Intravenous atropine treatment in infantile hypertrophic pyloric stenosis

Hypertrophic pyloric stenosis of infancy is a disorder of early infancy with typical clinical features and well-established radiological appearance of the pyloric canal. Many studies with surgical and medical treatment have been reported over the past fifty years. Pylorotomy has tended to become the favoured method of treatment as with expert paediatric, surgical, anaesthetic, and nursing services and specialised accommodation for infants, the outcome is good with low mortality, short stay in hospital and few complications. However, a variety of studies of medical treatment with anticholinergic drugs and successful outcomes in some large series of cases have also been reported from Sweden, United States of America and the United Kingdom.

Since 1996 this group of workers from Osaka, Japan, has revived an interest in medical treatment with reports of a new regime using methyl atropine nitrate intravenously. To achieve satisfactory short term outcomes considerable variation in drug dosage and modified feeding regimens were necessary which involved much medical supervision and careful monitoring for toxic effects of the drug, which were minimal. The treatment was successful in the relatively small number of infants in the trial (19) with two infants being referred for pylorotomy, no mortality and no serious complications. An interesting part of this paper is the long term clinical follow up of the successfully treated infants over two years and ultrasonography of the pyloric canal which demonstrated the changes in muscle thickness and length of the canal. The disadvantages of the treatment mentioned by the authors are length of hospital stay and the necessity to continue mentioned by the authors are length of hospital stay and the necessity to continue atropine medication orally after discharge home.

Comparing the use of this anticholinergic drug intravenously with oral treatment using methyl scopolamine nitrate and similar restricted feeding regime, oral methyl scopolamine nitrate suppressed vomiting more quickly and reliably, was also available for subcutaneous injection if vomiting recurred as size of feeds was increased, and no toxic effects were seen in any dosage used. It would be interesting if these workers would be prepared to try the use of methyl scopolamine nitrate intravenously as pharmacologically this compound was reported to have a spasmolytic effect on gut to two to three times greater than methyl atropine nitrate with lesser central nervous effects.

This paper serves to emphasise once more that these infants should always be treated in paediatric centres where there is a high level of experienced paediatric care and nurses trained for neonatal special care.

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Author’s reply

We appreciate the interest shown by Dr Beryl Corner with regard to our article. Unfortunately, intravenous atropine therapy is not widely accepted in European countries or the United States; it is however now becoming popular in Japan.

We are truly honoured to receive the comments of Dr Corner, who is a pioneering neonatologist and reported medical treatment with methyl scopolamine nitrate for infantile hypertrophic pyloric stenosis (HIPS) in 1955. She pointed out that methyl scopolamine might be better than atropine sulphate in terms of effectiveness and side effects. One of the reasons why atropine was used in our study is that methyl scopolamine is not available in our country. Scopolamine butyrylcholine is an available quaternary ammonium derivative of scopolamine and lacks toxic side effects. However, this agent tastes bitter and is difficult to give orally to infants. Therefore, this agent is only given intravenously in infants with HIPS.

We do not know if it is worthwhile to attempt combination therapy with intravenous scopolamine butyrylcholine and oral atropine rather than the intravenous and oral atropine therapy. Secondly, we already knew that an intravenous atropine injection of 0.01 mg/kg was effective enough to abolish transiently the phasic and tonic pyloric contractions characteristically of HIPS. We used an intravenous atropine injection of 0.01 mg/kg in our study to confirm that those pyloric contractions were the cause of the disturbed transpyloric flow in this condition by seeing that their inhibition with the dose of atropine ameliorated symptoms.

We agree with Dr Corner’s last comment, but believe that intravenous atropine therapy is possible not only in high level paediatric centres, but also in general hospitals where infusions therapy with intravenous atropine injections can be done safely in small infants. Clinical trials are now ongoing to establish more efficient treatment strategy for HIPS with medical and surgical therapy in our country.

