prevalence of epilepsy of 0·5%.[3] The difference in the prevalence comparing the epilepsy between the two groups was significant ($P < 0.01$, χ^2 test).

Comment

This study has shown an increased prevalence of epilepsy, particularly temporal lobe epilepsy, among patients with coeliac disease. The possible reasons for this association are not clear and may well be multifactorial. These include genetic factors and calcium, magnesium, and vitamin deficiencies. Four patients had had low calcium and magnesium concentrations, but correction of these deficiencies some years before coeliac disease was diagnosed did not improve the control of their epilepsy in three out of the four. Similarly, correction of folate deficiency in eight out of nine patients did not improve their epileptic control. The improvement in epileptic control in three patients after starting a gluten-free diet may have been due to the correction of deficiencies both known and unknown, although improved anticonvulsant absorption may have played an important part. Selective immunoglobulin A deficiency has been reported in association with coeliac disease[1] and familial epilepsy,[5] but it was not present in any of our patients.

A large survey of epileptics[3] showed that only 20% had temporal lobe epilepsy, which highlights the unusually high prevalence (77%) of this type in our series. This remains unexplained.

We think that the demonstrated association between epilepsy and coeliac disease raises important clinical implications in the management of both conditions. Greater awareness of the association should lead to the improved diagnosis of both epilepsy and coeliac disease.

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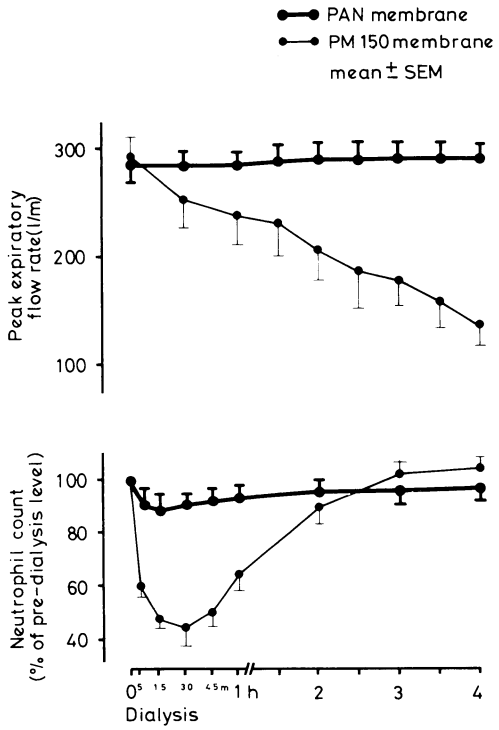
Haemodialysis-triggered asthma

Changes in respiratory function during haemodialysis using cellulose membranes include mild hypoxemia, variable hypocapnia, reduced carbon monoxide diffusing capacity, and increased lung closing volumes.[1] We describe a patient in whom haemodialysis was associated with severe asthma and leucopenia.

Case report

A 40-year-old housewife reached end-stage renal failure due to proliferative glomerulonephritis and began haemodialysis at home in 1972. She also had a three-year history of episodic attacks of dyspnoea, wheezing, and non-productive cough. She was a non-smoker and there was no personal or family history suggestive of atopy. Blood eosinophilia was absent and skin prick test results were negative to four common allergens; nevertheless, asthma had been triggered when she had taken salicylates, and intrinsic asthma had been diagnosed. Before dialysis her asthmatic symptoms had been mild and infrequent and usually associated with respiratory infection. Nevertheless, when dialysis began with a Cupruphane membrane (PM150) and a Meltec Multipoint 1 m^2 haemodialyser her asthma deteriorated. The usual pattern was that within an hour or so of starting haemodialysis the patient noticed chest tightness, dyspnoea, and wheezing, which became progressively worse as dialysis proceeded. On one occasion the FEV_1 changed from 1·82 to 0·97 litres and the FVC from 2·175 to 1·8 litres, after four hours' haemodialysis. She noticed that terminating dialysis early stopped the progression of symptoms but that she continued to wheeze for several hours after dialysis. Her general practitioner was frequently called on dialysis days to administer aminophylline and steroids. At no time had there been any clinical or radiological evidence of fluid overload or left ventricular failure. Treatment before and during dialysis with intravenous aminophylline and hydrocortisone or inhaled salbutamol and beclomethasone failed to prevent the dialysis-triggered attacks. Changing the osmotic pressure of her dialysate, changing the rate of ultrafiltration, and substituting Silastic for polyvinyl chloride lines were also ineffective. Substantial improvement followed a renal transplant in 1973 but her asthmatic symptoms reappeared when dialysis was started again after transplant rejection and nephrectomy in 1974. Some improvement was obtained with oral prednisone, 10 mg on dialysis days and 5 mg on other days, but dialysis was still frequently curtailed by dyspnoea.



