Asymptomatic peptic ulcer disease

Peptic ulcer disease is usually diagnosed on the basis of well recognised symptoms confirmed by endoscopy or barium study. Within the community an unknown number of people, however, may have peptic ulcer disease without symptoms, some of whom present only because of haemorrhage or perforation.¹ Roughly a third of the cases of perforated peptic ulcer, for example, occur in people without any previous symptoms, as do a fifth of gastrointestinal haemorrhage.^{2,3}

There are no series reporting asymptomatic people who have been studied with endoscopy, and though necropsy studies indicate that almost a fifth of the population may have some evidence of peptic ulcer disease—active ulcer, scars, or previous surgery—the proportion of those without symptoms has not been recorded; nor is it clear whether the ulcer was a feature of the terminal illness.⁴ Trials of treatment for duodenal and gastric ulcer have shown that ulcers may persist or recur in patients who have remained symptom free,^{5,6} although they are clearly different from the general population in that they have previously had symptoms and will probably also develop further ones within the next year.⁷ Elderly people, patients with rheumatoid arthritis, and those taking non-steroidal anti-inflammatory drugs are all at greater risk of peptic ulcer disease, which may remain asymptomatic until presentation with haemorrhage or perforation^{8,9}; moreover, the non-steroidal drugs may also mask the symptoms of peptic ulcer.¹⁰

The prognosis for true asymptomatic peptic ulcer disease must be good as it will be discovered only at necropsy or not at all. Even if it is found apparently incidentally at endoscopy, however, it is difficult not to attribute some of the patient's vague gastrointestinal symptoms to the lesion. For patients with a perforated ulcer simple oversewing of the ulcer appears successful in that perforation recurs in only a few.¹¹ Although most of these patients remain symptom free, this does not imply that the ulcer has healed. Asymptomatic ulcer presenting with haemorrhage is a cause for concern, as

the mortality may be high³ and rebleeding may occur; long term medical treatment or close endoscopic surveillance is required.

Peptic ulcer represents a disease process continuing for perhaps 10-15 years,¹² and most patients are treated for their symptoms or given maintenance treatment for only a fraction of this time. For reasons of cost and feasibility—and often desirability—medicine today still responds only to the patient's symptoms and does little to offer surveillance to an asymptomatic person other than to one with a recognised increased risk of cancer. Hence, although a lot of people in the community are likely to have unrecognised peptic ulcer disease, any proposals for endoscopic or radiological screening to discover it, followed by treatment to suppress the process, are hardly likely to find favour with clinicians.

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Cutaneous graft versus host disease

Graft versus host disease occurs when immunocompetent lymphoid cells of the graft are introduced into an immunosuppressed histoincompatible host. In man it is seen most commonly as a complication of bone marrow transplantation. It may also occur after cell transfer from the mother to a fetus with cell mediated immunodeficiency and after transfusion of non-irradiated blood or blood products in immunodeficient patients. The skin is a major target organ of the disease and our understanding of the immunopathogenesis has important implications for dermatology. Cutaneous graft versus host disease provides a unique model for studying the pathogenesis of several "idiopathic" skin diseases.

In a study of 100 consecutive bone marrow transplantations performed at Westminster Hospital before 1983 the incidence of graft versus host disease was 76%, and in all cases the skin was affected.¹ Since then the routine use of cyclosporin A² and more recently T cell depletion of

donor marrow^{3,4} has greatly reduced the incidence. Skin manifestations are usually the earliest sign of the disease; in its acute form graft versus host disease is seen as a generalised morbilliform eruption with erythema of the palms and soles. The patients who are severely affected develop an exfoliative erythroderma which may progress to toxic epidermal necrolysis.⁵ Chronic skin disease presents as a variety of lichenoid lesions⁶ that are clinically and histologically indistinguishable from lichen planus followed by pigmentary changes, poikiloderma, and sclerosis (morphoea).⁷⁻⁹ The last tends to be localised, although rarely it may become extensive (generalised morphoea). Oesophageal involvement and subcutaneous calcification, simulating scleroderma, have been reported.¹⁰ Chronic graft versus host disease may also present as various autoimmune or connective tissue diseases, sharing overlapping features of lupus erythematosus, dermatomyositis, polymyositis, Sjögren's