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References
Among their siblings, five of 126 had died of SIDS. All five children were full term infants. The average maternal age, birth weight, and age at death were respectively 27.4 (3.5) years, 3.3 (0.3) kg, and 3.5 (1.1) months. The rates of SIDS in siblings of children with VO were compared to the in the general population using the standardised incidence ratio (SIR), which is the ratio of the observed number to the expected number of cases of SIDS calculated using the French incidence rates. The expected number of SIDS was 0.17 and hence the SIR was 29.4 (95% CI 9.5 to 68.6; p < 0.000011). Our result showed an overall significant excess of SIDS among siblings of children with VO compared to the children who recruited did not come to the centre because of a family history of SIDS. Since children with a positive family history of SIDS could be followed up more regularly than others, we estimated the SIR separately among siblings of children recruited during their follow up and those of children recruited during their first visit, and verified that there was no significant difference in SIR between these cases. Despite the marked decline in SIDS, it is still the leading cause of postneonatal mortality. Better knowledge of other risk factors may allow identification of populations at high risk and may decrease in infant mortality from SIDS through the implementa- tion of appropriate prevention measures. Our findings suggest that VO may be involved in SIDS and that children with VO or a family history of VO may be a population at potential high risk of SIDS.

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**Vagal overactivity: a risk factor of sudden infant death syndrome?**

Since early 1990, the incidence of sudden infant death syndrome (SIDS) has dropped sharply because of public health campaigns decrying the dangers of the prone sleep position. The other known risk factors, such as preterm birth and young maternal age, are less susceptible to campaign intervention.

**Disordered autonomic function, including cardiorespiratory control, has been suggested to be involved in SIDS.**

Vagal overactivity (VO), characterised by breath holding spells and repeated synapses in specific circumstances, has been described as a manifestation of disordered autonomic function. To investigate a possible relation between VO and SIDS, we investigated 65 children presenting documented VO; for example, clinical characteristics and a positive test for eyeball compression and/or electrocardiographic monitoring. Participants of these children were interviewed about their family history, especially with respect to the occurrence of SIDS among their other children.

**Perforated duodenal ulcer disclosing medium chain acyl-CoA dehydrogenase deficiency**

Medium chain acyl-CoA dehydrogenase deficiency (MCADD; McKusick 201430) typically presents in the first two years of life with recurrent episodes of hypoketotic hypoglycaemia, lethargy, coma, or sudden infant death. The trigger may be fasting, intercurrent infections, anaesthesia, or surgery. Incidence in the UK is estimated at 0.45–1/10000 live births.1 We describe the case of a child who presented with marked encephalopathy unexplained by perforated duodenal ulcer, which led to the diagnosis of MCADD.

A 2 year old girl presented with a three week history of cryptal symptoms and three day history of frequent coffee ground vomiting. She was shocked, and had hepatomegaly and decreased conscious level. Blood glucose was 3.9 mmol/l (reference interval 3.5–5.3), plasma sodium 129 mmol/l (135–147), potassium 5.2 mmol/l (3.5–5.0), urea 17.8 mmol/l (3.3–6.6), creatinine 36 mmol/l (30–74), bicarbonate 15 mmol/l (21–28), albumin 30 mmol/l (4.5–0.5 mg/l. Liver function tests and clotting were normal. She was resuscitated with a total of 50 ml/kg of colloid and crystalloid. The following day she relapsed with abdominal distension, shock, and deteriorating conscious level. Investigations showed glucose 14.2 mmol/l, amylase 20 IU/l (8–85), AST 186 IU/l (10–45), and ALT 129 IU/l (10–40). An x ray examination of the abdomen showed free air under the right hemidiaphragm. Emergency laparotomy revealed a single, 1 cm × 1 cm acute perforation in the second part of the duodenum. Histology and rapid urease test (CUO) of the duodenal biopsy for *Helicobacter pylori* were negative. Fasting blood gastrin was 20 mU/l (10–100). She was discharged home taking omeprazole. Upper endoscopic biopsy (eight weeks later) for histopathology and CUO test from oesophagus, stomach, antrum, and duodenum were normal.

