would substantially reduce the incidence of serious injury. Thus we strongly recommend that all passengers should be adequately restrained in suitable safety devices.

This work was carried out under contract to the Transport and Road Research Laboratory, but the views expressed are ours and not necessarily those of the research laboratory or any part of the Department of the Environment, the Department of Transport, or other government department.

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(Accepted 5 August 1988)

Postnatal presentation of coeliac disease

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Coeliac disease in adults has an insidious onset; intestinal symptoms are minimal or even absent, and malabsorption and anaemia are the main symptoms.^{1,2} At our hospital two women who presented with coeliac disease in the postnatal period both had an acute diarrhoeal illness with evidence of severe malabsorption.

Case reports

Case 1—A 29 year old woman presented in June 1974, 16 weeks after her first delivery. She had had diarrhoea for eight weeks and intermittent acroparaesthesia and tetany for five days. She had breast fed her baby initially but stopped after two weeks. On admission she looked well nourished but had bilateral carpopedal spasm and Chvostek's and Trousseau's signs. Investigations confirmed a malabsorption state with hypocalcaemia (table). The electrolyte imbalance was corrected by intravenous treatment, and coeliac disease was diagnosed after jejunal biopsy. She was given a gluten free diet. Her symptoms resolved and haematological values returned to normal. One year later jejunal biopsy showed blunted villi and regression of inflammation consistent with treated coeliac disease.

Results of investigations in two women with coeliac disease

Investigation	Normal range	Case 1	Case 2
Haemoglobin (g/l)	115-160	115	114
Mean cell volume (fl)	81-100	78	70
Folate (mmol/l)	Serum 9.1-27.2	Serum 2.27	
	Red cell >227		Red cell 590
Sodium (mmol/l)	135-148	139	138
Potassium (mmol/l)	3.5-4.8	2.0	2.8
Albumin (g/l)	35-50	27	33
Calcium (mmol/l)	2.15-2.65	1.29	1.49
		1.54*	1.63*
Magnesium (mmol/l)	0.7-1.0	0.56	0.40
Average faecal fat (g/24 h)	<5	38	18

*Corrected (measured calcium + (40 - albumin) × 0.02).

Case 2—A 25 year old woman presented in December 1985, 10 weeks after her first delivery, having had watery diarrhoea for four weeks and acroparaesthesia and tetany for two days. She had been breast feeding since delivery. She looked well nourished but had bilateral carpopedal spasm and Chvostek's and Trousseau's signs. Initial investigations showed abnormalities similar to those in case 1 (table). In

addition, absorption of D-xylose, vitamin A, and vitamin B₁₂ was severely defective. Surprisingly, though she had been taking folate supplements during pregnancy, her red cell folate concentration was normal. The electrolyte imbalance was corrected with calcium, magnesium, and potassium salts given intravenously. An endoscopic duodenal biopsy showed changes typical of coeliac disease, and she was started on a gluten free diet. She remained well one year later, and results of repeat biopsy were consistent with treated coeliac disease.

Comment

The ratio of women to men who have coeliac disease is about 2:1, a difference that is due chiefly to a high proportion of cases in women of childbearing age. The supposition that this is the result of increased nutritional demands is consistent with the finding that the occurrence of anaemia and calcium malabsorption with coeliac disease is also more common in women.^{1,2} Pregnancy may precipitate the disease in some women and may reverse a remission in the teenage years experienced by women in whom the disease was diagnosed in childhood.⁴

In our patients pregnancy seemed to be the principal factor unmasking a predisposition to coeliac disease. By the criteria of maternal haemoglobin concentrations and weight gain and fetal development, however, they had met the nutritional burdens of pregnancy and lactation. Their disease presented as a diarrhoeal illness starting acutely at the end of the puerperium and characterised by fat malabsorption, hypocalcaemia, and hypokalaemia. The accompanying hypoalbuminaemia is unusual in coeliac disease and may merely have reflected its severity.

The distinctive presentation of coeliac disease in these two cases suggests an effect of pregnancy on women predisposed to the disease that is quite independent of depletion of nutrients. The onset was probably too long after pregnancy for the disease to have been precipitated by an endocrine mechanism, but changes in immune reactivity at this stage might have caused loss of borderline tolerance to gluten. This possibility could be tested by studying immune responsiveness to gluten before and after delivery in women with treated coeliac disease.

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(Accepted 10 August 1988)