oxalate, 10% magnesium ammonium phosphate, and the remainder calcium phosphate), but she has apparently made no more. Biochemical progress is shown in the figure.

Biochemical progress of two patients with hyperoxaluria treated with pyridoxine.

Conversion: SI to traditional units—Glycolate: 1 mmol/24 h $\approx$ 76.1 mg/24 h. Oxalate (anhydrous): 1 mmol/24 h $\approx$ 90 mg/24 h. Calcium: 1 mmol/24 h $\approx$ 40 mg/24 h.

Comment

Both patients showed hyperoxaluria that was clearly metabolic in origin since urinary glycolate excretion was also raised. The known causes for raised excretion of these acids are primary hyperoxaluria, pyridoxine deficiency, excess glycine load, and ethylene glycol intoxication. None of these fits the facts in these cases. The last two are obviously excluded, and there was no other evidence of pyridoxine deficiency (anaemia, convulsions). Nevertheless, some young women apparently exist on a low pyridoxine intake and show subclinical pyridoxine deficiency.³ So simple an explanation for hyperoxaluria seems unlikely in the second case, however, because of the finding of renal tubular acidosis, which has been described before in combination with hyperoxaluria.⁴ Primary hyperoxaluria is excluded by four facts. Firstly, in this condition urinary oxalate excretion is considerably higher than in these cases. Secondly, urinary glycolate excretion in primary hyperoxaluria is lower than urinary oxalate excretion. Thirdly, in primary hyperoxaluria the response to pyridoxine lasts only as long as the vitamin is given and immediately relapses on stopping it. These two patients did not relapse even after long periods of time. Fourthly, primary hyperoxaluria requires high doses of pyridoxine whereas in case 2 a remarkably low dose was effective.

It seems necessary to postulate a new cause of hyperoxaluria. There may have been a slightly increased requirement for pyridoxine, possibly induced by extraneous factors. The second patient had been taking oral contraceptives, a known cause of biochemical abnormalities, as found in pyridoxine deficiency,⁵ while the first patient had been an agricultural worker and might have been exposed to a pyridoxine antagonist. Whatever the cause of the hyperoxaluria the outcome was fortunate, with an apparently permanent remission on pyridoxine.

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² Kasidas GP, Rose GA. A new enzymatic method for the determination of glycolate in urine and plasma. *Clin Chim Acta* 1979;96:25-36.

³ Vir SC, Love, AHG. Effect of oral contraceptives on vitamin B₆ nutriture of young women. *Internat J Vit Nutr* 1980;50:29-34.

⁴ Dent CE, Stamp TGB. Treatment of primary hyperoxaluria. *Arch Dis Child* 1970;45:735-45.

⁵ Rose DP. The influence of oestrogens on tryptophan metabolism in man. *Clin Sci* 1966;31:265-72.

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Eosinophilia in patients undergoing dialysis

Over the past few years, many of our patients on maintenance haemodialysis have developed eosinophilia without apparent reason. To our knowledge, the published reports contain little information on the subject,¹ and major textbooks on dialysis do not even mention it.²

We report our experience with this phenomenon.

Patients, methods, and results

Nineteen (25%) out of 77 uraemic patients undergoing dialysis for 18 hours weekly with deionised water, RSP artificial kidneys, cuprophane coils, and polyvinylchloride tubing (all from Travenol Laboratories, Deerfield, Illinois) developed eosinophilia (>500 eosinophils/mm³). Dialysers were not reused. Mean number of eosinophils per mm³ was $1391 \pm (\text{SEM}) 319$; (range 500 to 9028). Eosinophilia appeared after an average of 20 ± 3 months of haemodialysis (range two to 35 months), and once it had appeared persisted in all patients but two, in whom the number of eosinophils returned to normal after having remained raised for six and 72 months. No cause for eosinophilia other than haemodialysis could be found in any patient. The stools of several patients with high degrees of eosinophilia were tested for ova and parasites, but results were negative. None of these patients had rashes, asthma, or other symptoms that could conceivably represent a hypersensitivity reaction related to the high eosinophil count. Nine of the 19 patients had pruritus, but this symptom was also present in 23 of the 58 patients without eosinophilia and was not significant. Age (43.5 ± 4.6 years), total duration of dialysis (61.7 ± 6.5 v 54.5 ± 4.7 months), clinical course, medications, and primary renal disease were similar in the patients with eosinophilia and those without it. Medications were basically: aluminium hydroxide, multivitamins, and—in some patients—propranolol and 25-hydroxycholecalciferol. In both groups primary renal diseases had a distribution similar to that usually seen in the general population. No patient had undergone splenectomy or had a history of malignancy, autoimmune disease, or collagen vascular disease.

Comment

This study shows that about 25% of those undergoing haemodialysis develop eosinophilia. It seems logical to assume that the raised eosinophil count is a consequence of the dialysis procedure per se if one considers that: (a) eosinophilia was not present before the start of haemodialysis and was therefore not due simply to renal failure, (b) there was little association with the diseases responsible for renal failure, (c) there was no obvious relation to any medication, (d) other causes of eosinophilia were not apparent, and (e) eosinophilia tended to persist once it had appeared, suggesting that it was somehow maintained by repetitive dialysis.

Patients undergoing haemodialysis are subject to many substances that may invoke hypersensitivity, such as heparin, plasticisers from tubing,³ particles of rubber from stoppers, etc. Any one of these substances, or a combination of them, could lead to the development of eosinophilia, and further studies are clearly needed to determine its causes.

Although serious cutaneous hypersensitivity³ and anaphylactic reactions⁴ have been associated with eosinophilia in patients undergoing dialysis, our experience appears to show that eosinophilia in patients undergoing dialysis is a benign laboratory abnormality that does not necessarily result in increased morbidity. Contrary to previous reports,¹ reuse of dialysers did not seem to be implicated.

¹ Hoy WE, Cestero RVM. Eosinophilia in maintenance hemodialysis patients. *Journal of Dialysis* 1979;3:73-87.

² Drukker W, Parsons FM, Maher JF, eds. *Replacement of renal function by dialysis*. The Hague: Martinus Nijhoff, 1979.

³ Bommer J, Ritz E, Andrassy K. Necrotising dermatitis resulting from hemodialysis with polyvinylchloride tubing. *Ann Intern Med* 1979;91: 869-70.

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