Analysis of urinary organic acids by gas chromatography and mass spectrometry, obtained a day after clinical presentation, revealed a marked 5-hydroxyhexanoic acid (21% of total organic acids); a modest dicarboxylic acidiciduria (sulbic acid accounted for 8% and adipic 6% of total organic acids); and a small but significant quantity of hexanoyl glycine (2% total organic acids) in the absence of ketonuria.

Blood obtained a week after clinical presentation, when analysed by tandem mass spectrometry, showed octanoylcarnitine 0.19 mmol/l (0.19), hexanoylcarnitine 0.67 mmol/l (0.29), and deconooylcarnitine 0.63 mmol/l (0.10), with a subnormal concentra- tion of acetylcarnitine 4.0 mmol/l (6.2–27.5). This profile was consistent with MCADD. Polymerase chain reaction/restriction digests based method revealed two mutations in the MCAD gene.

The clinical details coupled with the absence of ketones and the increased 5-hydroxyhexanoic acid led us to look for an abnormality in the oxidation of fatty acids, and resulted in identification of the minor constituent, hexanoylglycine that is recognised as an indicative marker of MCADD. Increases in urinary hexanoylglycine and 5-hydroxyhexanoic acids in the absence of ketonuria have been reported previously in MCADD patients during clinical attack, and also in a boy who died.1 Our case was unusual in that the amount of 5-hydroxyhexanoic acid was greater than even the sum of the individual dicarboxylic acids present, although high levels of 5-hydroxyhexanoic acids are reported in acute episodes.1 The increased concentration of octonoyl carnitine in blood was also consistent with a diagnosis of MCADD. We believe that this is the first report of MCADD presenting with duodenal ulcer. It could be argued that the ulcer was the primary problem and that the decompen- saion was caused by the subsequent illness.
Thus, any child who has unexplained en- 
cephalopathy, regardless of its cause and 
clinical setting, should be screened for 
MCADD.

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Glucose metabolism in sleep 
disordered breathing

An association between sleep disordered 
breathing (SDB) and impaired glucose 
tolerance has been reported in adults.1 Although 
SDB has been reported in diabetic children, 
no data are available on glucose metabolism 
in children with SDB. We used glycated 
haemoglobin (HbA1c) for the preliminary 
assessment of glucose metabolism in paediat-
ric SDB patients.

HbA1c was measured in 12 children aged 
26–116 months (mean 63) with suspected 
SDB. Overnight polysomnographic studies 
were obtained from the child if older than 5 years of 
age. The study excluded 11 studies comparing single-dose or 
single-day treatment. Inclu-
ded 3 days of treatment 
to 7–14 days of treatment, whereas Michael et al compared 2–4 days of 
treatment to 7–14 days of treatment and 
excluded 11 studies comparing single-dose or 
single-day treatment to standard duration treatment.1-3

The reasons for this exclusion are unclear, 
although we assume that they felt single-dose or single-day treatment was not a fair comparison with 7–14 day treat-
ment. However, a number of randomised 
controlled trials (RCTs) made this compar-
ison, suggesting that clinicians are, in fact, 
interested in the potential efficacy (and 
safety) of short course treatment. When 
we excluded these studies in a sub-group 
analysis of 3-day versus long course (7–14 
day) treatment, the risk of treatment failure 
fell to 1.36 (95% CI 0.68 to 2.72) (NNT=50; 
95% CI 33–13).

Thus, our meta-analysis demonstrates 
that single dose or single day antibiotic 
treatment is not as effective as long-course 
treatment for UTIs in children. The two 
meta-analyses together suggest that: 
(1) “longer” short-course therapies may be as 
effective as 7–14 days of antibiotics and

Short versus standard duration 
antibiotic treatment for UTIs: a 
comparison of two 
meta-analyses

Having recently published a meta-analysis on the 
same clinical question, it was with great 
interest that we read Michael et al’s systematic 
review of short versus standard duration anti-
biotics for urinary tract infections (UTIs) in 
children.2 Given the publication (in close 
collision) of two meta-analyses on the same 
question with (on the surface) strikingly dif-
ferent results, we thought a comment was in 
order.

