Selected Summaries

Helicobacter Pylori Infection: A Cause of Growth Delay in Children

[Perri F, Pastore M, *Leandro G, Clemente R, Ghoos Y, Peeters M, et al. Helicobacter pylori infection and growth delay in older children Arch Dis Child 1997; 77:46-49].*

Helicobacter pylori infection is a common chronic infection acquired early in life and like other chronic diseases may impair growth. Hence the aim of this study was to evaluate the prevalence of *H. pylori* infection in healthy Italian children and to look for differences in height between infected and non infected children. Two hundred and sixteen children aged 3 to 14 years were tested for *H. pylori* infection by ISC-urea breath test and centile values for height were calculated. Composite indices for socioeconomic class and household crowding were also determined.

Forty nine of 216 children (22.7%) were *H. pylori* positive. No difference in sex between *H. pylori* positive and negative subjects was found. The prevalence rate of infection increased with age, from zero per cent in the youngest group (3-4 years) to 33% in the oldest (13-14 years). Eight of 49 *H. pylori* positive children (16.3%) were below the 25th centile for height, compared with 13 of 167 *H. pylori* negative children (7.8%). This difference became significant in children aged 8.5-14 years; in this group (n=127), eight of 31 infected children (25.8%) were below the 25th centile for height, compared with eight of 96 non-infected children (8.3%). A significant correlation was found between socioeconomic conditions, household crowding and *H. pylori* status. By using stepwise logistic regression, only the centile value for height was significantly related to *H. pylori* status in older children.

It was concluded that *H. pylori* infection was associated with growth delay in older children, poor socioeconomic conditions and household overcrowding. This finding is consistent with hypothesis that H. pylori infection is one of the environmental factors capable of affecting growth.

Comments

The overall prevalence of *H. pylori* infection is higher in developing countries like India and Africa(l,2) compared to 23% in the present study. Several studies have shown poor socioeconomic conditions associated with overcrowding and inadequate hygiene at home to be important risk factors for *H. pylori* infection(3).

The mechnaisms by which *H. pylori* infection might lead to short stature are largely unknown. Of the various possible explanations, dyspeptic symptoms leading to low nutritional intake does not seem to play a significant role as far as this study is concerned since majority of children did not suffer from dyspeptic symptoms. Moreover, no difference in the centile value for weight or in body surface area was detected among infected and non infected children. It is also presumed that growth delay may be due to poor socioeconomic status and malnutrition but logistic regressions analysis revealed that height was not influenced by socioeconomic status or overcrowding. Hence, though poor socioeconomic conditions favor early acquisition of *H. pylori* infection, the mechanism of

short stature is independent of these factors. Another possible explanation is based on the assumption that long standing infection induces low grade chronic gastric inflammation and release of cytokines like interleukin 8(4) which would in turn affect growth(5). However, there is no confirmatory data available for this mechanism.

This study did not demonstrate difference in growth velocity among infected and non infected children but demonstrated only a difference in height which can be influenced by mid parental height or genetic factors in the children studied. Also change in growth velocity before and after eradication of *H. pylori* infection needs to be demonstrated to confirm the role of *H. pylori* infection in growth delay.

Findings of the present study have two implications. Firstly, that *H. pylori* infection could be one of the environmental causes of growth delay. A decline in the incidence of *H. pylori* infection related to general improvement in socioeconomic status could be responsible in part for the trend towards increased height in developed countries.

Secondly, therapeutic interventions against *H. pylori* could be recommended not only to prevent ulcer disease but also to avoid interference with growth. Hence a well designed study is recommended in which growth velocity rather than height is evaluated either before or after *H. pylori* eradication. Also investigations are needed to discover whether therapeutic intervention in otherwise healthy *H. pylori* infected children would avoid restricted or delayed growth.

Anju Aggarwal,
Department of Pediatrics,
Kalaivati Saran Children's Hospital
and Lady Hardinge Medical College,
New Delhi 110 001.

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Corticosteroids in Tuberculous Meningitis

[Schoeman JF, Van Zyl LE, Laubscher JA, Donald PR. Effect of corticosteroids on intmcranial pressure, computed tomographic findings, and clinical outcome in young children with tuberculous meningitis. Pediatrics 1997; 99: 226-231].

