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Campylobacter pylori Gastritis and Peptic Ulcer in Children

Sir.—We read with great interest the article by Kilbridge et al[1] in the December 1988 issue of *AJDC* on *Campylobacter pylori* (CP)–associated gastritis and peptic ulcer disease in children, which are becoming growing problems in pediatric gastroenterology. In fact, according to some authors CP is present in antral biopsies of up to 32%[2,3] of children with recurrent abdominal pain severe enough to require gastroscopy. Even if the prevalence of CP infection is known to increase with age and, therefore, it is expected to be lower in the pediatric population, in children with primary gastritis the percentage of CP infection is as high as 60% to 80%[4,5] and quite similar to the rate found in adults.

In a retrospective study we carried out on all gastroscopies performed in our center from 1983 to 1987, only 58 patients (8.6%) with primary gastritis were found, but in 70.7% of them CP was identified when the histological slides were reviewed. We feel that this figure, like the 55% reported by Kilbridge et al, underestimates the real prevalence of CP infection. In fact, we used to take biopsy specimens only from those gastric antra showing some changes at endoscopy, but we are now aware, as Kilbridge et al pointed out, that CP can colonize a normal-appearing antrum. The low figures reported could be due to the fact that their study was retrospective. We would like to emphasize the high prevalence of the CP infection in children that, in a prospective study we are presently conducting, is reaching more than 90% in children with primary gastritis or peptic ulcer disease.

Two points in the article by Kilbridge et al are of particular interest:

1. There was a predominance of chronic infiltrate antral CP gastritis in children, with less diffuse acute inflammatory component than in adults. Their hypothesis that this represents an earlier phase of infection is attractive but does not seem to be confirmed by some of our observations.[6] In fact, we too observed a higher prevalence of chronic gastritis in children with CP infection (26 cases vs 16 with acute inflammatory infiltration), but after amoxicillin treatment, which cleared the infection in 85% of the children and healed the gastritis in 67%, we observed a recurrence in 73% of the patients 3 months after stopping the treatment. We think that these recurrences could be considered as an early phase of infection since 3 months earlier gastritis was not present. Sixty percent of these children who suffered a relapse showed an acute gastritis.

2. There was a high relapse rate (80%) of duodenal ulcer in patients with CP gastritis. This finding may indicate that CP presence in antral mucosa portends an unfavorable prognosis. In a long-term follow-up study of children with peptic ulcer treated with ranitidine, we observed a relapse rate of 47% of 31 patients; CP was present in 85% of children who had a relapse but only in 29% of those who did not.[7]

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Sir.—Although a rapidly expanding body of literature provides much information concerning the prevalence and the significance of CP in adults,[1] there is little information as to whether the association of this organism with histologic gastritis represents in childhood a causal relationship or merely an association. After reading the article by Kilbridge et al, we were stimulated to report in brief our preliminary data to address this question.

Recently we initiated a prospective study in which we attempted to identify CP in antral biopsies obtained from children undergoing upper gastrointestinal tract endoscopy for chronic abdominal pain, hematemesis, vomiting, and unexplained iron-deficiency anemia. To date, complete evaluation has been obtained on 24 children.

In 11 of the 24 antral biopsies, the light microscopy showed evidence of chronic gastritis, which was characterized by a diffuse inflammatory cell infiltration of the lamina propria consisting of plasma cells and lymphocytes. *Campylobacter pylori* was detected by means of histological stains and culture in 10 of the 11 children with chronic gastritis. None of the 13 patients with histologically normal antral mucosa had CP demonstrated on their antral biopsy specimens. The presence of the bacterium was associated with mild as well as moderate to severe inflammatory changes of the antral mucosa. The association of CP even with mild gastritis provides further support that this organism represents an inciting factor for antral gastritis in children.

Surprisingly, the antral biopsies did not indicate the presence of CP as a marker of "active" histological gastritis. In fact, the histological appearances were never associated with signs of activity.[2] Kilbridge et al suggested that the "inactive" chronic inflammation observed in the younger subjects in their study may represent an earlier phase of infection.

