preparation for examinations in addition to a practical day-to-day guide. I would recommend the book to all candidates.

D P Smith
Scimitar syndrome as a differential diagnosis in a child with recurrent wheeze

Respiratory symptoms of cough, wheeze, and breathlessness account for 40% of referrals to a general paediatric clinic. The majority of these children suffer from “wheeze secondary to upper respiratory tract infection” and “asthma.”

A 7 year old girl was referred by her general practitioner to the clinic with a two month history of persistent cough and recurrent wheeze; she had been treated for suspected asthma with fluticasone and salbutamol since early childhood. There was a history of infantile eczema. She was growing well on the 50th centile. General examination was normal. There was no cyanosis or clubbing. Respiratory and cardiovascular system examinations were unremarkable.

She had been admitted at the age of 14 months with cough and wheeze; chest x ray showed right lower lobe consolidation which improved on antibiotics. Both radiographs were studied and the pattern of curvilinear density (scimitar) in the right lower zone suggestive of scimitar syndrome was recognised. She was referred to the paediatric cardiology department for echocardiography, which showed right lower lobe consolidation which may be a mild variant of scimitar syndrome.

A cardiac catheterisation and coil embolisation of the systemic pulmonary collateral from the descending aorta to the right lower lobe is scheduled.

Scimitar syndrome is a name given to the triad of: (1) curvilinear vascular density in the right lower zone; (2) hypoplastic right lung; and (3) dextroposition of the heart. It has a wide spectrum of presentation and may sometimes only present in adulthood with symptoms of wheeze, recurrent chest infections, or pulmonary hypertension. It remains a notoriously difficult diagnosis to make without a strong index of suspicion. In this case, pattern recognition on chest radiograph helped us to suspect the diagnosis. Examination and ECG may be entirely normal or just show right sided strain. Echocardiogram may also be normal or show dilated right sided chambers (as in this case). Diagnosis can be missed in up to 33% cases by echocardiography. More sensitive tests would include computed tomography scan, cardiac catheterisation, and magnetic resonance imaging with 3D MRA. Obstructive and early symptomatic types will usually need corrective surgery after stabilisation. Milder scimitar variant will probably do well with occlusion of the collateral supply.

We have presented this case to highlight the fact that one has to keep an open mind regarding the final diagnosis in any child with recurrent wheeze, as all wheezes are not “asthma.”

P R Desai, M Babu
Department of Paediatrics, St John’s Hospital, Chelmsford CM2 9B, UK; prpravin@yahoo.com

References

Assessment of acute admissions by middle grade trainees and consultants will reduce the need for overnight hospital admissions

We carried out an audit to assess the impact on hospital admissions of patients being assessed by either middle grade trainees (residents) or consultants in a district general hospital (DGH). Our aims were to establish:

- Number of children kept in hospital overnight and those discharged the same day
- Number of readmissions of those discharged the same day
- Any adverse events in those discharged home the same day

We studied retrospectively all acute admissions to the children’s wards at Doncaster Royal Infirmary, a medium sized district general hospital, over the months of January and July 1998. We excluded all surgical and non-acute admissions. At the time of the study the Children’s Hospital did not have a day or acute assessment unit. Therefore the children were reviewed following admission to the wards. Whether trainees or consultants reviewed patients was an entirely random process, dependent on willingness and time to carry out ward rounds in late afternoon or early evening. The review could also be triggered by nursing staff or parents. The interval between the time of admission to the ward and the time the patients were reviewed varied from immediate review to a few hours. The decision to discharge children was usually taken jointly by medical and nursing staff, provided that parents were willing to look after their children at home. The parents of children discharged home on the same day as admission were given open access to the children’s ward—that is, they could either telephone the ward for advice or return with the child if concerned.

A total of 512 sets of case notes were reviewed by MMM and RAS. A pro forma was used to collect the data, which was stored on an Excel spreadsheet.

A total of 173 (34%) patients were under 1 year, 150 (29%) were 1–2 years, 53 (10%) were 3–4 years, 41 (8%) were 5–6 years, and 95 (18%) were over 6 years (fig 1). The source of referral was documented in 499 case notes. Of these, 287 (58%) were via a general practitioner, 178 (36%) were via the accident and emergency department, and 29 were from other sources. The commonest reason for admission was breathing difficulties followed by fever.

Of the 512 patients admitted, 260 (51%) were reviewed by middle grade trainees or consultants. Of those reviewed, 109 (42%)
were discharged home the same day. The age
group distribution (fig 1) and reason for
admission (fig 2) of those reviewed was simi-
lar to that of the total sample. More children
under 1 year were kept in overnight than were
discharged home the same day; the reverse
was true for those in the 1–2 year and 3–4 year
age groups. The reason for admission of those
discharged home the same day was also simi-
lar to that of the total sample. Slightly more
patients were admitted in January than in July,
but more patients were discharged home the
same day in July than in January (26% v
15%; fig 3). This could well be due to the fact
that there is more pressure on beds in the
winter months. However, it could also be due
to a different spectrum and severity of
diseases.