Corticosteroid therapy, although controversial, is widely used in the routine management of tuberculous meningitis (TBM). This study attempted to establish the exact mechanism of action of high dose prednisone in children with moderate to severe TBM, along with its effect on the clinical outcome. In the study, 141 consecutive children with TBM (Stages II and III) were included, and were randomly allocated to a steroid or non-steroid treatment group. Seventy patients received steroids while 71 children were in the non-steroid group. The first 16 patients in the steroid group were given prednisone in a dose of 2 mg/kg/day and the remaining 54 patients received it in a dose of 4 mg/kg/day for the first month of treatment. Antituberculous treatment consisted of isoniazid, rifampicin, ethionamide and pyrazinamide and all these drugs were given for 6 months. The clinical outcome was assessed after completion of 6 months of antituberculous treatment; parameters included intelligence, vision, motor function and mortality. Continuous lumbar cerebrospinal fluid (CSF) pressure monitoring, along with contrast CT scans in all patients were performed at admission and were repeated regularly (CSF pressure at weekly intervals, CT scans were repeated after 1 month and 6 months). The administration of corticosteroid significantly improved the survival and intellectual outcome of children with TBM. Four patients in the second group and 13 in non-steroid group died before completing 6 months of antituberculous therapy. No significant difference was found between the two treatment groups with regard to motor deficit, blindness or deafness. The clinical outcome of children receiving high-and low-dose prednisone did not differ significantly. No significant difference in intracranial pressure or the degree of hydrocephalus was noted between the two groups after first month of treatment. The effect of steroids on intracranial pressure could be evaluated in only 116 children with communicating hydrocephalus, because children with non-communicating hydrocephalus underwent ventriculo-peritoneal shunting after admission. Neither rate of occurrence nor size of infarct (as demonstrated by computed tomography) differed significantly at admission and subsequent follow up. A high incidence (8%) of tuberculoma was observed during the first month of treatment. Both the response of the tuberculoma to treatment and the incidence of delayed occurrence of tuberculomas were significantly improved after steroid therapy. Enhanced resolution of basal exudate was also demonstrated by serial CT scans.

Comments

Corticosteroids, in past, have been reported to accelerate clinical improvement and return of CSF glucose and protein levels to normal. One of the major concerns about the widespread use of corticosteroids in the management of TBM is the possibility that reduction in meningeal inflammation that accompanies their use may decrease the CSF penetration of anti-tuberculous drugs(l). A review(2) supported their use in patients with Stage III disease. A Chinese study(3) supported this view, as lesser number of steroid treated Stage-Ill patients died as compared to those who were not on steroids. Survival in Stage-II

TABLE I— *Staging of Tuberculous Meningitis Developed by British Medical Research Council(6).*

Stage I:	Presence of non-specific symptoms without alteration of consciousness.
Stage II:	Disturbed consciousness without coma or delirium, and minor focal neurological signs.
Stage III:	Presence of stupor or coma, severe neurological deficit, seizures or abnormal movements.

Adapted from Reference 6.

group was also better. The study under consideration further confirmed that oral administration of prednisone improves the chances of survival in Stage-III children of TBM. Similarly, a study from Egypt(4) of 280 children with TBM showed that parenteral administration of dexamethasone improved overall survival and significantly reduced the number of permanent sequelae. However, another large study observed that corticosteroids seemed to reduce the number of deaths during acute phase of illness, but they increased the risk of relapse and did not significantly affect overall mortality in children with TBM(5).

Other potential indications for corticosteroids use include tuberculous encephalopathy in children, raised intracranial pressure, signs of spinal arachnoiditis, and evidence of cerebral vasculitis. However, sufficient data to support use of corticosteroids in these conditions are not available. The current study tried to answer this issue also and observed that corticosteroids did help in early resolution of basal exudates and tuberculomas. Corticosteroids did not affect intracranial pressure or the incidence of basal ganglionic infarction significantly.

In conclusion, Stage-III disease *(Table I)* is a definite indication for using corticosteroid and role for other indications need further evaluation.

B. Karak,
R.K. Garg,
Division of Neurology,
Institute of Medical Sciences,
Banaras Hindu University,
Varanasi 221 005.

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