LETTERS TO THE EDITOR

Rapid responses
If you have a burning desire to respond to a paper published in ADC or F&N, why not make use of our “rapid response” option?

Log on to our website (www.archdischild.com), find the paper that interests you, click on “full text” and send your response by email by clicking on “submit a response”.

Providing it isn’t libellous or obscene, it will be posted within seven days. You can retrieve it by clicking on “read rapid responses” on our homepage.

The editors will decide, as before, whether to also publish it in a future paper issue.

Iron deficiency anaemia, helicobacter pylori infection and delayed pubertal growth
We read with great interest the paper by Choe and colleagues which investigated a possible relationship between Helicobacter pylori infection, iron deficiency anaemia (IDA), and subnormal growth at puberty.1 We read with great interest the paper by Choe and colleagues which investigated a possible relationship between Helicobacter pylori infection, iron deficiency anaemia (IDA), and subnormal growth at puberty.1

We wish to respond to Dr Morley’s letter as he has raised some important issues concerning our paper on iron status and development.1 Firstly, we wish to respond to two matters not discussed in Dr Morley’s letter.

Firstly, when the paper was published, there was prominent media coverage, erroneously concluding that our paper rendered the British Medical Journal (BMJ) obsolete. However, we believe that these results need some consideration.

H pylori infection may cause IDA in different ways: (a) the bacteria can cause a decrease in the gastric juice of the concentration of ascorbic acid, which is the best promoter of non-heme iron absorption;2 (b) H pylori may increase iron demand because iron is an essential bacterial growth factor;3 (c) H pylori contains a 19.6 kDa protein resembling ferritin with a binding activity for heme iron in erythrocytes;4 (d) acute or chronic blood loss and IDA are associated with typical H pylori related gastroduodenal lesions. In Choe’s study, the treatment of H pylori infection with antibiotics (but also with proton pump inhibitors) was probably linked to a more rapid response to oral iron replacement because of the effects of proton pump inhibitors in healing some of the lesions.

However, IDA, failure to thrive, and delayed pubertal growth are important features of subclinical and silent coeliac disease. Therefore, coeliac disease should be suspected in children or adolescents with these signs, especially when IDA is resistant to oral iron replacement.

In a recent study,1 IDA seemed to be the most frequent extra-intestinal marker of coeliac disease in both children and adults, followed by short stature for children. Thus, it would be advisable to screen paediatric and juvenile population with IDA, failure to thrive, or delayed pubertal growth for coeliac related antibodies (anti-endomysial, anti-transglutaminase, antigliadin) in order to identify coeliac patients who need lifelong gluten withdrawal for recovery of iron, and improved metabolism and growth.

Finally, it is reasonable that H pylori infection may make iron deficiency worse in coeliac patients and subsequently impair growth in children who need a large amount of iron from this essential element.


Iron status and development

EDITOR,—You have recently published a response by Dr Morley to Dr Stevens concerning our paper on iron status and development.1 We wish to raise two matters not discussed in Dr Morley’s letter.

Firstly, when the paper was published, there was prominent media coverage, erroneously concluding that our paper rendered the BMJ obsolete. However, we believe that these results need some consideration.

H pylori infection may cause IDA in different ways: (a) the bacteria can cause a decrease in the gastric juice of the concentration of ascorbic acid, which is the best promoter of non-heme iron absorption;2 (b) H pylori may increase iron demand because iron is an essential bacterial growth factor;3 (c) H pylori contains a 19.6 kDa protein resembling ferritin with a binding activity for heme iron in erythrocytes;4 (d) acute or chronic blood loss and IDA are associated with typical H pylori related gastroduodenal lesions. In Choe’s study, the treatment of H pylori infection with antibiotics (but also with proton pump inhibitors) was probably linked to a more rapid response to oral iron replacement because of the effects of proton pump inhibitors in healing some of the lesions.

However, IDA, failure to thrive, and delayed pubertal growth are important features of subclinical and silent coeliac disease. Therefore, coeliac disease should be suspected in children or adolescents with these signs, especially when IDA is resistant to oral iron replacement.

In a recent study,1 IDA seemed to be the most frequent extra-intestinal marker of coeliac disease in both children and adults, followed by short stature for children. Thus, it would be advisable to screen paediatric and juvenile population with IDA, failure to


Iron status and development

EDITOR,—You have recently published a response by Dr Morley to Dr Stevens concerning our paper on iron status and development.1 We wish to raise two matters not discussed in Dr Morley’s letter.

Firstly, when the paper was published, there was prominent media coverage, erroneously concluding that our paper rendered the BMJ obsolete. However, we believe that these results need some consideration.

H pylori infection may cause IDA in different ways: (a) the bacteria can cause a decrease in the gastric juice of the concentration of ascorbic acid, which is the best promoter of non-heme iron absorption;2 (b) H pylori may increase iron demand because iron is an essential bacterial growth factor;3 (c) H pylori contains a 19.6 kDa protein resembling ferritin with a binding activity for heme iron in erythrocytes;4 (d) acute or chronic blood loss and IDA are associated with typical H pylori related gastroduodenal lesions. In Choe’s study, the treatment of H pylori infection with antibiotics (but also with proton pump inhibitors) was probably linked to a more rapid response to oral iron replacement because of the effects of proton pump inhibitors in healing some of the lesions.

