well controlled with topical agents until four days earlier, when he had purchased a 1 kg packet of Dead Sea Mineral Salts, which he added to his bathwater. Within two days he had become generally unwell and his psoriasis erythrodermic. He required admission to hospital and treatment with methotrexate and oral retinoids. His psoriasis remained well controlled until two years later, when it was noted to be once again inflamed and unstable; on direct questioning he admitted that he had recently again tried the Dead Sea Mineral Salts, though on this occasion he had used only about 250 g per bath. The stated ingredients of the preparation used (Dead Sea Health Products, Kibbutz Ein Gedi MP, Dead Sea, Israel) included potassium chloride 22-28%. magnesium chloride 30-34%, bromides 0·2-0·4%, and sulphates 0·1-0·2%.

Case 3—A 22 year old woman with lifelong mild topic eczema visited a naturopath, not medically qualified, who recommended an injection of a homoeopathic remedy, Nat Mur 200. Within 12 hours she was in an acute erythrodermic state, requiring intensive medical treatment. The mechanism of this reaction was obscure, since homoeopathic remedies are not normally administered by injection and are usually in such a highly diluted form as to contain no measurable quantity of the original ingredients.

Comment

Alternative or complementary medicine is widely used for a range of disorders. Many patients find benefit, and though few of these therapies have been subjected to the same critical evaluation of safety and efficacy as conventional medicines, even the most sceptical of doctors generally regard them as harmless; indeed, it may well be the apparent safety of these forms of treatment which contributes to their popular appeal.

These three cases illustrate severe cutaneous reactions to different sorts of alternative medicines. The prevalence of such adverse reactions is impossible to assess, since there is no central monitoring authority equivalent to the Committee on the Safety of Medicines; furthermore, patients may be reluctant to admit to their doctor that they have employed a non-medical remedy. Alternative medicines are also not subject to the same requirements of standardisation as pharmaceuticals.

Many forms of complementary medicine are of potential value. Nevertheless, it is also important to be alert to their potential hazards. A valuable first step would be a central monitoring body.

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Effect of milk on patients with duodenal ulcers

A diet with a high milk content is often advised for patients with a duodenal ulcer, perhaps because milk is effective at neutralising acid.[1] Moreover, epidemiological data suggest that the incidence of peptic ulcers is inversely related to milk consumption.[2] In contrast, a diet with a high milk content stimulates significantly greater acid production than a normal diet[3]; its value to patients with duodenal ulcers has therefore been questioned.[4] The effect of milk on the healing of duodenal ulcers has not, however, been clearly established, so we performed a controlled therapeutic trial on patients admitted to hospital with ulcers.

Patients, methods, and results

Sixty five consecutive patients with duodenal ulcers confirmed by endoscopy were included in the study. On admission to this hospital they were randomly allocated to one of two groups, group I consumed a normal hospital diet, and group II received a diet consisting exclusively of milk (500 ml at breakfast, 750 ml at lunch, and 750 ml at dinner). They were allowed to add sugar according to their taste, and both groups were allowed to consume seasonal fruits. The total daily intakes of calories (1800-2000) and protein (60-70 g) in the two groups were very similar. Both groups received cimetidine 200 mg three times daily and 400 mg at bedtime, making a total of 1 g a day. Treatment continued for four weeks, after which patients underwent endoscopy again. The ulcers were then categorised as either healed, with or without residual duodenitis, or not healed (irrespective of size).

Three of 33 patients (9%) in group II developed milk intolerance in the form of diarrhoea and colicky abdominal pain and were excluded from the study. Weekly comparison of symptomatic relief showed no appreciable difference between the two groups throughout the study (table). Endoscopic assessment after four weeks of treatment showed that the proportion of healed ulcers was significantly higher in patients receiving a normal diet (78%) compared with those who consumed only milk (53%) ($p<0·05$). Serum calcium and creatinine concentrations were within the normal range in both groups before and after treatment.

Age of and clinical information on patients with ulcers participating in study

	Group I (normal diet)	Group II (milk diet)
Patients:		
Total	32	33
Dropped out	0	3
Completed treatment	32	30
Age (years):		
Mean (SD)	37·4 (9·3)	39·6 (9·9)
Range	22-60	24-70
Median	35	40
Ratio M:F	32:0	30:0
Duration of symptoms (years):		
Mean (SD)	5·2 (3·0)	6·1 (3·2)
Range	1-12	1-14
Median	5	6
No (%) of smokers	27 (84)	23 (77)
No (%) of patients whose symptoms were relieved:		
Week 1	11 (35)	11 (37)
Week 2	21 (66)	19 (63)
Week 3	24 (75)	21 (70)
Week 4	28 (88)	25 (83)
No (%) of patients whose ulcers healed as shown by endoscopy	25 (78)	16 (53)*

*$\chi^2=4·24$; $p<0·05$. There was no significant difference in the other parameters between the two groups.

Comment

In a previous study Doll *et al* found that a diet with a high milk content and even an intragastric milk drip did not influence the healing of peptic ulcers.[1] Their study, however, was based on a barium meal examination, which is not as accurate as endoscopy in assessing ulcer healing, especially in the presence of a deformed duodenal bulb. In our study the proportion of healed ulcers in patients on a normal diet was 78%, which is the usual response obtained with cimetidine. In contrast, there was a significant reduction in the proportion of healed ulcers in patients who received a diet with a high milk content. Pain relief in the two groups was very similar, and this is perhaps why milk has been found to be useful in treating duodenal ulcers.

The precise mechanism by which milk delays ulcer healing is conjectural. There is no evidence to suggest an interaction between milk and cimetidine. One possibility is that the high content of calcium in milk stimulates excess acid production. Extracellular calcium plays a part in gastrin mediated acid production, and patients with duodenal ulcers are more sensitive to this effect than normal subjects.[5] Calcium may not be the only factor, however, as even milk that is low in calcium produces a significantly greater increase in acid secretion in patients with duodenal ulcer than in normal subjects.[4]

We suggest that a diet with a high milk content has an adverse effect on the healing rate of duodenal ulcers and should not be recommended for these patients.

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1 Doll R, Friendlander P, Pygott F. Dietetic treatment of peptic ulcer. *Lancet* 1956;i:5-8.
2 Paffenbarger RS Jr, Wing AL, Hyde RT. Chronic disease in former college students. XIII. Early precursors of peptic ulcer. *Am J Epidemiol* 1974;**100**:307-15.
3 Bingle JP, Lennard-Jones JE. Some factors in the assessment of gastric antisecretory drugs by a sampling technique. *Gut* 1960;**1**:337-44.
4 Ippolite AF, Maxwell V, Isenberg JI. The effect of various forms of milk on gastric acid secretion. Studies in patients with duodenal ulcer and normal subjects. *Ann Intern Med* 1976;**84**:286-9.
5 Christiansen J, Kirkegaard P, Olsen PS, Peterson B. Interaction of calcium and gastrin on gastric acid secretion in duodenal ulcer patients. *Gut* 1984;**25**:174-7.

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