SHORT REPORTS

Serum N-acetyl- β -D-glucosaminidase and circulating immune complex activities in systemic lupus erythematosus

Systemic lupus erythematosus (SLE) is an autoimmune disease associated with the production of circulating immune complexes which are believed to contribute to the tissue damage. The mechanism of this damage in vivo is not yet established but in-vitro work indicates that these complexes (and also activated complement fragments, especially C3b) lead to the selective release of lysosomal enzymes from macrophages, which can then degrade tissue components.^{1,2} Preliminary studies in patients with active SLE seemed to suggest that, while the activity of a lysosomal marker enzyme, N-acetyl- β -D-glucosaminidase (NAG) was frequently raised, the immune complex concentration in serum was not always raised above normal. We therefore decided to study the relation between these two activities in a patient presenting with acute SLE to see whether any correlation would be observed during the course of the disease in an individual patient.

Case report

A 69-year-old man was admitted to hospital with a three-month history of an extensive macular erythematous eruption occurring mainly on light exposed areas of skin with purpuric spots on the palms, loss of weight, fever, and difficulty in walking. He had been taking hydrochlorothiazide daily for six months. Examination showed signs of acute SLE, with foot drop. Laboratory investigation gave the following results: haemoglobin 7.9 g/dl, white blood cell count $3.6 \times 10^9/l$, erythrocyte sedimentation rate 50 mm in first hour, antinuclear factor positive at 1/40 dilution, LE cells present, DNA-antibody binding 28% (normal up to 30%). The initial concentration of serum N-acetyl- β -D-glucosaminidase was 47 IU/l (normal range 12–30 in adults) (method based on that of Walker *et al*³) and of serum immune complex was 180 ng anti-IgG bound per 16.7 μ l serum (upper limit of normality for adult controls defined as mean + 2 standard deviations = 20 ng anti-IgG bound/16.7 μ l serum) (method of Hay *et al*⁴). Serum complement concentrations were C3 0.4 g/l (normal 0.7–1.7) and C4 0.2 g/l (normal 0.1–0.7). A skin biopsy specimen showed histological features compatible with acute SLE.

As the SLE was considered to be drug induced, the hydrochlorothiazide was discontinued and the patient started on prednisolone, 60 mg daily, whereupon the rash promptly cleared. Nevertheless, when the dose was reduced to 10 mg daily, the rash reappeared on the face and hands, but cleared once again when the dose was increased to 20 mg daily. Thereafter

the dose of prednisolone was reduced much more gradually without further mishap, so that after a six-month interval the patient remained well with a clear skin.

Comment

The time course study of the patient's circulating immune complex concentration and NAG activity shows that: (a) both activities were very high before the start of treatment; (b) there was a general parallelism between the two activities throughout the whole six-month period of observation; (c) a simultaneous increase of both activities heralded a recrudescence of the rash; (d) at the end of the observation period both activities were still above normal, even though the skin remained clear and the haemoglobin concentration and white blood cell count were normal.

We suggest that in patients with high concentrations of circulating serum immune complex activity the determination of serum NAG may be of value in monitoring the course of the disease. On this basis it should be possible to determine the severity of the disease and possibly to predict periods of relapse. Nevertheless, where a substantial proportion of the immune complex is no longer circulating—for example, fixed in the tissues—this relationship may not remain valid, in which case a high serum activity of NAG may occur together with a low circulating immune complex concentration.

¹ Cardella, C J, Davies, P, and Allison, A C, *Nature*, 1974, **247**, 46.

² Schorlemmer, H U, Davies, P, and Allison, A C, *Nature*, 1976, **261**, 49.

³ Walker, P G, Woolen, M E, and Pugh, D, *Journal of Clinical Pathology*, 1960, **13**, 353.

⁴ Hay, F C, Nineham, L J, and Roitt, I M, *Clinical and Experimental Immunology*, 1976, **24**, 396.

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Serial concentrations of serum NAG and immune complex in the patient with SLE.

New hand clamp for hand surgery

Conventional methods of hand restraint during surgical procedures are not entirely satisfactory, and the commonly used "lead hand" is rather cumbersome and lacks versatility. A hand restrainer has been designed, developed, and tested which is suitable for all forms of hand surgery.

The device

After investigation of materials capable of withstanding the process of autoclaving, we decided that a prepregated plastic base with securing straps of chloroprene rubber would be most suitable for cleaning and handling.

The base measures 20 cm square and is moulded to slope upwards away from the wrist (figure). A simple method of restraining the fingers and thumb has been incorporated using four rubber straps. A one-piece moulded strap with a high-density hard head and a less dense flexible prolongation was developed. The leads of the straps fit into the slots on the base, and the two side straps slide for adjustment. The end of the strap can be easily and instantaneously attached to studs in the base.