In our study, patients with CP-associated gastritis were older (mean age, 9.8 years) than those who did not present this condition (mean age, 6.7 years). As previously described in adults,[3] it is possible that "active" chronic gastritis due to CP may be limited also in children to a patchy distribution and may not be present at the site of detection of the organism. Finally, six patients who received a 4-week course of monotherapy with amoxicillin trihydrate (50 mg/kg per day in three divided doses) exhibited eradication of the organism, which was associated with a marked improvement or complete resolution of the inflammatory changes of the gastric mucosa. After 6 to 18 months of cessation of therapy, the patients continue to be asymptomatic.

As Kilbridge et al point out, additional prospective long-term follow-up studies may help to clarify further the role of CP in the pathogenesis of antral gastritis in children.

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In Reply.—Chiesa et al and Oderda et al agree with us regarding the high prevalence of CP infection and its relationship to antral gastritis and duodenal ulcer.

The highly effective results of antimicrobial therapy reported by Chiesa et al are interesting. To date, we have en unsuccessful in eradicating CP with amoxicillin monotherapy. Our results are in agreement with recently reported adult studies by Rauws et al,[1] as well as pediatric studies by De Giacomo et al.[2] The latter group had no success in eradicating CP or improving the underlying gastritis in their pediatric patients. The experience cited above by Oderda et al also indicates failure to achieve permanent eradication of the organism with ampicillin. We look forward to the publication of the study done by Chiesa et al. Perhaps we may be able to determine why we and others have not been successful in our efforts to eradicate CP. We agree with Dr Oderda and his colleagues that children with CP gastritis and duodenal ulcer are very likely to suffer ulcer relapse if they are treated with standard H2 antagonist therapy alone and the CP infection is not eradicated.

Both Chiesa et al and Oderda et al have additional observations regarding the relative roles of acute and chronic inflammation in CP gastritis. In pathologic parlance, the term *active gastritis* as used by Chiesa et al means acute inflammatory cells are present in the tissue. They did not observe active gastritis in any of their CP infections; all the gastritis they observed was histologically chronic inflammation. Their experience again differs from ours and from the other published series of CP gastritis in children,[3-5] where both chronic and acute inflammation are described. Oderda and colleagues also observed, as we did, that both chronic and acute inflammation are present, with chronic gastritis predominating. Oderda et al suspect that acute inflammation is present from the start of CP infection in some patients, since they observed acute gastritis within 3 months of reinfection with CP.

European gastroenterologists, such as Chiesa and Oderda, appear to be more interested than their American colleagues in CP and more convinced of its pathogenic role in upper gastrointestinal tract disease.

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Hyperuricosuria and Microhematuria in Childhood

Sir.—Microhematuria is a common finding in pediatrics that frequently requires multiple diagnostic procedures[1] to determine its cause, which in many cases remains uncertain.

Among the multiple possible causes, Stapleton et al[2] pointed out the role hypercalciuria would play in the pathogenesis of certain types of microhematuria. We have found an association between hyperuricosuria and microhematuria in five otherwise-healthy children. We think that a pathogenic mechanism is operative that is similar to the one reported by Stapleton et al[2] for microhematuria secondary to hypercalciuria. The small number of patients tested only suggests this possibility and the pilot experience reported herein will require further investigation.

Patients and Methods.—Five children (2 boys and 3 girls), whose ages ranged from 4 to 9 years, were selected from a total of 139 patients with microhematuria because they presented abnormally high levels of uricosuria with no other underlying cause.

In one of the children the onset of the disorder was accompanied by gross hematuria and colic pain (with no calculi expulsion) and microhematuria that persisted after this episode. In the remainder of the patients the condition was asymptomatic and the persistence of microhematuria was assessed by examination of at least five consecutive urine specimens.

Renal function (serum urea and creatinine levels and creatinine clearance) was normal in all patients. Results of coagulation studies, 24-hour calciuria measurement, intravenous pyelography, and cystourethrography were all normal. Diseases causing hypouricemia were eliminated and the possibility of exposure to drugs or heavy metals was also considered.

Microhematuria was defined by the presence of more than 15 red blood cells per high-power field in fresh urinary sediment