

Antenatal screening for syphilis

Still important in preventing disease

Papers p 1617

Much congenital infection is now preventable. Antenatal screening is an important measure in reducing vertical transmission of syphilis, hepatitis B, and HIV, as effective interventions are available but their delivery depends on identifying infected women. Maternal syphilis is readily treatable with parenteral penicillin, which prevents the sequelae of miscarriage, stillbirth, neonatal death, and congenital infection—with its long term morbidity of learning difficulties, interstitial keratitis, and neural deafness.

Syphilis is now uncommon in the United Kingdom. In 1996 only 91 cases of women with early, potentially transmissible infection were reported by genitourinary medicine clinics in England.¹ Congenital syphilis is even rarer, and many paediatricians have never seen an infected child. Nevertheless, syphilis is currently the only chronic infection for which women are routinely screened during pregnancy (M L Newell et al, unpublished data).

In view of this perceived rarity, and the absence of formalised national policy, some units are considering discontinuing screening. To inform policy making, Hurtig et al and the British cooperative clinical group carried out active surveillance to measure the incidences of syphilis in pregnancy and congenital syphilis throughout the United Kingdom over three years (p 1617).² During this time 139 women were treated for syphilis in pregnancy, of whom 121 were detected by antenatal screening. Thirty one women had early, congenitally transmissible infection. Nine cases of congenital infection were identified: one followed inadequate maternal treatment and the remainder absent or delayed antenatal care. Reporting was incomplete, so these were minimum figures.

Which women were most at risk? There was significant geographical variation, with 73% of women reported from the Thames regions and none from East Anglia. Country of birth was stated by 136 women, of whom 80% were born outside the United Kingdom. Infection was commonly imported, with acquisition abroad in 18 out of 23 women with transmissible syphilis. Information about ethnicity was provided for 134 women: 25% were white, 14% Asian, 31% black African, and 19% black Caribbean.

Would selective screening be helpful in identifying infected women? Although being born abroad or being of a non-white ethnic group were strong risk factors, cases were reported in white women born in the United Kingdom. Thus cases would be missed even if a selective screening programme was implemented

optimally—whereas in reality high risk individuals are often missed in such programmes.³ In addition user acceptability could militate against such an approach. At present syphilis screening is often carried out with little or no discussion, and no mention in information leaflets, and many mothers are unaware that they have ever been tested for syphilis. Women might legitimately feel upset if it became known that, for example, antenatal clinics were testing only non-white women for this sexually transmitted infection.

Geographical distribution might be a more logical basis for limiting testing. There is no room for complacency, however, as syphilis is far from being eliminated and remains both a major pathogen in its own right and a factor increasing HIV transmissibility. Cheap and easy international travel can facilitate the movement of infections as well as people. Syphilis is endemic in Africa and south Asia, and there is currently a major epidemic in Russia, with a 62-fold increase in notifications since 1988.⁴ In Bristol last year there was an outbreak of 46 cases of early infectious syphilis, of which three were identified by antenatal screening (P Horner, personal communication). Many examples exist where the relaxation of monitoring and prevention measures for sexually transmitted infections has been followed by rapid re-emergence of disease. Continuing surveillance also provides an early warning of infections⁵—which is especially beneficial in a population in which treatment prevents disease in at least two people.

How costly is syphilis screening? Blood is being taken anyway, so the costs are those of the laboratory tests—about 88p per live birth. Stopping antenatal screening nationally would currently release about £660 000 but result in missing at least 10 women a year with early syphilis, and consequent fetal deaths and congenital disease. We would also lose a major early warning system for adult infection. Even in East Anglia, where the prevalence is lowest, a cost benefit analysis concluded that antenatal screening remained worthwhile.⁶ A formal options appraisal by the Public Health Laboratory Service recommends that universal antenatal screening for syphilis should be continued.⁷

Instead therefore of abandoning screening we should ensure that we have an effective national programme, with standards for the screening, diagnosis, and management of expectant mothers and their infants. Such a scheme will be most effective and least costly if integrated closely with routine antenatal screening for other infections such as hepatitis B and

rubella—and HIV as this test becomes normalised and uptake increases. If we are to prevent congenital infection, we must ensure that sexually transmitted agents are not neglected for, human nature being what it is, they are unlikely ever to be eradicated.

