Correspondence

Intussusception in older children

Sir,

Turner et al.¹ did not mention whether they had considered cystic fibrosis in the differential diagnosis of their patients, or whether they had performed sweat tests. The association is well known in the older child and deserves a passing reference at least. They reported on a group of children who were mainly underweight; did any of them have cystic fibrosis?

Reference


M Super
Department of Clinical Genetics,
Royal Manchester Children’s Hospital,
Pendlebury, Manchester M27 1HA

Mr Rickwood comments:

Although the association is well known it is rare. Generally the association is primarily in the context of a patient with established cystic fibrosis who develops an intussusception. Our two hospitals have a considerable number of patients who are suffering from cystic fibrosis, but the only one who had an intussusception during the period under review was excluded from the series because he had presented at another hospital.

None of the patients we presented was known to have cystic fibrosis, nor has any subsequently been shown to have this disease. We did not perform a sweat test in any patient, and we doubt whether routine screening for cystic fibrosis in older children with intussusceptions would prove a rewarding exercise in the absence of other clinical evidence pointing to the condition. It is true that many of our patients were underweight, but most of those who were greatly so gained weight after the lesion had been dealt with.

A M K Rickwood FRCS
Paediatric Surgical Unit,
Children’s Hospital,
Western Bank,
Sheffield S10 2TH

Prolactin deficiency, obesity, and enlarged testes

Sir,

In the article by Roitman et al.² the investigation of the patient and the way the reported X-linked syndrome of mental deficiency and megalotestes is referred to, imply that the child’s chromosomes were not studied by the method recommended³ for detecting the presence of the fragile X-chromosome syndrome so often described. In this case the association of unusual clinical features could be an indication of a greater than usual defect in the long arm of the X chromosome. Performance of karyotyping in the family may help in our understanding of aspects of the fragile X syndrome should an abnormality be shown.

References

³ Laron and Dr Roitman comment:
We intend to karyotype the whole family to look for the fragile X chromosome. The family is very uncooperative but, nevertheless, we hope to be able to do it in the near future.

We still think that the most outstanding feature of the syndrome is the isolated deficiency of prolactin.

Z Laron and A Roitman
Institute of Paediatric and Adolescent Endocrinology,
Beilinson Medical Centre,
Petah-Tiqva, Israel

Nonaccidental poisoning: the elusive diagnosis

Sir,

We thank Lorber et al.¹ for their comments about our paper,³ and sympathise with their difficulties. In two of our cases the first toxicology test failed to provide a diagnosis and with this in mind we advised repeat testing on the return of symptoms, and discussion of possible poisons with the laboratory taking into account the symptoms and the drugs available to the family.

We are concerned because interest in the diagnostic problem of nonaccidental poisoning, which has been evident from many papers published since our report, may have diverted attention from the equal problem of subsequent management. The sudden hospital admission of the mother at the time of diagnosis was a feature shared by Lorber’s case and two of ours; when the escape route from her own problems provided by the child’s ‘illness’ was about to close she took refuge in ‘illness behaviour’ of her own. The removal of a child under a Place of Safety

A M K Rickwood FRCS
Paediatric Surgical Unit,
Children’s Hospital,
Western Bank,
Sheffield S10 2TH

156
Order, necessary to protect its life, may evoke strong feelings in that child after the break-up of what has previously been a close-knit relationship.

We think the psychiatric aspects of this tragic family disorder differ in important respects from those of classical child abuse and that at present they are a long way from being fully understood. The analogy with the Munchausen syndrome is valid as a description of the family's presentation and of the pathological cry for help, but it cannot explain the disturbed family relationship. We note that in Lorber's account the father was strikingly present through a complete absence of any description at all, and also that the mother was described as 'a psychopathic personality and . . . a pathological liar' without reference to the family system she is, or was, a part of. Our cases have been managed with a psychiatric approach and psychiatric inpatient treatment for parent or child; this approach, rather than punitive legal action, seems to have been successful for an earlier patient reported from Sheffield.

Much might be learnt from long-term follow-up of the many cases now known to paediatricians throughout the country, and we would like to co-operate with other colleagues.

