was clearly not deficient in vitamin D, since her estimated vitamin D intake and plasma 25-hydroxy vitamin D concentrations were normal on admission. She responded slowly to vitamin D treatment both clinically and histologically despite supraphysiological plasma concentrations of 25-hydroxy vitamin D and high calcium absorption.

The osteomalacia most probably resulted from chronic phosphate depletion secondary to long-standing hyperparathyroidism. Subsequently, the increased absorption of phosphate that results from vitamin D treatment would be countered by high urinary losses. Final correction of the problem was achieved only by parathyroidectomy. The fall in plasma parathyroid hormone concentrations observed during vitamin D treatment occurred without any consistent rise in plasma calcium and may reflect a direct action of a vitamin D metabolite on parathyroid function. It serves, however, to underline the complex interaction of these calcium-regulating hormones in normal and pathological conditions.

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Requests for reprints to Dr R R Ghose.

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# Treatment of unidentified viper bites

In the tropics many people die from snakebites, and all snakebites, although many produce no signs of systemic poisoning, must be considered as potentially life-threatening.[1] Specific antivenom is effective treatment, but I believe that it is overused and too often given unnecessarily early. Most authorities suggest that antivenom should be used when there are signs of systemic poisoning—coagulation or neurological disorders.[1 2] Traditionally at this hospital patients bitten by snakes were seen by nurses and virtually all given subcutaneous antivenom automatically. We thought that most of these patients did not need antivenom, and so in August 1978 we introduced selective use of the antivenom, and I report our experience.

## Patients, methods, and results

Eighteen patients (16 males, 2 females, aged 12-45) presented between August 1978 and November 1979. All were questioned, examined, and had a test for clotting time using 2 ml of venous blood in dry, clean glass tubes. The test result was considered negative if the blood clotted within 10 minutes. All patients bitten within 12 hours of arrival at the hospital and all those with severe local reaction or signs of systemic poisoning were admitted and had their clotting time repeated after 4-6 and 12 hours. Those without signs of systemic poisoning were treated symptomatically and observed closely. Those with neurological disorders or clotting defects were given antivenom intravenously—20 ml Pasteur Institute's anti-Bitis-Echis-Naja-serum in 1 litre of normal saline over 1-2 hours. If normal clotting was not re-established this was repeated.

All but one of the 18 had swelling around the site of the bite, which was always painful. (The remaining one had fang marks as evidence of a definite bite.) Some had enlarged tender lymph nodes, none had visible tissue necrosis, and in 17 the bite was on the legs (one had been bitten on the hand). Ten patients described the snake, but only one brought the dead snake with him. Patients arrived at the hospital between one hour and three days after the bite, and 11 had had some treatment before coming. The treatment given had no effect as far as we could tell in all but one case, where an incision made by a local healer had caused severe prolonged bleeding because of a clotting defect. Sixteen patients were admitted, none developed neurological disorders, four had clotting defects on arrival, and of the other 12 none displayed clotting defects. The four patients with clotting defects came to hospital more than 12 hours after being bitten (one three days after), but all eventually responded well to treatment (details in table). The other 12 were discharged without developing any complications.

## Comment

We may reasonably assume that these patients were bitten by vipers, and we have shown that most patients coming to our hospital do not need antivenom. The selective use of antivenom not only reduces costs but also protects patients from the known hazards of giving heterologous serum. I emphasise, however, that those not given antivenom must be carefully observed as clotting defects can develop as late as 27 hours after the bite.[1]

The Pasteur Institute does not recommend intravenous use of its anti-Bitis-Echis-Naja-serum, but we have had no problems using it intravenously. Nevertheless, should the amount of physiological saline as diluent of the antivenom be reduced in patients at risk of congestive cardiac failure? The advantages of intravenous infusion are that it offers better control and may be quickly discontinued if problems develop, that an intravenous line is to hand for any emergency treatment of anaphylaxis, and that an intramuscular haematoma is avoided.

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*Clinical details of four patients who developed clotting defects after bites by unidentified vipers*

| Case No | Age (years) | Sex | Local reaction to bite | Degree of bleeding | Result of clotting test | Dose of antivenom given before clotting test negative | Course and comments |
|---|---|---|---|---|---|---|---|
| 1 | 40 | F | Severe oedema of leg | Slight bleeding of gums | + | 40 ml | Discharged after three days with still moderate oedema |
| 2 | 26 | M | Mild oedema of bitten foot | Bleeding of gums, slightly bloody sputum, and slight bleeding at the bite site after superficial incision | + | 40 ml | Spontaneous bleeding stopped after 20 ml of antivenom, clotting test negative after 40 ml, discharged on 4th day, completely recovered |
| 3 | 20 | M | Moderate oedema of leg | Severe bleeding from superficial incision on the head | + | 60 ml | Transfusion of 2 units of whole blood to correct shock and anaemia, after 40 ml of antivenom forming of a small clot within 10 min in clotting test, completely dissolved 20 min later; normal clotting test and definite haemostasis after 60 ml; slight foot oedema when discharged on 5th day |
| 4 | 20 | M | Moderate oedema of leg | Bleeding of gums and epistaxis | + | 20 ml | Discharged on 5th day with slight oedema |