Of those discharged home the same day, seven
(6%) were readmitted within seven
days, four because of the same complaint and
three with a different complaint. There were
no adverse events. Those who were reviewed
but kept in overnight had a similar distribu-
tion of the reason for admission to that of the
total sample and those who were reviewed but
with an excess of vomiting and/or diarrhoea.

In conclusion, assessing the need for
admission resulted in 20% of all admissions
(40% of those reviewed) being discharged
home the same day. Vomiting and/or diar-
rhoea were more likely to result in patients
being kept in overnight. We believe the
number of patients who can be discharged
home the same day will be much higher if all
acute admissions are reviewed and assessed
in the way described. This policy seems safe
and acceptable to parents.

With the planned reduction in the number
of specialist registrars, it seems that expand-
ning the number of consultants would achieve
the dual benefit of moving closer towards a
consultant provided service and will also lead
to reduction in the number of children requir-
ing an overnight hospital admission.

### Parkinson-like syndrome as the major presenting symptom of Epstein–Barr virus encephalitis

The main symptoms of Epstein–Barr virus
encephalitis (EBV) encephalitis are fever, sei-
zure, bizarre behaviour, headache, and
metamorphosis. Bradykinesia, akathisia, in-
voluntary hand movements, drooling, and
torticollis are symptoms of Parkinson-like
syndrome, which has never been described as
a manifestation of EBV encephalitis. We
report the case of a previously healthy boy
who presented with Parkinson-like syndrome
as the major symptom of EBV encephalitis.

A 12 year old, previously healthy boy was
referred to our hospital because of severe
cough with sputum and intermittent fever
for seven days. Abdominal discomfort and vomit-
ing were also noted one day before admission.

On admission, his consciousness was clear
without focal neurological sign, no hepato-
splenomegaly, no lymphoadenopathy, and no
exudative tonsillitis or skin rash. There was no

previous personal or family history of seizure
disorder or migraine, and both the boy and his
family denied being exposed to some possible
hallucinogenic or neuroleptic drug. Blood
analysis was normal except for a mild
leucocytosis with a left shift (10 500/mm
3, 84.6% neutrophils), and there were no atypi-
cal lymphocytes. C reactive protein (CRP)
level was 0.17 mg/dl (normal <0.3 mg/dl).

After admission, mucolytic agents and
bronchodilators were prescribed. Fever, ab-
dominal discomfort, and vomiting subsided
soon. Twenty four hours after admission, his
condition deteriorated with drowsiness, invol-
untary rhythmic finger tapping movement,
resting tremor, bradykinesia, photosphobia,
and staring eyes. But perceptual distortion
was not noted. Cerebrospinal fluid (CSF)
examination yielded yellow, clear fluid and
normal opening pressure without microor-
ganisms on Gram stain or culture. The cell
count, protein, and sugar of CSF were all
within normal limits. Table 1 lists laboratory
evaluations for EBV and other possible patho-
gens of encephalitis. Brain magnetic reso-
nance imaging (MRI) showed no abnormali-
ties. Tc-99m HMPAO brain SPECT (Tc-99m
hexamethylenepropyleneamine oxime single
photon emission tomography) showed dimin-
ished perfusion in the region of the right cau-
date nucleus. Electroencephalography (EEG)
revealed diffuse slowing of background activ-
ity. His signs and symptoms showed gradual
improvement under close observation in the
following three weeks. He was then dis-
charged in a stable condition. Follow up for
four months later showed no residual neurological
sequelae.

Parkinson-like syndrome (extrapyramidal
symptoms) is characterised by various neuro-
logical symptoms including akathisia, brady-
kinesia, torticollis, drooling, and involuntary
hand movement. This syndrome develops in
at least a quarter of children treated with
neuroleptics due to disruption of the balance
between the dopaminergic system and the
cholinergic system within the basal ganglia.
But Parkinson-like syndrome has also been
recognised as a sequela of acute viral encepha-
litis, including coxsackie B, cytomegalovirus,
measles, herpes simplex virus, Japanese B
encephalitis virus, and encephalitis lethargi-
gica. Mycoplasma pneumoniae infection has also
been recognised as a cause of Parkinson-like
syndrome. In our patient, exposure to possi-
bile hallucinogenic or neuroleptic drugs was
denied. Serological tests and culture for other
possible pathogens were negative. EBV ence-
phalitis was diagnosed by serological and
CSF polymerase chain reaction findings
which fulfilled the diagnostic criteria.