However, IDA, failure to thrive, and delayed pubertal growth are important features of subclinical and silent coeliac disease. Therefore, coeliac disease should be suspected in children or adolescents with these signs, especially when IDA is resistant to oral iron replacement.

In a recent study,1 IDA seemed to be the most frequent extra-intestinal marker of coeliac disease in both children and adults, followed by short stature for children. Thus, it would be advisable to screen paediatric and juvenile population with IDA, failure to


Iron status and development

EDITOR,—You have recently published a response by Dr Morley to Dr Stevens concerning our paper on iron status and development.1 We wish to raise two matters not discussed in Dr Morley’s letter.

Firstly, when the paper was published, there was prominent media coverage, erroneously concluding that our paper rendered the BMJ obsolete. However, we believe that these results need some consideration.

H pylori infection may cause IDA in different ways: (a) the bacteria can cause a decrease in the gastric juice of the concentration of ascorbic acid, which is the best promoter of non-heme iron absorption;2 (b) H pylori may increase iron demand because iron is an essential bacterial growth factor;3 (c) H pylori contains a 19.6 kDa protein resembling ferritin with a binding activity for heme iron in erythrocytes;4 (d) acute or chronic blood loss and IDA are associated with typical H pylori related gastroduodenal lesions. In Choe’s study, the treatment of H pylori infection with antibiotics (but also with proton pump inhibitors) was probably linked to a more rapid response to oral iron replacement because of the effects of proton pump inhibitors in healing some of the lesions.

However, IDA, failure to thrive, and delayed pubertal growth are important features of subclinical and silent coeliac disease. Therefore, coeliac disease should be suspected in children or adolescents with these signs, especially when IDA is resistant to oral iron replacement.

In a recent study,1 IDA seemed to be the most frequent extra-intestinal marker of coeliac disease in both children and adults, followed by short stature for children. Thus, it would be advisable to screen paediatric and juvenile population with IDA, failure to

Consider absolute risks in SIDS prevention

EDITOR,—The demonstration by Blair et al of an association between poor postnatal growth and an increased risk of sudden infant death syndrome (SIDS),1 is a useful addition to our understanding of the aetiology of this condition. It is unfortunate that the conclusion in the abstract that “Poor postnatal weight gain was independently associated with an increased risk of SIDS” was definitely associated at the routine six week assessment” goes beyond the data presented.

It can be estimated from the data in this study that the overall risk of SIDS was 0.77/1000 live births. The risk in babies with birth weights greater than the 15th centile, the group in whom the relation with postnatal growth was detected, was 0.68/1000. Given DRS Blair, Fleming, and Platt comment:

EDITOR,—The demonstration by Blair et al of an association between poor postnatal growth and an increased risk of sudden infant death syndrome (SIDS),1 is a useful addition to our understanding of the aetiology of this condition. It is unfortunate that the conclusion in the abstract that “Poor postnatal weight gain was independently associated with an increased risk of SIDS” was definitely associated at the routine six week assessment” goes beyond the data presented.

It can be estimated from the data in this study that the overall risk of SIDS was 0.77/1000 live births. The risk in babies with birth weights greater than the 15th centile, the group in whom the relation with postnatal growth was detected, was 0.68/1000. Given


Are we requesting too many DMSA scans?

EDITOR,—The recent article by Christian et al highlights the value of clinical features in assessing the risk of renal scarring and therefore the need for dimercaptosuccinic acid (DMSA) scan after urinary tract infection (UTI).2 We recently performed a retrospective study to assess the recording of fever, malaise, recurrent UTI, and urine culture results in children investigated with DMSA scan after UTI. Between April 1996 and October 1997 there were 171 DMSA scans in our hospital that fitted these criteria; 30 case notes could not be traced. There were 105 girls (74%) and 36 boys. Age when UTI was diagnosed ranged from 9 days to 15.3 years (mean 4.2 years, SD 3.2).

Urine culture results were: UTI (>10^5 cfu/ml) in 82 cases (58%), contaminant (<10^5 cfu/ml) in 27 cases (19%), no growth in 21 cases (15%), and no urine culture in 11 cases (8%). There were 17 (12%) cases of definite or probable renal scar, none of which followed a sterile or contaminated urine culture. Of the 141 case notes, there was no mention of fever in 48 (34%), and no mention of malaise in 76 (54%). In 69 case notes reviewed there was no mention of previous history of UTI in 14 (20%) cases. Of those with a history of fever, 19% (10/53) had an abnormal DMSA scan compared to 10% (4/40) in those without fever. Eighteen percent (9/50) of those unwell at the time of UTI had an abnormal scan compared to 13% (2/13) of those not ill.

These data suggest that in a substantial proportion of cases, the decision to request a DMSA scan is apparently not influenced by salient clinical features and urine culture results. In this series, 19% of those that children with sterile or contaminated urine cultures should not have had a DMSA scan. This would have saved the cost and burden of 48 scans, 34% of this series, over an 18 month period. It is likely that these findings are peculiar to our district.

A H SACKEY
Leighton Hospital, Middlesbrough Road, Cresswell Cross, DH3 95/3. Crown copyright, 1995:4.


