aids in ensuring the compliance that is required to avoid the nutritional consequences and the risk of malignancy.

Diagnosis by diet

There is absolutely no place for giving patients with suspected coeliac disease an empirical therapeutic trial of a gluten-free diet. Some people with a normal small bowel develop symptoms such as bloating and diarrhoea from the fermentation of wheat starch. This is referred to as non-coeliac gluten intolerance. They will improve after removal of gluten. Their symptoms recur if they are rechallenged but most are unwilling to do this if it is decided to rule out coeliac disease. The time to investigate someone for coeliac disease is at the time when the suspicion is first raised, and before prescribing a gluten-free diet.

Diagnosis of coeliac disease in children

The principles of diagnosis in children are the same as in adults. A general anaesthetic may be required for endoscopic biopsy. Children have a high frequency of transient IgA deficiency, meaning that IgA antibody tests are less reliable and measurement of total IgA is important. The serological and histological changes of coeliac disease might not occur until children have had gluten in their diet for at least two years. It is therefore important to ask about the amount and duration of gluten intake and whether this has been normal or restricted. Negative serological or other investigations done before two years of age should be repeated at a later time if coeliac disease is still suspected.

Confirmation of diagnosis

In children and adults the diagnosis of coeliac disease should be confirmed by a repeat small bowel biopsy after at least six months on a gluten-free diet. Symptom resolution alone is not a reliable guide to histological improvement. In the majority, the mucosa will have returned to normal. In some there may be persistent villous atrophy, although this is usually mild and improved compared with the pre-treatment appearance.

In the past, it was recommended that all children with coeliac disease undergo gluten challenge and biopsy as final confirmation of the diagnosis. However, recent guidelines recommend that this only be done in selected children where there is doubt about the initial diagnosis on clinical or histological grounds.[5]

Additional investigations

The determination of HLA phenotype is of little value in diagnosis or screening because of its frequency in the general population, despite the strong association of the HLA-B8, DR3, DQ2/DQ8 haplotype with coeliac disease.

Tests for nutritional deficiencies, such as iron, folate, calcium and vitamin D, may give a clue as to the possibility of malabsorption and the need for diagnostic testing but do not help in the diagnosis. They also give a guide to nutritional therapy. The same is true of measurement of bone mineral density. Specific tests for malabsorption, such as the d-xylose test, are no longer used.

Conclusion

Coeliac disease is more common than previously thought.[1] A high index of suspicion is important. Diagnosis still depends on the demonstration of villous atrophy on small bowel biopsy, with repeat biopsy after at least six months on a gluten-free diet. Antibody tests alone are not sufficient for diagnosis, but are useful in screening. All patients with detectable antibodies should undergo biopsy.

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Self-test questions

The following statements are either true or false (answers on page 47)

3. Before children with suspected coeliac disease are subjected to endoscopy, they should be given a trial of a gluten-free diet.
4. Coeliac disease can be excluded if the patient has no IgG antigliadin antibodies.

Patient support organisations

The Coeliac Society of Australia

The Coeliac Society of Australia supports people who have been diagnosed with coeliac disease, and their families. It also supports sufferers of dermatitis herpetiformis and those medically diagnosed as requiring a gluten-free diet.

The State and Territory societies (see opposite) give advice and information about the gluten-free diet, ingredients and where to buy them, recipes and cooking, overseas travel, educational material, and research into coeliac disease. The Society works with food authorities and manufacturers to promote standards and labelling of food products.

Support groups have been set up throughout the States and Territories. Coeliac Awareness Week is held each year in March.

Contacts

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Your questions to the PBAC

Bisphosphonates

A recent case highlighted the problems with authority prescriptions for bisphosphonates. A man with steroid-induced osteoporosis is at risk of fractures, but is unable to be prescribed bisphosphonates under the current conditions of the Pharmaceutical Benefits Scheme (PBS). In this case bone densitometry showed clearly that the patient had very low bone density.

The consultant has decided to use alendronate to improve this patient's prognosis. My question to the Pharmaceutical Benefits Advisory Committee is why is it necessary to wait until the patient inevitably cracks some bones before therapy can commence. A private prescription is quite expensive – about $90 for one month of treatment with alendronate 10 mg.

I was informed by the PBS Hotline that alendronate is not subsidised for male patients, however calcium/etidronate or calcitriol are available. Nevertheless the authority conditions for these drugs require the patient to have had a fracture.

It seems to me that on one hand the PBS is moving in the right direction in terms of preventative medicine. We now have few restrictions on COX-2 inhibitors which should reduce the gut ulceration caused by non-steroidal anti-inflammatory drugs. Yet we are not moving as fast with the bisphosphonates.

Phil Day

Pharmacist

Queen Elizabeth II Hospital

Brisbane

PBAC response:

Under current legislation, the PBAC can only recommend that a preparation be listed as a pharmaceutical benefit for those conditions in which use has been shown to be effective, safe and of reasonable cost-effectiveness. This ensures that the money the community spends in subsidising the PBS represents good value.

The subsidy of drugs used for the treatment of osteoporosis, such as alendronate sodium, disodium etidronate/calcium carbonate, calcitriol, and raloxifene, is limited to patients with osteoporosis who have experienced a fracture due to minimal trauma. This is because this is the only patient group in which cost-effectiveness has been demonstrated. To date, no manufacturer or other applicant has presented data to substantiate that these drugs are cost-effective in preventing osteoporotic fractures. Since the PBAC's decisions are evidence based, it cannot recommend a change to listing in the absence of the necessary supporting cost-effectiveness data.

Furthermore, the PBAC is aware of the importance of prevention of disease. It takes into account many factors in assessing the cost-effectiveness of a medication proposed for PBS listing. These include costs of hospitalisation or other medical treatments that may be required if the medication is not available, as well as less tangible factors such as patients' quality of life. If these preparations were to be listed for the primary prevention of fractures, the PBAC has decided (based on the evidence presented) that the benefits would be relatively small compared to the considerable cost of therapy.

Under the legislation on which the PBS is based, there is no provision for exceptions to be made to suit individual circumstances, even when the use of the drug may be beneficial, or where significant financial hardship is being incurred.

While I appreciate that this means the cost of alendronate will need to be borne as a private prescription, the Commonwealth Government has no control over the prices of non-PBS medicines.