First, we applaud the authors on their 
methodologically sound review. The litera-
ture search was explicitly described and 
exhaustive. In fact, the authors identified a 
number of studies that we had missed.3 “The study 
outcomes for meta-analysis (frequency of 
positive urine cultures at 0–7 days after treat-
ment and at 10 days to 15 months after treat-
ment, and development of resistant organ-
isms and recurrent UTIs) were relevant and 
clearly defined.

The authors provided appropriate and im-
portant meta-analysis measures including 
summary relative risks (RRs) and a quasi-
NNT calculation with varying risk of treat-
ment failure in the standard treatment group 
and confidence intervals corresponding to “best” and “worst” case scenarios.

For their primary outcome, frequency of 
positive urine cultures 0–7 days after treat-
ment, the authors found no significant 
difference between short course (3 days) and 
standard (7–14 days) treatment (RR 1.06; 
95% CI 0.64 to 1.76). This is in contrast to our 
finding of a 94% increased pooled risk of 
treatment failure with short course treat-
ment (vs 3 days) compared to standard treat-
ment (7–14 days) (RR 1.94, 95% CI 1.19 to 
3.15; NNT=15, 95% CI 100 to 7). Why the 
discrepancy? We postulate a few possible 
explanations and conclude that the two 
meta-analyses, on closer inspection, actually 
have very similar results.

Our omission of certain studies identified 
by Michael and colleagues may have biased 
our results. However, of the three studies 
that we missed and that they included in 
their analysis of treatment failure at 0–7 days 
after completion of treatment, two favoured 
standard duration treatment, which would 
have supported our pooled RR result. Another 
possible explanation for the observed discrep-
ancy was the use of different definitions of 
treatment failure. For our definition of 
treatment failure we pooled persistent infection 
and treatment failure to eradicate the organism 
within 2 weeks of cessation of treatment as 
their primary outcome measure of treat-
ment failure. If infections relapsed within 7 days 
after cessation of treatment occurred more 
often in recipients of short course treatment, 
then Michael et al’s definition of treatment 
failure could have failed to capture the thera-
pic advantage of standard duration treat-
ment.3

However, the most likely explanation for 
the divergent results was the different ways 
in which the study question was framed and 
the resulting differences in studies included 
in the meta-analyses. We compared 3 days of 
treatment to 7–14 days of treatment, whereas Michael et al compared 2–4 days of 
treatment to 7–14 days of treatment and 
excluded 11 studies comparing single-dose or 
single-day treatment to standard duration treatment.3

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treatment to 7–14 days of treatment and 
excluded 11 studies comparing single-dose or 
single-day treatment to standard duration treatment.3
(2) there is probably a duration of treatment threshold for “short-course” antibiotic treatment, above which longer duration of treatment confers no therapeutic advantage.

Michael and colleagues suggest that as little as 2 days of treatment may be sufficient. However, only one of the trials in their meta-analysis studied 2-day treatment and that only in combination with short-course treatment with a RR of UTI 0–7 days after completing short course treatment of 2.17 (95% CI 0.48 to 9.76). The duration of treatment threshold may be 3 days, but the point estimate of relative risk of treatment failure with 3-day treatment in their meta-analysis suggests otherwise. If the duration of short-course treatment for which there is no difference in efficacy compared with standard treatment is actually greater than 3 days, then the added convenience and cost-savings of “short-course” treatment become marginal. In the absence of appropriately powered RCTs (or meta-analyses) examining outcomes (treatment failure, reinfection, emergence of resistant organisms and cost) with “longer” short course treatment regimens (3, 4, and 5 days), we think that clinicians should continue to treat UTIs in children with at least 7 days of antibiotics.

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References

Table 1 Results of three systematic reviews of randomised controlled trials comparing short duration with standard duration of antibiotic treatment for lower tract urinary tract infection.

