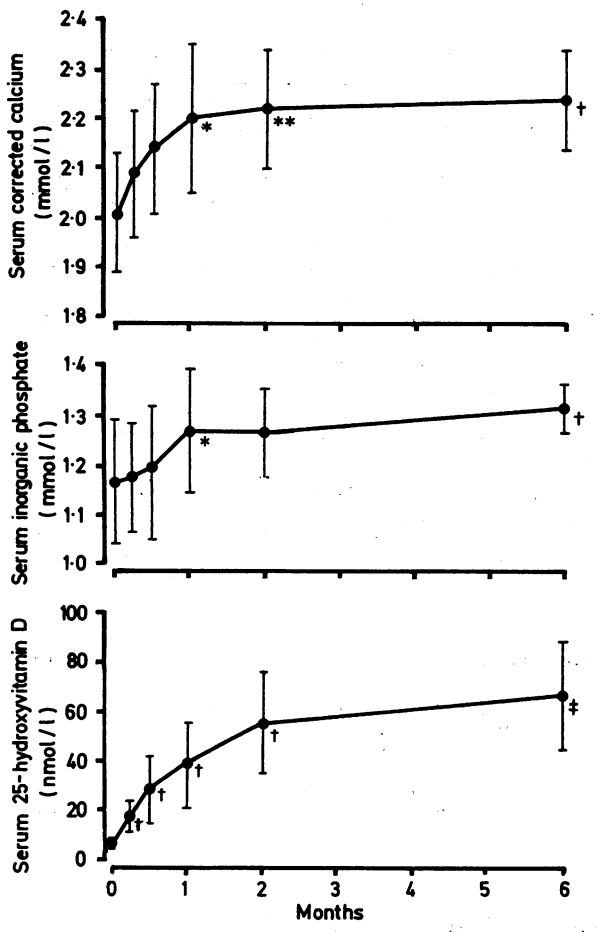
and inorganic phosphate were still normal in the three patients followed up beyond six months. Serum 25-hydroxyvitamin D concentrations were still adequate in each case (28, 85, and 55 nmol/l respectively (11·2, 34, and 22 ng/ml)).



Mean (SD) serum corrected calcium, inorganic phosphate, and 25-hydroxyvitamin D concentrations before and after 15 mg calciferol intramuscularly. Serum calcium concentration was corrected for serum albumin concentration by the formula: corrected calcium (mmol/l)=measured calcium (mmol/l)+ 0·023 (40−albumin (g/l)).

Paired t test comparing values before and after treatment: *$p<0·05$; **$p<0·02$; †$p<0·01$; ‡$p<0·001$.

Conversion: SI to traditional units—Corrected calcium: 1 mmol/l≈4 mg/100 ml. Inorganic phosphate: 1 mmol/l≈3 mg/100 ml. 25-hydroxyvitamin D: 1 nmol/l≈0·4 ng/ml.

Comment

The biochemical responses observed in our seven patients were similar to those previously described in treated osteomalacia.[3] We found that a single 15 mg injection of vitamin D was effective in initiating and sustaining healing of osteomalacia for at least six months. In three subjects the single injection ensured adequate serum 25-hydroxyvitamin D concentrations for at least a year. As with other vitamin D regimens, subsequent treatment depends on the results of regular biochemical monitoring. The effectiveness of our single dose treatment in increasing 25-hydroxyvitamin D concentrations is comparable with that of single large oral doses of vitamin D in Asian immigrants without osteomalacia.[4] Our regimen is safe: no patient developed hypercalcaemia, and concentrations of 25-hydroxyvitamin D remained well below values seen in poisoning with vitamin D. Vitamin D given intramuscularly probably remains at the site of injection and is released slowly into the blood.[5]

Poor compliance is a problem in elderly people especially in those living at home, as were four of our patients. Patients may already be taking drugs on a regular basis for other ailments, and so a method of treatment that does not require "another bottle of tablets" would be worth while.

