Discussion

The prevalence of rheumatic heart disease among schoolchildren has previously been assessed in various countries.^{3 5} In 1970 Morton *et al* found a relatively high prevalence of rheumatic heart disease among the schoolchildren in the San Luis Valley, which is a region of low socioeconomic level.⁵

Our higher rate for rheumatic heart disease in children from a low socioeconomic group is consistent with these reports.^{4 5} The overall incidence, which was 0.6% in our series, is also considerably higher than in developed countries.

The second part of our study shows a decline in the morbidity rate of rheumatic heart disease in the last decade. As the children from only one school from the low socioeconomic group were screened we compared the result with the rate found in this school alone, in 1976. Although the number of children may seem inadequate a parallel decline noted in the number of patients with rheumatic heart disease seen in our cardiology clinics seems to support our thesis. The reasons for the decreasing incidence in rheumatic fever and rheumatic heart disease has not been completely understood. Markowitz agrees that widely disseminated programmes for the use of penicillin play an important part but insists that other unknown factors could be important.³ Gordis has concluded that changes in the occurrence of rheumatic fever cannot be fully accounted for just by medical care and antibiotic use, but that we lack adequate information regarding other possible cofactors and that although group A streptococcus is the critical aetiologic agent of rheumatic fever, the disease is probably multifactorial in origin.⁶ In Turkey we believe that improving medical care and greater access to health care have also appreciably influenced the documented decline. Further studies from developing countries might serve to enlighten the subject.

References

- ¹ Markowitz M. Eradiction of rheumatic fever. *Circulation* 1970; 41:1077–84.
- ² Besterman E. The changing face of acute rheumatic fever. Br Heart J 1970;32:579-82.
- ³ Markowitz M. The decline of rheumatic fever: role of medical intervention. J Pediatr 1985;106:545–9.
- ⁴ Markowitz M, Gordis L, eds. *Rheumatic fever. Major problems in clinical pediatrics*. Philadelphia: WB Saunders, 1972.
- ⁵ Morton WE, Warner AL, Well SV. Rheumatic heart disease epidemiology. III. The San Luis Valley prevalence study. *Circulation* 1970;**41**:773–81.
- ⁶ Gordis L. The virtual disappearance of rheumatic fever in the United States. *Circulation* 1985;72:1155–62.

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Endemic bladder stones in Nepal

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SUMMARY Bladder stones account for a large proportion of surgical admissions in many developing countries. We report in detail the clinical features and risk factors of one such case, a 5 year old Nepali boy, and propose the theory that a low calcium intake, by causing hypocalciuria, predisposes to bladder stone formation.

Bladder stones are endemic in an area stretching as a belt from Turkey and Iran across India to Thailand and beyond to Indonesia and Papua New Guinea. However, they are rare in Africa.¹ They are also rare in the developed world, although this has not always been the case: they were common in England before the turn of the century when itinerant lithotomists flourished especially in 'stone districts', where the prevalence was unusually high, such as Norfolk.

In these areas of endemic bladder stones, boys are affected more than girls with a peak incidence at 5 years of age and the stones, which are rarely present in the kidneys, usually consist of uric acid, ammonium acid urate, or oxalate.¹ This is in contrast with the stones found in people from developed countries which are rare, occur in adults rather than children with a roughly equal sex distribution, are more common in the upper urinary tract, and usually consist of phosphates.

The following is, as far as we are aware, the first detailed case report and analysis of a bladder stone from Nepal. In addition to the commonly held dietary cause, we suggest that a low calcium intake may contribute to stone formation.

Case report

A 5 year old Nepali boy had a one year history of dysuria, and his urine was often cloudy and sometimes blood stained. He was one of five children living in a family in a hill village about five hours' walk from Pokhara, Nepal's second largest town. His father worked on the land. His diet was vegetarian, consisting mainly of white rice, sometimes eaten with lentils, vegetables in season, and milk less than once a week. He drank fresh spring water from a nearby source.

He weighed 13.5 kg (just below the third percentile for age). He was normal when examined awake, but under anaesthesia it was possible to palpate a hard plum sized suprapubic mass.

An abdominal radiograph showed a 4 cm radioopaque bladder stone. The urine was sterile with a macroscopic sediment and a microscopic haematuria. Urine concentrations of calcium and oxalate were both low (0.42 mmol/l (normal 1.2-8.8) and 0.08 mmol/l (normal 0.2-0.6) respectively) and phosphate was normal (20.07 mmol/l (normal 11-32)). Serum concentrations of calcium (2.23 mmol/l), albumin (38 g/l), and urate (0.23 mmol/l) were normal while the concentration of phosphate was raised (3.24 mmol/l (normal 0.8-1.5)); creatinine was low (35 µmol/l (normal 50-130)).