<table>
<thead>
<tr>
<th>Systematic review</th>
<th>Comparison of duration of therapy</th>
<th>Number of data sets</th>
<th>Risk for persistent bacteriuria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tran et al, 2001</td>
<td>1–4 days v 5–10 days</td>
<td>13</td>
<td>RD 4.26 (95% CI 0.95, 9.48)</td>
</tr>
<tr>
<td>Keren &amp; Chan, 2002</td>
<td>3 days v 7–14 days</td>
<td>5</td>
<td>RR 1.36 (95% CI 0.68, 2.72)</td>
</tr>
<tr>
<td>Michael et al, 2002</td>
<td>2–4 days v 7–14 days</td>
<td>8</td>
<td>RR 1.06 (95% CI 0.64, 1.76)</td>
</tr>
</tbody>
</table>

*RD, risk difference; CI, confidence intervals; RR, relative risk.

Because there is no significant difference between short duration and standard duration antibiotic treatment for the number of children with persistent UTI after treatment, it is not possible to calculate a number needed to treat to prevent one episode of persistent bacteriuria.

From our systematic review, we are not able to determine whether there is an “optimum duration of treatment threshold” as postulated by Keren and Chan. Only one study included in the meta-analysis, examining the effects of short duration and standard duration treatment in clearing bacteriuria, compared 2 days of treatment with 10 days’ treatment. In their letter above, Keren and Chan argue that this study favours standard duration treatment. However, there was no significant difference between treatments in the number of children with persistent bacteriuria at the end of treatment (RR 2.17; 95% CI 0.48 to 9.76) although the wide confidence intervals do not allow the possibility that short duration treatment could be more or less effective than standard duration treatment.

No significant differences in the number of children with persistent UTI after treatment between short duration and standard duration antibiotic treatment have been found in three systematic reviews of randomised controlled trials despite different study inclusion criteria and definitions of persistent infection. As addressed in our review, the wide confidence intervals around the summary estimates indicate residual imprecision in the results. However, this statistical imprecision is of doubtful significance for most children, who are at a low risk (1–3%) of persistent UTI at the end of treatment following their first lower tract UTI. Therefore, we do not support Keren and Chan’s conclusion that clinicians should continue to treat UTI with standard duration treatment. Instead, we believe that short duration treatment may be used to treat children with lower tract UTI.

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References

Authors’ reply
In response to Keren and Chan’s thoughtful letter regarding our recent systematic review, we need to emphasise that the study question we addressed was different from that addressed by Keren and Chan in their own systematic review of randomised controlled trials comparing short with standard duration treatment in the treatment of children with urinary tract infection (UTI). The aim of our study was to determine the relative efficacies of short (2–4 days) and standard duration (7–14 days) treatment with the hypothesis that short duration may be as effective as standard duration treatment and provide potential advantages such as improved compliance. Therefore, we did not include trials in which single dose treatment was compared with standard duration treatment. In addition we chose to limit the review to trials in which the same antibiotic was used to treat each group, to avoid confounding.

The response to single dose treatment appears different from short course, suggesting that it is inappropriate to pool studies comparing single dose and standard treatment with those comparing short course and standard treatment. Three systematic reviews1 have now demonstrated that there is no significant difference in the number of children with persistent bacteriuria after short duration or standard duration treatment (see table 1). In contrast, Keren and Chan found that significantly more children had persistent bacteriuria following single dose compared with standard duration treatment (7 data sets compared, OR 3.73, 95% CI 1.38 to 10.89). Similarly, Tran et al in their meta-analysis of 22 studies comparing both single dose and short duration treatment with standard duration treatment found the latter to be more effective (risk difference 6.38; 95% CI 1.88 to 10.89).
Is life long follow up for patients with Kawasaki disease indicated?

Brogan et al recommended life long follow up for patients with Kawasaki disease, including those who do not have coronary artery involvement. The reason they quoted was to document the blood pressure and provide general advice regarding other risk factors.

The American Heart Association recommends echocardiographic (EKG) evaluation of the coronary arteries at presentation and follow up EKG at 6–8 weeks and 6–12 months after the onset of symptoms for those who did not have or just have transient coronary artery involvement. They do not recommend follow up after first year unless cardiac disease is suspected.