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Proliferation of IgDκ plasma cells after agranulocytosis induced by dapsone

Although an excess of lymphoid, plasma, and stromal cells is a known characteristic of bone marrow in idiosyncratic neutropenia induced by drugs, proliferation of monoclonal plasma cells in this disorder has not previously been reported. We report on a patient in whom dapsone caused a reaction with these characteristics and the M component was IgDκ.

Case report

A 52 year old man was admitted to hospital having received dapsone for 30 days for suspected dermatitis herpetiformis. He showed general malaise, had been feverish for the past three weeks, and in the past few days had developed tonsillitis and white plaques in the oropharynx. His peripheral blood showed a white cell count of $2·1\times10^9$/l, neutrophils $0·12\times10^9$/l, haemoglobin concentration 12·6 g/dl, and platelets 680×10^9/l.

Examination of aspirated bone marrow showed a heavy infiltrate of immature plasma cells, many of them multinucleate and containing Russell bodies and 90% of them containing intracytoplasmic IgDκ when observed by direct immunofluorescence. Promyelocytes were the only myeloid element present, while cells of the erythroid and megakaryocytic lines, though morphologically normal, were reduced in numbers. The serum contained an IgDκ M component and the urine κ chains. Bone studies and studies aimed specifically at virus antibodies, protozoans, and mycobacteria yielded negative results. The buccal plaques contained *Candida albicans*. Cutaneous biopsy showed pityriasis lichenoides.

Empirical treatment with antibiotics including antifungal drugs was started. On the eighth day the fever and oropharyngeal disorders abated and signs of myeloid regeneration were observed. After a period of thrombocytosis the peripheral blood counts had returned to normal by the 20th day in hospital. He was found to be asymptomatic and was discharged. On the day before discharge the bone marrow was normal; plasma cells accounted for at most 3% in all fields and were morphologically normal. Immunoelectrophoresis, performed in parallel with the intial sampling, confirmed the disappearance of IgD paraprotein from the serum and light κ chains from the urine.

Comment

Idiopathic monoclonal gammopathy is associated with many diseases. It is also found in healthy subjects, some of whom may subsequently develop B lymphocyte disorders, but whether the gammopathy is the cause or effect in such cases is not known. In only about 4% of patients with idiopathic monoclonal gammopathy and another disease does the gammopathy regress at the same time as the associated disease,[1] and this has not previously been reported when the gammopathy has featured IgD as the M component. Furthermore, dyscrasia of plasma cells has been associated with agranulocytosis only once before, and in that case it was not possible to prove any cause and effect relation.[2] So far as we know an increase in monoclonal antibodies in serum as a reaction to agranulocytosis has never been reported.

It is difficult to establish a pathogenic relation between idiosyncratic agranulocytosis induced by dapsone and the proliferation of monoclonal IgD.[3] Although it seems to be well established that B cells bearing IgD have a role in the propagation of immunological memory, the biological role of IgD in the serum remains controversial.[4]

Nevertheless, antibody activity against insulin and penicillin has been associated with IgD,[5] which led us to wonder whether this immunoglobulin molecule represents a septor for the antigenic determinants in dapsone, although we could not confirm this in our patient.

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Upper gastrointestinal endoscopy in the elderly

Upper gastrointestinal endoscopy in the elderly is safe[1] and reliable and gives a high diagnostic yield.[2 3] We studied 100 patients aged over 70 to determine whether endoscopy helps in the overall management of elderly patients.

Patients, methods, and results

We studied 104 patients aged over 70 who had been referred consecutively for diagnosis by upper gastrointestinal endoscopy. Endoscopists did not select patients whose management might be helped by endoscopy or give advice about management except on specific request. Four patients were excluded because of inadequate records, leaving 47 women and 53 men (median age 76). Sixty nine patients were in their 70s, 27 in their 80s, and four over 90. Three patients suffered minor complications—namely, transient bradycardia with hypotension, brief confusion, and bronchospasm. One patient died within a few hours of endoscopy due to continuing gastrointestinal blood loss.