Peak expiratory flow rate changes during dialysis (mean ± SEM of ten studies done with each membrane) and neutrophil count (mean ± SEM of percentage of change of predialysis values of three studies done with each membrane.

Formalin used as a sterilising agent was suspected[2] to be responsible but inhalation of formalin vapour failed to produce any symptoms or change in peak expiratory flow rate. Dramatic improvement occurred when the Cupruphane membrane was substituted by a polyacrylonitrile membrane (Rhône-Poulenc, RP6 dialyser). The figure shows the progressive reduction in peak flow during haemodialysis with Cupruphane membrane (PM150) compared with that of polyacrylonitrile (PAN); changes in neutrophil count are also shown. When, unknown to the patient, she was again dialysed with a Cupruphane membrane a dramatic reduction in peak flow was once again seen. She is now dialysing again at home with a polyacrylonitrile membrane (RP6 dialyser), a Bellco double-headed single-needle dialysis blood pump, and a Lucas Mark II proportionating unit; for economic reasons the RP6 dialyser is re-used.[3] She has been free from asthma for six months since using the RP6 and her prednisone has been reduced to steroid replacement dosage only.

Comment

Selective leucopenia affecting neutrophils and activation of complement occur during haemodialysis with cellulose membrane dialysers.[1] The leucopenia arises from sequestration of neutrophils in the pulmonary capilary bed.[1] There is evidence that complement is concerned in asthma, particularly when allergic factors are not prominent.[4] Our patient's asthma triggered only by Cupruphane-membrane dialysis could be due to either activation of complement or pulmonary leucostasis with subsequent release of bronchoconstrictor

substances from impacted neutrophils in the lungs. Since polyacrylonitrile membrane dialysers also activate complement but do not cause appreciable leucopenia,[3] it is more likely that the mechanism implicated in our patient was pulmonary leucostasis.

There are several other unexplained complications of dialysis which might be related to complement activation or leucostasis or both. They include arthralgia, abdominal discomfort, migraine, and ascites. Trials of polyacrylonitrile membrane (avoiding neutropenia) or polycarbonate membrane (avoiding complement activation) would seem worthwhile in these conditions.

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Osmotic fragility of erythrocytes in Duchenne muscular dystrophy

Duchenne muscular dystrophy is an X-linked recessive disorder affecting young boys. It is one of the more severe forms of muscular dystrophy and is transmitted by healthy female carriers. Onset is usually at 3-5 years and affected individuals become chairbound by the end of the first decade. Death occurs in the late teens or early twenties from cardiac failure or pneumonia. The basic defect in this disorder is as yet unknown but several recent studies[1-4] have suggested that there may be a generalised membrane abnormality affecting erythrocytes as well as muscle. Fisher *et al*[4] have reported an increase in the osmotic fragility in cases of what they refer to as "pseudohypertrophic muscular dystrophy." We report here our findings in a series of 10 patients with confirmed Duchenne muscular dystrophy and four definite carriers. (A definite carrier is defined as the mother of an affected boy with another affected male relative. The four carriers studied here had serum creatinine kinase levels of 83, 132, 213, and 344 IU/l (normal upper limit 85 IU/l).)

