short reports

small parathyroid adenoma. There was no evidence of bone or renal disease.

It is suggested that the diagnosis of hyperparathyroidism should be considered in a child with unexplained abdominal pain.

We wish to thank Dr. J. L. H. O'Riordan for the estimation of the serum concentration of parathormone, Mr. C. W. A. Falconer who performed the operation, and Professor J. A. Strong for his helpful advice.

REFERENCES


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Congenital rickets due to maternal vitamin D deficiency

Rickets and osteomalacia are common in immigrant Asians in this country (Holmes et al., 1973) and are probably due to dietary deficiency of vitamin D (Moncrieff, Lunt, and Arthur, 1973; Preece et al., 1973). Pregnancy is the period of greatest metabolic activity in adults and osteomalacia occurs particularly frequently in Asian immigrants at this time (Holmes et al., 1973). Though this might be expected to cause rickets in the fetus, to date this has been reported in only two babies in this country (Ford et al., 1973).

This paper describes a further example of congenital rickets in a baby whose mother had biochemical evidence of osteomalacia, and presents new evidence that neonatal hypocalcaemia may be due to maternal vitamin D deficiency.

Case report

The mother was a 30-year-old Asian who had had 3 children in the preceding 3 years. During the latter part of the present pregnancy she complained of pain in her sacrum and had a waddling gait. After delivery she was found to have a serum calcium level of 8.2 mg/100 ml, phosphorus 2.9 mg/100 ml, alkaline phosphatase 220 IU/l., and 25 hydroxycholecalciferol (25-HCC) 7.1 ng/ml (control 20 ng/ml). X-ray examination of the pelvis did not show osteomalacia. Her daily intake of vitamin D, estimated from dietary recall, was 150 IU (normal 600, Davidson and Passmore, 1966).

![Fig.—X-ray of wrist showing rickets.](http://adc.bmj.com/ on October 14, 2017 - Published by group.bmj.com)
Her baby daughter was born at term by a spontaneous delivery, weighing 2·75 kg. The sutures were widely separated, the fontanelle large, and the skull bones soft and easily indented. There was no swelling at the wrists or ankles, but the rib ends seemed enlarged. The clinical diagnosis of rickets was confirmed by a wrist x-ray (Fig.) and the results of biochemical investigations showed a low serum calcium and raised alkaline phosphatase levels. These and subsequent investigations are shown in the Table. Her level of 25-HCC was performed routinely in antenatal care of Asian women.

**Summary**

A newborn Asian baby with congenital rickets is described. Her mother had osteomalacia, which, in view of her low dietary intake of vitamin D and low level of circulating 25–HCC, was probably nutritional in origin. Biochemical screening of pregnant Asians for osteomalacia should be a routine part of antenatal care.

Dr. T. Stamp kindly measured the levels of 25–HCC.

**TABLE**

<table>
<thead>
<tr>
<th>Age (mth)</th>
<th>Calcium (mg/100 ml)</th>
<th>Phosphorus (mg/100 ml)</th>
<th>Alkaline phosphatase (IU/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 dy</td>
<td>6·7</td>
<td>5·5</td>
<td>227</td>
</tr>
<tr>
<td>10 dy</td>
<td>7·9</td>
<td>6·0</td>
<td>222</td>
</tr>
<tr>
<td>1</td>
<td>9·0</td>
<td>6·0</td>
<td>265</td>
</tr>
<tr>
<td>3</td>
<td>8·9</td>
<td>6·5</td>
<td>284</td>
</tr>
<tr>
<td>4</td>
<td>10·5</td>
<td>6·5</td>
<td>225</td>
</tr>
<tr>
<td>5</td>
<td>9·5</td>
<td>6·1</td>
<td>188</td>
</tr>
<tr>
<td>6</td>
<td>9·5</td>
<td>5·7</td>
<td>150</td>
</tr>
</tbody>
</table>

8·3 ng/ml. The infant was treated with 1000 units calciferol daily, later increased to 3000 units, calcium gluconate 300 mg three times a day for a fortnight, and was fed on S.M.A. After 6 months the rickets had healed.

**Comment**

The baby had clinical, biochemical, and radiological features of rickets detected in the first few days of life, and her mother had biochemical and clinical evidence of osteomalacia. The mother's dietary intake of vitamin D was lower than that required in pregnancy and the level of 25–HCC considerably below normal, which has not previously been shown in a case of neonatal rickets. These findings suggest that the mother had simple nutritional osteomalacia causing rickets to develop in the fetus before birth: true congenital rickets. This supports the suggestion made by Purvis et al. (1973) that maternal vitamin D deficiency is a cause of hypocalcaemia in the neonate.

Over the last few years a number of Asian neonates have been seen in Derby with prolonged hypocalcaemia and were regarded as examples of transient idiopathic hypoparathyroidism (Prader and Fanconi, 1969). Rickets was not suspected and the babies were treated with large doses of vitamin D. In retrospect, some of the cases were probably examples of congenital rickets. We agree with Ford et al. (1973) that rickets should always be considered as a cause of hypocalcaemia in an Asian neonate, and that biochemical survey for osteomalacia should be

**REFERENCES**


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**Hyperglycaemia and convulsions in children**

It is common practice to measure blood sugar content in patients with acute episodes of convulsions. However, very few reports deal with blood glucose levels during fits in infancy. Millichap (1968) cited studies of 86 patients with febrile convulsions who had normal values. Zellweger (1948) found raised values (from 117 to 296 mg/100 ml) in 6 out of 12 children examined during or immediately after a febrile convolution. Those authors cited by Millichap (1968) who found raised CSF glucose levels in children do not report the corresponding blood values. We wish to report our experience.
Congenital rickets due to maternal vitamin D deficiency.

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