

complementaemia at the beginning of the disease, while in the latter case it seems most unlikely that there could be 8 cases with a 'silent' course of chronic hypocomplementaemic persistent nephritis in a single group of patients. Apart from this, reduction of C3 concentration found in patients within a period of 4 to 6 months was only slight or at the lower limits of normal values (Table). In some patients slight falls in concentration

TABLE

Sequential determination of serum C3 levels (mg/100 ml) in 8 patients with acute poststreptococcal glomerulonephritis

Case no.	Week					Month			
	1	2	3-4	5-6	7-8	3	4	5-6	6-12
1		30					62	82	
2		27			70	82	71		
3	8		30	56	60	67	69	81	83
4	15		35	72	59	80	54		
5	8, 8	10	29					68	
6	11		33	66				66	
7		7	27		54			70	
8		31	53			86		56	

Note: Cases 1 to 4 with reduced C3 levels in 4th month of the disease, Cases 5 to 8 in 5th to 6th month. Normal values (mean \pm SD) 109.5 \pm 32.6 mg/100 ml.

occurred even after normal values were obtained. C3 concentration in patients with hypocomplementaemic chronic glomerulonephritis is usually markedly reduced. Similar findings in a previously biopsied group of patients with AGN were also found by Treser *et al.* (1969). 2 of their 23 patients had low C3 levels for 4 months, and another 2 showed transient depression in the 24th month after the onset of the disease.

The age of Case 8 (Table III) of my paper was misprinted as 15 years instead of 5, so that the average age of the patients in the postnephritic hypocomplementaemic group was 9.6 at subsequent testing. It is interesting to note that the average age of these patients at the onset of the disease was 5.8 years.

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Hyperglycaemia and uraemia in hyperosmolar dehydration

Sir,

I read with interest the report by Heggarty, Trindade, and Bryan (*Archives*, 1973, **48**, 740) concerning the hyperglycaemia which accompanies hypernatraemic dehydration. As was suggested by the authors, the metabolic acidosis may have contributed to the defective peripheral glucose utilization. However, we have shown (Nitzan and Zelmanovsky, 1968a) that hypernatraemia as such, of the degree encountered clinically, may have a diabetogenic effect. Rats rendered hypernatraemic by water deprivation and subcutaneous injections of hypertonic sodium solutions showed increased fasting blood glucose as well as a decreased ability to dispose of an exogenous glucose load, even in the presence of normal acid-base balance. Other contributory factors may be the severe uraemia shown by the dehydrated infants (O'Brien and Sharpe, 1965) and/or the intracellular potassium depletion which may be present in hypernatraemic states (Spergel *et al.*, 1967; Finberg, 1957). Hyperosmolarity has been reported to stimulate glucose uptake and ¹⁴C incorporation from ¹⁴C-labelled glucose into CO₂ in rat adipose tissue and diaphragm *in vitro* (Kuzuya, Samols, and Williams, 1965). In view of this observation, the possibility that the hypernatraemic glucose intolerance which was found *in vivo* might be causally related to a disturbed secretion of the hormones which regulate carbohydrate metabolism should also be considered. Since hypernatraemia may cause injury to the central nervous system (Finberg, 1959), further studies will be necessary to determine whether lesions of the hypothalamus play a role in the production of the glucose intolerance (Schoolman, Dubin, and Hoffman, 1955).

Uraemia invariably occurs in clinical syndromes associated with hypernatraemia (Schoolman *et al.*, 1955). The raising of urea levels can be explained by underperfusion of the kidneys due to the dehydration which often accompanies hypernatraemic states. In addition, severe degrees of hypernatraemia, even without circulatory disturbances, can induce definite histological damage in the renal tissue (Rush *et al.*, 1961). We have shown (Nitzan and Zelmanovsky, 1968b) that hypernatraemia exerts a catabolic effect leading to enhanced production of urea as the principal end product of protein metabolism. This excessive tissue catabolism may have contributed to the severity of the uraemia in the cases

described by Heggarty *et al.* Furthermore, the increased urea production associated with hypernatraemia may suggest that enhanced gluconeogenesis is one of the underlying mechanisms responsible for the hyperglycaemia in hyperosmolar dehydration.

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sensitivity is suspected. In the following case the decision to perform a jejunal biopsy was made when these studies were positive.

Case report. A 3-year-old girl presented in November 1972 with a history of 'wheezy bronchitis' from the age of 4 months. She had a cough with purulent sputum but her appetite was good and her stools were normal. On examination she had scattered crepitations and minimal finger clubbing. *Staphylococcus aureus* and *Haemophilus influenzae* were cultured from her sputum. Her sweat sodium was 101 mmol/l. and chloride 103 mmol/l., and the diagnosis of cystic fibrosis was made. Treatment was begun with pancreatic extracts, vitamin supplements, antibiotics, and physiotherapy. 7 months later during a relapse of her chest infection she was noted to have a moderately distended hypotonic abdomen, though gluteal atrophy was not evident.

Investigations. Hb 12.4 g/100 ml, WBC 11,800 mm³, normochromic, normocytic blood film, serum iron 87 mg/100 ml, TIBC 450 mg/100 ml, saturation 19%. Urea, electrolytes, liver function tests, calcium, phosphorus, and alkaline phosphatase were normal. Faecal fat excretion was increased at 8 g/24 hours. Immunoglobulins showed absent IgA with normal levels of IgM and IgG. Food protein antibody to cereal antigens, wheat, oatmeal, and gluten, and to ruminant antigens, cow's milk, and calf serum were persistently detected in the patient's blood. Jejunal biopsy showed partial villous atrophy with shortening, broadening, and branching of the villi. There was a satisfactory clinical improvement after gluten restriction.

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Cystic fibrosis and coeliac disease

Sir,

The coexistence of cystic fibrosis and coeliac disease has recently been reported (Hide and Burman, 1969; Goodchild, Nelson, and Anderson, 1973; Taylor and Sokol, 1973), and it has been suggested that cystic fibrosis may predispose to the later development of coeliac disease. The diagnosis of coeliac disease is largely dependent upon the demonstration of histological changes in the small intestine, but jejunal biopsy may be distressing to a small child. It is suggested that food protein antibody studies may be of value in the investigation of children with cystic fibrosis in whom gluten

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