A cystotomy was performed at which a beige 21 g stone was removed. He made an uneventful recovery, returning to his village one week later.

Both the stone and urine sediment were found to consist mainly of urates. x Ray crystallography showed a central core to the stone comprised mainly of ammonium acid urate with a much smaller proportion of calcium oxalate.

The mineral content of his drinking water was analysed. The concentration of calcium was low at



Figure Bladder stone removed from Nepali boy.

8.4 mg/l (Thames Water average at Surbiton, 99 mg/l). Other salts were as follows, with Thames Water average figures in brackets: magnesium 3.6 mg/l (4.3), phosphate 0.1 mg/l (3.1) and silicon dioxide 10.7 mg/l (range 3.8-11).

Discussion

Bladder stones are, in our experience, a common condition and an important cause of morbidity in Nepal. One of us (MA) anaesthetised about one such case each week at Gandaki Zonal Hospital, Pokhara: the stones were rapidly removed and placed among a large collection of similar stones, the heaviest of which were distributed to the surgical staff to be used as paperweights. Cystolithotomy constituted about 10% of operations on children in this hospital, a figure similar to that found in other hospitals in the 'bladder stone belt'.¹

The cause of the uneven distribution of bladder stones is unknown. They are not causally related to dehydration, malnutrition, vitamin A deficiency, urinary tract infections, hypercalciuria, nor hyperoxaluria. The dietary theory has gained most acceptance and proposes that bladder stones occur in communities that subsist on just one cereal-for example, rice, millet, or wheat but not maize, the staple in much of Africa.¹ The probable mechanism for this association is that such a diet raises the urinary ammonia concentration which, in the presence of the high uric acid concentration found in all normal children (falling to adult concentrations by the age of 10 years), precipitates out as ammonium acid urate.² In this case it was not possible to measure the urinary ammonia concentration because of storage.

We hypothesise that a low calcium intake may also predispose to bladder stones by causing hypocalciuria, which has been shown in vitro to favour the precipitation of ammonium acid urate.³ The calcium requirement of this boy was 400-500 mg a day⁴; this is much lower than in a temperate climate because of exposure to abundant sunshine. Nevertheless, his intake would have been inadequate: rice is a poor source of calcium, and milk was rarely taken. In hard water areas, drinking water can be a major source providing up to half of daily requirements but one litre of the drinking water in this case would have contributed a mere 8.4 mg of calcium. In response to a low calcium intake, hypocalciuria maintained the calcium balance in this case and he had not developed biochemical rickets. Other studies have reported that among stone sufferers there is a low dietary calcium intake, soft drinking water, and hypocalciuria; these studies, however, were performed before it was known that hypocalciuria could influence precipitation of ammonium acid urate and so a causal relationship was not suggested.¹⁵

The only other report linking drinking water to bladder stone formation is from Chad where stones were associated with high concentrations of silicon dioxide in the water⁶; the concentration was normal in the spring water drunk by the Nepali boy in this case.

Further study is needed to assess whether communities who eat a one cereal diet, as do most rural communities in Nepal, are protected from bladder stones if their dietary calcium intake is adequate or if the drinking water is hard. analysis of the water specimens and Dr M Dillon of The Hospital for Sick Children, Great Ormond Street, and Dr G Haycock of Guy's Hospital for help in preparing this manuscript.

References

- ¹ Andersen DA. The nutritional significance of bladder stones. Br J Urol 1962;34:160-77.
- ² Brockis JG, Bowyer RC, Ryan G, et al. Endemic bladder stones in Indonesia. In: Smith LH, Robertson WG, Finlayson PB, eds. Urolithiasis. Clinical and basic research. London: Plenum Press, 1981:325–8.
- ³ Bowyer RC, Brockis JG, McCulloch RK. The role of common urinary constituents in the precipitation of ammonium acid urate. *Clin Chim Acta* 1979;**99**:221-7.
- ⁴ Indian Council of Medical Research. *Recommended dietary intake for Indians*. New Delhi: Indian Council of Medical Research, 1981:26.
- ⁵ Aurora AL, Taneja OP, Gupta DN. Bladder stone disease in childhood. Acta Paediatr Scand 1970;59:177-84,385-98.
- ⁶ Cabrol D. La lithiase vesicale en Tchad. *Med Trop (Mars)* 1975;35:47-54.

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