References

3 Szur R. Psychotherapy with a child who has been poisoned. Child Abuse and Neglect 1979; 3: 505–8.

D W ROGERS AND A BENTOVIM
The Hospital for Sick Children, Great Ormond Street, London WC1N 3JH
J H TRIPP
Royal Devon and Exeter Hospital, Barrack Road, Wonford, Exeter EX2 5DW, Devon

Professor Lorber comments:

I agree with Rogers et al. that a psychiatric approach in the management of the mother was very important; a psychiatric colleague was directly concerned early on in the case and she was also an important part of the team dealing with the mother. However, the future risk to the child was so great that neither the social services nor the doctors felt that it was safe to leave the mother entirely in the hands of professionals without taking the matter to Court and asking for its decision.

J LORBER
Department of Paediatrics, Children's Hospital, Western Bank, Sheffield S10 2TH

Hyperglycaemia in Lomotil poisoning

Sir,

I should like to draw your attention to the hitherto unreported association of transient hyperglycaemia with Lomotil (diphenoxylate and atropine) poisoning.

An 11-month-old boy, who had previously been well, was admitted as an emergency. He was comatose, had shallow respirations, constricted pupils, and peripheral cyanosis. He had been put to bed 5 hours earlier and had not drunk or eaten since that time. Two consecutive Dextrostix recordings, carried out immediately on admission, read '>13.9 mmol/l' (>250 mg/100 ml). His parents then admitted the possibility of Lomotil ingestion and he was given 0.1 mg naloxone with immediate clinical effect. A laboratory estimation of plasma glucose on an initial specimen, taken on arrival, was 30.8 mmol/l (355 mg/100 ml) with urea 4.5 mmol/l (27 mg/100 ml), sodium 135 mmol/l, potassium 4.3 mmol/l, total CO2 20.5 mmol/l, and chloride 102 mmol/l. Three hours later (6 to 8 hours after ingestion) this had fallen to 5 mmol/l (90 mg/100 ml) with similar electrolytes. Urine, first obtained 8 to 10 hours after ingestion, showed 0.25 to 0.5% sugar on Clinitest testing. Subsequent testing of blood and urine showed neither hyperglycaemia nor glycosuria. The possibility that Lomotil might interfere with the assay of plasma glucose was investigated and excluded.

A search of the literature has not revealed a similar reported occurrence, and neither of the active components of Lomotil (diphenoxylate or atropine) has singly been associated with hyperglycaemia. It is difficult to understand the causative mechanism, although it may be suggested that the relative preponderance of sympathetic activity in atropine poisoning is contributory.

I suggest that Lomotil in overdosage should be considered in the unconscious child with hyperglycaemia in whom the clinical and biochemical findings are not those of diabetic ketoacidosis.

I thank Dr D B Horn for carrying out the biochemical assays and Dr A J Keay for permission to report this case.

NEENA MODI
Medical Paediatric Unit, Western General Hospital, Crewe Road, Edinburgh EH4 2XU

Clostridium difficile-associated colitis

Sir,

My 5-month-old son received a 5-day course of oral phenoxymethyl penicillin for pharyngitis. About one week later he passed blood-stained motions for 2 days, but appeared well. For the next 5 months he had frequent loose bowel actions but no more blood was seen and he remained well. At this stage a stool was tested and found to contain toxin neutralisable by Clostridium sordelli antitoxin, suggesting the presence of Clostridium difficile infection. Treatment with oral metronidazole caused the disappearance of the toxin within 24 hours and the loose frequent bowel actions ceased. Toxin was absent from
Nonaccidental poisoning: the elusive diagnosis.

D W Rogers, A Bentovim and J H Tripp

*Arch Dis Child* 1981 56: 156-157
doi: 10.1136/adc.56.2.156-d

Updated information and services can be found at:
[http://adc.bmj.com/content/56/2/156.5.citation](http://adc.bmj.com/content/56/2/156.5.citation)

**Email alerting service**
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
[http://group.bmj.com/group/rights-licensing/permissions](http://group.bmj.com/group/rights-licensing/permissions)

To order reprints go to:
[http://journals.bmj.com/cgi/reprintform](http://journals.bmj.com/cgi/reprintform)

To subscribe to BMJ go to:
[http://group.bmj.com/subscribe/](http://group.bmj.com/subscribe/)