Tuohy et al demonstrated, in their multi-institutional review of 536 patients, that no patient with a normal follow up EKG, performed within 2 months following disease onset, subsequently developed echocardiographic coronary artery abnormalities. Even those patients with initial echocardiographic abnormalities that became normal at 1–2 months remained normal thereafter.

Scott and colleagues showed that no patient with a normal EKG at 2 weeks to 2 months after the onset of symptoms had subsequent ECGs that revealed coronary artery abnormalities, and questioned the value of 6–12 month ECG in relation to growth hormone therapy are methodologically flawed because neither have accounted for the change in apparent bone density, which will occur in any child who grows better for any reason when assessed by modalities such as dual energy x-ray absorptiometry.

As illustrated by another article in the August 2002 edition of Archives, there is a lack of good evidence on which we can base preventive management although thiazide and vitamin D supplements are routinely used by some paediatric rheumatologists, there appears to be only one short term study suggesting this may be beneficial for bone density. The use of bisphosphonates in relation to growth hormone therapy is imperative, however further research is required.

The use of glucocorticoids in children with chronic disease does not involve the use of bisphosphonates in children with chronic disease and low bone density without properly designed studies and satisfactory outcome measures.

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Management of childhood osteoporosis

I read with interest this recent review article that summarises current knowledge about this subject. I have a number of comments that are pertinent to the discussion. As the authors allude to, there is currently a lack of good evidence on which we can base preventive management. Although thiazide and vitamin D supplements are routinely used by some paediatric rheumatologists, there appears to be only one short term study suggesting this may be beneficial for bone density. The use of bisphosphonates in relation to growth hormone therapy is methodologically flawed because neither have accounted for the change in apparent bone density, which will occur in any child who grows better for any reason when assessed by modalities such as dual energy x-ray absorptiometry.

As illustrated by another article in the August 2002 edition of Archives, there is a lack of good evidence on which we can base preventive management although thiazide and vitamin D supplements are routinely used by some paediatric rheumatologists, there appears to be only one short term study suggesting this may be beneficial for bone density. The use of bisphosphonates in relation to growth hormone therapy is imperative, however further research is required.

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Newborn screening for Duchenne muscular dystrophy

Elliman, Dezaute, and Bedford, in their recent leading article on newborn and childhood screening, include reference to newborn screening for Duchenne muscular dystrophy (DMD). They argue that the main value of such a screening programme is to warn parents that future sons may be affected, and support this statement with reference to Jarvinen et al. This paper does not report a newborn screening study but the results of a retrospective study of 23 females in Finland carrier tested for DMD during childhood. However, a newborn screening programme for DMD has been running in Wales since 1990 (1990–8 as a research evaluation and from 1998 health authority funded). During the research period interim evidence was published. More recently the full results of our prospective study have been published. Our evaluation has demonstrated that a newborn screening programme for DMD can be acceptable to both parents and health professionals, providing that a rigorous service delivery protocol is in place and the programme is supported by an effective infrastructure, in particular by paediatric and genetic services.

References


www.archdischild.com
The effect of sanctions on children of Iraq

Sanctions were imposed on the people of Iraq in 1990. Iraqi people are still suffering, especially children. Infant mortality (IM) has increased more than five times. Previously it had decreased from 139 in 1960 to 20 in 1989, which was comparable to developed countries. In 1992 it went up to 111. In 1999, a decade later, IM was still high at 104. The Gulf War and trade sanctions caused a three-fold increase in mortality among Iraqi children under 5 years of age. It has been estimated that more than 46,900 children died between January and August 1991.1

The study of the UN Food and Agricultural Organisation, published in a letter to the BMJ in 1995, concluded that deaths of more than 150,000 children could be attributed to UN sanctions. It also stated that the death rate among children under 5 years in Baghdad had increased fivefold since the war ended in 1991.2 Data for 1994–99 showed that mortality among under 5 years was 131 per 1000 live births, compared with 56 for 1984–9, 9 for 1979–83, and 14 for 1980–88, increased to 21% in 1994.