Patients, methods, and results

Heparinised samples of venous blood were obtained from patients (aged 1-18), their unaffected brothers (aged 1-16), and female carriers (aged 23-56) when the families attended the muscular dystrophy clinic in the department. Control samples were obtained from young boys (aged 4-15) before orthopaedic operations for disorders unrelated to neuromuscular abnormalities and healthy women volunteers (aged 17-67). The amount of lysis at various concentrations of sodium chloride was determined by the method of Dacie and Lewis.[5] The concentrations of sodium chloride resulting in 50% lysis of each sample were derived graphically.

Per cent lysis (mean ± SD) in male controls, boys with Duchenne muscular dystrophy (DMD), their unaffected brothers, female controls, and definite carriers

Sodium chloride concentration (g/l)	Male controls (n = 10)	DMD patients (n = 10)	Brothers (n = 5)	Female controls (n = 36)	Definite carriers (n = 4)
1·0	100	100	100	100	100
3·6	95·1 ± 2·9	94·4 ± 5·9	93·4 ± 6·0	95·5 ± 3·6	91·6 ± 2·8
3·8	90·7 ± 3·0	91·2 ± 7·2	83·4 ± 13·2	89·9 ± 8·5	86·5 ± 3·5
4·0	79·4 ± 12·5	83·4 ± 13·6	63·6 ± 18·3	78·8 ± 14·4	75·7 ± 11·7
4·2	50·7 ± 17·6	66·7 ± 18·3	38·5 ± 11·9	64·4 ± 18·1	49·5 ± 20·1
4·4	13·9 ± 9·9	34·0 ± 19·6	9·5 ± 2·1	40·4 ± 17·7	18·1 ± 14·9
4·6	4·5 ± 4·2	16·3 ± 13·0	4·6 ± 2·1	18·6 ± 11·3	5·8 ± 7·1
4·8	1·7 ± 1·8	6·9 ± 7·0	1·3 ± 0·7	6·8 ± 6·7	2·1 ± 3·3
5·0	0·0	1·3 ± 1·5	0·0	1·2 ± 1·9	0·0

Signs of lysis first became apparent in affected boys at a concentration of 5·0 g/l but in the controls and normal brothers at a concentration of 4·8 g/l. At all concentrations of sodium chloride from 4·4 to 5·0 g/l (see table) there was significantly greater lysis in affected boys than in either their unaffected brothers or controls ($P < 0.05$). The concentration (mean ± SD) of sodium chloride which resulted in 50% lysis was 4·18 ± 0·10 g/l for male controls, 4·30 ± 0·13 g/l for affected boys, and 4·08 ± 0·12 g/l for their unaffected brothers, the difference between affected boys and either their unaffected brothers or controls being statistically significant ($P < 0.05$). There was no difference in erythrocyte osmotic fragility between definite carriers and the control series of normal women.

Comment

These results provide additional evidence that there is a probable defect in the erythrocyte membrane in patients with Duchenne muscular dystrophy.

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Endoscopic removal of a swallowed ball bearing from stomach of a 4-year-old child

Foreign bodies commonly pass uneventfully through the gastrointestinal tract to be voided with the faeces. Thus patience and expectant treatment are usually recommended unless the size of the foreign body or its nature makes obstruction or perforation likely.[1] Hitherto in these circumstances removal by rigid endoscope or operation has been undertaken, but recently reports have described removal of various objects using fibreoptic endoscopes.[2] Grasping forceps and snares have been developed and ingenious techniques devised to retrieve objects with perforations or a waist—and even razor blades.[3] We were presented with a boy who had swallowed a ball bearing, and, as a suitable device was not available, we evolved a new method.

Case history

A fit West Indian boy aged 4 swallowed a steel ball bearing. Although well, he was brought to hospital by anxious parents. The results of examination were normal but radiographs of chest and abdomen showed a dense sphere 17 mm in diameter in the stomach. During the next six weeks he took a normal diet and did not vomit, yet serial radiographs showed the ball still in the stomach. As there was no sign of passage beyond the pylorus surgical removal was contemplated, but before laparotomy endoscopic removal was attempted.

A standard latex contraceptive condom was attached by cotton thread to the distal end of an Olympus GIFD2 endoscope. Three equally spaced ties were passed through the condom around the rolled rim and the free ends threaded through the lumen of the rubber hood detached from the