Appreciable abnormalities were found in 67 patients (table), but management was changed in only 35 of the 100 patients (seven of the 31 over 80 years old). Of these, 23 were prescribed an H_2 antagonist, eight had operations (five for carcinoma of the stomach and one each for recurrent carcinoma of the stomach, benign stomal ulcer, and hiatus hernia), three had oesophageal dilatation (one also had radiotherapy), and one was given ketoconazole for oesophageal candidiasis. Of the remaining 65 patients, eight were already receiving H_2 antagonists, five required further investigations, and in three the examinations were technical failures. Appreciable abnormalities presumed to be associated with the patients' clinical problems included duodenitis and gastritis but excluded thickened folds and small polyps. This diagnostic decision was arbitrary, and treatment for these conditions is of doubtful efficacy; therefore analysis of the effect of endoscopy on management is even more relevant.

Barium meal examinations were performed before endoscopy in 49 patients. Only one such examination gave completely normal results, but a benign gastric ulcer was identified by endoscopy in this case. Results were confirmed by endoscopy in 28 patients, and in three endoscopy was a technical failure. The remaining 18 patients showed disparity between the two examination results. In 10 gastric carcinoma was suspected on barium meal examination, but at endoscopy four had normal stomachs, three had gastritis, two had benign ulcers, and one had a benign polyp. Of three patients in whom duodenal ulcers were suspected radiologically, one had duodenitis and two had normal duodenums. Four of the nine patients with confirmed duodenal ulcers had barium meal examinations, which showed the ulcer. Three patients with suspected oesophageal carcinoma proved to have benign strictures at biopsy, and one with suspected achalasia had a normal oesophagus.

Management was changed in one third to a half of the patients in whom the indication for endoscopy was dyspepsia, bleeding, anorexia, or dysphagia. Endoscopy was not helpful, however, in determining the management of iron deficiency anaemia: only three lesions were found in 11 patients, and management was changed in only one patient, who was given an H_2 antagonist.

Comment

This study confirms that upper gastrointestinal endoscopy gives a high diagnostic yield in the elderly (77% or more).[2 3] We showed for the first time, however, that management is changed in half the elderly patients in whom an appreciable abnormality is diagnosed by endoscopy. Endoscopy was disappointing only in the search for a cause of iron deficiency anaemia.

Reports have shown a discrepancy between results of barium meal examination and endoscopy in the elderly.[2 3] In our study this was largely due to suspicion of malignancy in lesions in the stomach and oesophagus. Biopsy performed during endoscopy remains the definitive method of confirming or refuting a diagnosis of carcinoma. We emphasise that in the critical assessment of a diagnostic technique its influence on management is more relevant than its diagnostic yield.

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Yield of diagnoses and changes in management by indication for endoscopy

	Indication						
	Total	Dyspepsia	Acute gastrointestinal bleeding	Iron deficiency	Dysphagia	Weight loss or anorexia	Other*
No of patients	100	45	26	11	10	6	2
No with appreciable abnormality (%):	67 (67)	36 (80)	17 (65)	3 (27)	7 (70)	3 (50)	1 (50)
Oesophagitis with or without hiatus hernia	10	6	1		3		
Benign stricture with or without hiatus hernia	3			1	2		
Oesophageal ulcer	2		2				
Oesophageal carcinoma	3	2			1		
Oesophageal candidiasis	1	1					
Gastritis	7	4	1	1		1	
Gastric ulcer†	17	6	9		1	1	
Gastric carcinoma‡	12	10				1	1
Duodenitis	3	3					
Duodenal ulcer	9	4	4	1			
No without appreciable abnormality or technical failure	33	9	9	8	3	3	1
No whose management was changed (%)	35 (35)	15 (33)	13 (50)	1 (10)	4 (40)	2 (33)	0

* One epigastric mass and one endoscopy for biopsy of small bowel.
† Including one stomal ulcer.
‡ Including one recurrent stomal carcinoma.