In a 1995 survey 1.6% of children below −2SD in urban Baghdad was 28% for stunting, 29% for underweight, and 12% for wasting.2,3 Children who were malnourished (>−2SD) was noted among children, 10% for stunting, 7% for underweight, and 3% for wasting. The survey by FAO in the year 2000 indicated the prevalence of wasting in children under 5 years was increased and experienced serious problems with lighting, cleaning, water supply, and sewage. The population had been burdened by a rapid rise in serious infections, nutritional deficiencies among children and pregnant women, and other treatable conditions for which neither drugs nor operations were available.4

Paediatricians have been isolated by the intellectual embargo from international medical community. Physicians who wish to attend international conferences face travel restrictions, like denial of visas to European or American medical journals was a possible link to products—now recognised outside their traditional area of incidence. Furthermore, the initial clinical picture of these disorders can be atypical and incomplete and can change during the clinical course. So, considering the provenance of Galanakis’ series (Greece), we cannot be surprised if some cases had BD or FMF, that will be recognised in the future. Nowadays, with increased diagnostic sensitivity and multi-ethnic societies, periodic fevers are being recognised outside their traditional area of incidence. Close follow up is essential in further years, in these patients. A possible genetic screening for gene causing FMF, HIDS, or immunological assay for HLA B51 could also be useful.

References


Mechanisms of pulmonary hypertension in Bordeletella pertussis

Casano et al describe a case of refractory pulmonary hypertension with severe Bordeletella pertussis infection. Their description of the literature is incomplete. We describe two cases of fatal pulmonary hypertension (PHT) in a series of 13 critically ill infants with B. pertussis. The cases that developed PHT all presented with severe hyperleukocytosis (WCC>100 × 10^9/L) which was unresponsible to all currently available modalities including extracorporeal membrane oxygenation. Hyperleukocytosis was an independent predictor of death when corrected for presentation severity and extracorporeal membrane oxygenation. Hyperleukocytosis was an independent predictor of death when corrected for presentation severity and extracorporeal membrane oxygenation. Hyperleukocytosis was an independent predictor of death when corrected for presentation severity and extracorporeal membrane oxygenation. Hyperleukocytosis was an independent predictor of death when corrected for presentation severity and extracorporeal membrane oxygenation.
hypoxic vasoconstriction. Therefore Dr Casano’s recommendation for the early use of pulmonary vasodilators is unlikely to be sufficient in this context. We are assessing the impact of strategies aimed at reducing lymphocyte numbers and adhesion in addition to standard treatments for pulmonary hypertension.

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References

Authors’ reply

As Peters comments in his letter, we know that hyperleukocytosis has been postulated as a factor for pulmonary hypertension in Pertussis infection, but necessary brevity did not make it possible to report. Nevertheless, our patient never reached these values of leucocytosis; it’s possible, as in many other diseases, that several pathogenic mechanisms contribute to pulmonary hypertension, making a concomitant treatment approach necessary.

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CORRECTIONS

In the paper by Clarkson and Choonara in the December issue of ADC (Arch Dis Child 2002; 87:462–7) the following corrections have been noted:

Results; first sentence: there were 331 deaths with 390 suspected drugs (not 390 and 389 respectively as stated in the paper).
Results; section “Corticosteroids”: the third sentence starting “No details were avail… should be deleted.
Results; section “Non-steroidal anti-inflammatory drugs (NSAIDs)”: the second sentence “All reports for NSAIDs have occurred since 1990” should be deleted.
Discussion; fifth paragraph: the penultimate sentence should be “as recently as 1999 our study found a single fatality” (not 2 reported fatalities).

The following figure should have appeared with the letter by Desai and Babu in the October issue of ADC (Arch Dis Child 2002; 87:357).

Figure 1 Scimitar syndrome. Chest x ray showing a curvilinear density which extends from the right hilum towards the right hemi-diaphragm which represents the anomalous pulmonary vein.
The effect of sanctions on children of Iraq

L Al-Nouri and Q Al-Rahim

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