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Proton pump inhibitors may mask early gastric cancer

Dyspeptic patients over 45 should undergo endoscopy before these drugs are started

Gastric cancer is still widely regarded as an incurable condition in the West. However, this nihilistic approach is no longer tenable as this cancer is eminently curable if it is diagnosed and treated at an early stage.¹ The five year survival of patients undergoing appropriate surgery for early gastric cancer is greater than 90%. Screening of the asymptomatic population, such as occurs in Japan, would not be feasible or cost effective in Western countries, so early diagnosis has to rely on symptomatic patients presenting to their general practitioners, who then recognise the importance of the symptoms and refer them for endoscopy. Since the early symptoms are often indistinguishable from those of benign ulcer disease, the inappropriate use of powerful antiulcer drugs has had the effect of masking the true diagnosis in some cases.

A significant proportion of patients with early gastric cancer do experience symptoms and in most these are typical dyspeptic symptoms.² For this reason referral for endoscopy is recommended for all patients aged over 45 with new onset dyspepsia, who comprise the group at risk for gastric malignancy.³ Widely available open access endoscopy services now make it possible to diagnose at least 20% of gastric cancers at an early stage, when disease is confined to the mucosa or submucosa of the stomach.¹ It is thus worrying that a significant delay in diagnosis in symptomatic patients still occurs before referral for endoscopy.⁴

Although the reasons for delay are multifactorial, one element is undoubtedly the prescription of ulcer healing drugs before endoscopy. This continues to occur despite repeated advice against this practice. Studies of referrals to endoscopy services consistently show that a significant proportion of patients are still prescribed antisecretory drugs before gastroscopy.⁵ Soon after the introduction of H₂ receptor antagonists there was evidence that these drugs could mask the symptoms of gastric cancer.⁶ The mechanism of action is presumed to be similar to that for benign ulcers.

The more recently introduced and more powerful acid suppressing proton pump inhibitors produce significantly more rapid symptom control and healing of benign ulcers. It is not surprising to find that these

drugs also rapidly abolish the dyspeptic symptoms of early gastric cancer.⁷ Importantly, well documented cases also exist of ulcerated early gastric cancers that have healed endoscopically after a short course of a proton pump inhibitor.⁸ Ulcerated lesions visible at an initial gastroscopy may be virtually undetectable even by experienced endoscopists after less than four weeks' treatment with these drugs. Such "healing" may occur even more quickly, although for ethical reasons this has not been formally investigated. There is also no information on how long it takes for such patients to become symptomatic again after they stop treatment. Those with missed early cancers also risk being labelled as having non-ulcer dyspepsia and might therefore receive repeated courses of antisecretory drugs, including proton pump inhibitors, thus further delaying the diagnosis.⁵

Thus two points exist at which the inappropriate prescription of proton pump inhibitors may delay or even prevent the diagnosis of early gastric cancer. Firstly, rapid control of dyspepsia may lead the patient or general practitioner to underestimate the importance of this symptom, so referral for endoscopy is delayed or even deferred. Secondly, if the patient should later undergo a gastroscopy then the prior treatment with these drugs may mask the endoscopic signs and the diagnosis may be missed. Although some endoscopy units instruct patients not to take antisecretory drugs for 7-14 days before the examination, there is no evidence that early lesions will become endoscopically visible again over this period. There must be serious concern that curable early gastric cancers are being missed and patients treated inappropriately. In some cases patients may not be diagnosed until they develop the more sinister symptoms of advanced cancer—which can be years later.^{9 10}

On the basis of the present evidence and the unanswered questions about the effects of even short courses of proton pump inhibitors in patients with early gastric cancer the message has to be reinforced. The manufacturers of these drugs state that they should not be prescribed to "at risk" dyspeptic patients without an endoscopic diagnosis,¹¹ as does the *British*