EBV encephalitis is generally considered to
be a benign, self limited disease associated
with few sequelae. However, an incidence of

---

**Table 1: Serological evidence for EBV encephalitis**

<table>
<thead>
<tr>
<th>Day 1</th>
<th>Day 14</th>
</tr>
</thead>
<tbody>
<tr>
<td>EBV VCA IgM</td>
<td>1/32 [positive]</td>
</tr>
<tr>
<td>EBV VCA IgG</td>
<td>1/160</td>
</tr>
<tr>
<td>EBV determined nuclear antigens</td>
<td>Negative</td>
</tr>
<tr>
<td>EBV CSF PCR</td>
<td>Positive</td>
</tr>
<tr>
<td>Mycoplasma pneumoniae IgM</td>
<td>1/140</td>
</tr>
<tr>
<td>Culture for enterovirus</td>
<td>Negative</td>
</tr>
<tr>
<td>Coxsackie B virus IgM</td>
<td>Negative</td>
</tr>
<tr>
<td>Cytomegalovirus IgM</td>
<td>Negative</td>
</tr>
<tr>
<td>Herpes simplex IgG and IgM</td>
<td>Negative</td>
</tr>
<tr>
<td>JBE virus IgM</td>
<td>Negative</td>
</tr>
</tbody>
</table>
neurological sequelae after EBV encephalitis as high as 36% was reported by Domachowske and colleagues. The present case, therefore, not only draws attention to the role of EBV in infectious neurological disorders, but also suggests that an EBV aetiology should be considered in cases of Parkinson-like syndrome in childhood.

J C Hsieh, K H Lue, Y L Lee
Chung Shan Medical University Hospital, 23 Sec. 1 Taijunggang road, Shi Chiu, Taichung, Ta, Republic of China; kent0837@yahoo.com.tw

References

Hyperextension of spine: unusual presentation of Guillain-Barré syndrome
Guillain-Barré syndrome (GBS) classically presents as ascending symmetric areflexic weakness with positive sensory symptoms. Recently, we managed a child presenting with unusual posture and hyperextension of the whole spine.

A 9 year boy presented with inability to hold books and write, and a limp. Over 12 hours he had developed tingling sensations and pain in the calf muscles; pain progressed to involve the neck and back by 24 hours and he was unable to flex his neck and extend his limbs. During the next two days weakness increased, especially of the lower limbs. By day 4, he had developed hyperextension of the cervical and thoracolumbar spine with flexed and adducted limbs. On day 6 when he presented to us, vital signs including blood pressure were normal and remained so during the hospital stay. He had painful restriction of passive extension at all joints; motor power and tone could not therefore be assessed. He had bilateral symmetrical weakness: shoulders (abductor, adductor, 2/5), elbows (flexor 3/5, extensor 2/5), wrists (dorsiflexor, palmar-flexor, 2/5), finger flexors (2/5), hand grip (20–30%), hip flexors (2/5), knees (flexor, extensors, 2/5), ankle (0/5), toes (0/5). Deep tendon reflexes were absent except for the biceps, which also disappeared by day 12. Cere- mastic and abdominal reflexes were present; plantar reflexes were absent bilaterally. The spine was normal except for hyperextension of the cervical and thoracolumbar region. Respira- tory muscles, higher mental functions, speech, cranial nerves, and bowel and bladder functions were normal. A plain radiograph of the spine showed mild thoracic lordosis. Cerebrospinal fluid examination on day 11 showed high protein (95 mg/dl). On day 12, spinal hyperextension and abnormal limb postures disappeared following improvement in pain as a result of analgesic therapy. Kernig’s and Brudzinski’s signs could be elic- ited. Lasegue’s sign and the straight leg rais- ing test were also positive. Symmetrical hypo- tonia became obvious.

GBS was suspected in view of progressive bilateral symmetrical weakness, severe radiculopathy, and albumino-cytological dissociation. Nerve conduction studies, performed on day 23, showed reduced nerve con- duction velocity in the motor nerves. Sensory nerves were normal. We could not determine F wave conduction velocity. The pain and tenderness subsided gradually. With regular physiotherapy the neck became soft, motor power improved, and he was able to sit unsupported by day 20. Four months later, neurological examination was normal. A stool culture for poliovirus was negative.

Children with GBS frequently have pain in the back and lower limbs, which is aggravated on the straight leg raising test in most of them. These pain syndromes are attribut- able to radiculitis, an early and predominant feature in GBS. Prominent radiculitis in this case might have led to severe pain in the back, causing generalised paravertebral muscle spasm. This resulted in the unusual posture of hyperextension of the spine.

A K Baranwal, S C Singh
Department of Pediatrics, BP Koirala Institute of Health Sciences, Dharan, Nepal; baranwal1970@yahoo.com

CORRECTIONS

An error occurred in the letter by S Ashraf and M Z Mughal in the September issue (Arch Dis Child 2002; 87: 263–4). In the fifth paragraph, the first sentence should read “According to the 1991 census data there were approxi- mately 4000, 6–36 month old children of ethnic minority background resident in the city of Manchester.” The journal apologises for the error.

The photograph of children in the Morijate area in Delhi on the cover of the August issue was taken by the American photographer Mark Ingersken of Dallas, Texas.