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LETTERS to the EDITOR

Necrotising enterocolitis—a community-acquired infectious disease?

SIR,—Necrotising enterocolitis (NEC) is an important acquired problem of neonatal intensive care arising during the first few weeks of life, often after premature birth. The aetiology is unknown but epidemiological observations suggest roles for infection and enteric feeding.[1] Clustering of cases has led to the assumption of a hitherto unidentified hospital-specific, ward-acquired infectious agent transmitted from patient to patient, and one epidemic of NEC was successfully tackled by standard measures for infectious disease control.[2] Breast milk protects against NEC,[3] and the disease can be prevented by supplementing formula feeding with an IgA-rich preparation from pooled human serum.[4]

From March to August, 1991, we observed, after several years of single cases only, an epidemic of 8 cases of NEC in the Children's Hospital, University of Würzburg. The criteria for a diagnosis of NEC were bloody stools, systemic manifestations suggestive of neonatal sepsis, and pneumatosis intestinalis on abdominal X-rays. Histological features in 2 patients undergoing surgery were compatible with NEC. No infectious agent could be implicated by blood or stool culture or serology. We were much surprised to learn that, over the same period, 4 cases of NEC had occurred in the other, independent children's hospital serving the same catchment area (a regional capital and surrounding rural communities). In all 4 patients the diagnosis was confirmed by histology of surgical or necropsy specimens.

The simultaneous occurrence of epidemic NEC in two children's hospitals serving the same population might be fortuitous but we favour a different view. When NEC appeared in both hospitals an unidentified pathogen might have been circulating in the catchment area by faecal-oral transmission. In adults and children beyond neonatal age the infection ran a symptomless course. However, in susceptible hosts such as premature babies the infection, transmitted by symptomless care-givers and parents feeding the child, produced NEC, probably in conjunction with other pathogenetic factors. When the population in the catchment area had acquired protective immunity to the hypothetical pathogen the epidemic ended in town and, subsequently, on the wards.

The hypothesis of a ubiquitous (as opposed to nosocomial) infectious agent with a central role in the pathogenesis of NEC is consistent with outbreaks recorded here and with previous information [2-4] and with a report on an epidemic of NEC in two Paris hospitals.[5]

Besides infection control measures we suggest that all care-givers should be regarded as potential transmitters of the disease and be supervised and instructed accordingly.

H-I. H. thanks Dr Mike Noble (University of British Columbia, Vancouver, Canada) for his comments.

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Sunbathing and gallstones

SIR,—The development of cholelithiasis is a multifactorial process. In their interesting article (Nov 2, p 1117), Professor Paumgartner and Professor Sauerbruch state that, in industrialised countries today, 80% of gallstones consist mainly of cholesterol. They focus on factors influencing the solubility of cholesterol in bile but do not mention that almost all cholesterol stones contain a central pigmented nucleus with radial or lamellar pigmented bands alternating with layers of crystalline cholesterol.[1] This pigmented centre may be critical in cholesterol gallstone pathophysiology.[2-4] The pigment centre seems to contain a high-molecular-weight complex,[4] very probably a polymer, which does not seem to have exactly the same composition as the polymer network in pigment gallstones.[3,5] In the pigment stones, bilirubin and its degradation products represent an important part of the pigment.[6,7] The factors responsible for initiating the polymerisation remain obscure.

The identification of reactive indole metabolites of the skin pigment cells led to a new hypothesis on the pathogenesis of some gallstones.[8] This suggests that the increased concentration of these substances in bile triggers the polymerisation. The activation of the skin pigmentary system by ultraviolet light might increase their bile concentration—and thus the risk of gallstone formation. This holds especially true for people with light complexion, whose storage capacity for melanin seems to be limited.

In our case-control study we found that a positive attitude to sunbathing was associated with a higher risk of gallstones, there being a significant dose-effect relation.[9] In the 206 white-skinned individuals, a positive attitude was associated with twice the risk of cholelithiasis of those with a negative attitude to sunbathing (relative risk 2·1 [95% confidence interval 1·0–4·5]). When the effect of sun attitude was considered for individual skin pigmentation types, the association was almost entirely restricted to people who always burn after long sunbathing (skin type I). In that group, the relative risk was 25·6 (95% CI 3·2–205) for a positive attitude compared with a negative one.

The received wisdom that gallstone patients are often of fair complexion is no longer thought valid but our findings suggest that pigmentation in skin and pigment in gallstones may have something in common.

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