After cleansing the hand with antiseptic it is placed on the sterilised base and the wrist strap attached. The other straps are then adjusted in a convenient position and, if one particular finger is to be operated on, this can be lifted free from the transverse strap which restrains the other fingers. The

The instrument ready for use.

instrument has been found to be useful in all forms of hand surgery. It may easily be clamped to a hand table and is equally useful for operations performed under either local or general anaesthesia.

Comment

An instrument has been designed for securing the hand during surgery. Simple fastening on any size of hand, left or right, is permitted and access to either flexor or extensor surface is equally convenient. We have established the efficiency of the instrument in use.

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Spontaneous reversible renal artery stenosis after renal allotransplantation

Renal artery stenosis is a known complication of renal transplants. We report a patient who developed this complication which regressed spontaneously.

Case report

A 54-year-old man with glomerulonephritis received after 18 months of haemodialysis a renal cadaver allograft from a 19-year-old man. His blood pressure was always normal. Bilateral nephrectomy was performed before transplantation. There was one HLA-B locus incompatibility. The donor kidney, preserved with Collins's solution, had a duplication of the renal artery

on a single aortic patch. This was anastomosed end-to-side to the left external iliac artery without difficulties. Renal function improved rapidly. There were no symptoms of rejection. After four weeks the patient was discharged taking 30 mg prednisolone and 200 mg azathioprine daily. No anticoagulant or antiplatelet agents were given. Creatinine clearance was 95 ml/min, and blood pressure (BP) without salt restriction was 110/70 mm Hg. A soft bruit was heard over the graft.

Three months after operation his BP gradually increased to 160/110 and the creatinine clearance dropped to 75 ml/min. At no time did he have fever. There was no protein or fibrin split-products in the urine. The abdominal bruit had appreciably increased in intensity. Angiography showed a severe stenosis distal to the anastomosis in both renal arteries (fig 1a). The intrarenal vasculature was normal. The patient was treated with restricted salt intake and spironolactone 100 mg daily. When his BP remained high 1 g methyldopa, 160 mg propranolol, and 80 mg hydralazine chloride were added. The diastolic pressure decreased to 95 mm Hg, but creatinine clearance dropped to 65 ml/min with a renal plasma flow (RPF) of 220 ml/min. Plasma renin activity (PRA) was 13.8 nmol angiotensin I/l/h (18 ng/ml/h) before propranolol and 11.5 nmol/l/h (15 ng/ml/h) thereafter (normal limits 1.27 ± 0.59 nmol/l/h (1.6 ± 0.77 ng/ml/h)).

After three months' antihypertensive medication this treatment was stopped for several days. Surprisingly, his BP did not rise, but its maximum level was 160/95 mm Hg. The patient was kept under strict supervision without antihypertensive treatment. His BP decreased further and was normal one-and-a-half years after transplantation, even on unrestricted salt intake. At that time PRA was 1 nmol/l/h (1.3 ng/ml/h), RPF 325 ml/min, and creatinine clearance 110 ml/min. The bruit had decreased considerably. Angiography showed that both stenoses had decreased significantly (fig 1b).

Renal angiography (a) three months and (b) one-and-a-half years after transplantation.

Discussion

Several mechanisms have been held responsible for the development of renal artery stenosis in patients who have had renal transplants—for example, vessel damage by clamping, intimal dissection during renal perfusion, or reaction to suture material.^{1,2} Localised vascular rejection has also been postulated as causing stenosis,^{3,4} because in resected large arteries lesions have been seen similar to those observed in smaller renal vessels undergoing chronic rejection—for instance, cellular intimal proliferation and subintimal fibrosis.

Raphael⁵ described a patient who had had a transplant and developed hypertension and renal artery stenosis shown angiographically. No details were given, but apparently the stenosis resolved spontaneously within a year and the BP became normal. A kidney biopsy specimen showed the changes of rejection, but no mention was made of vascular changes.

The cause of both stenoses in our patient remains a matter of conjecture. Vascular injury at nephrectomy cannot be ruled out, although transplantation was uneventful and no signs of injury were visible. If the arteries had been damaged by clamping with resulting oedema and cell proliferation in the vessels, one would have expected an earlier onset of hypertension. If the hypertension had been the result of secondary fibrosis with narrowing of the lumen, spontaneous regression would have been highly unlikely.

The long period between transplantation and the onset of hypertension is consistent with a mild and presumably self-limiting rejection reaction against the donor vessel wall, but, apart from an impaired creatinine clearance and RPF and the hypertension, no other signs of rejection were observed. A kidney biopsy was not performed. Nevertheless, it is unknown whether local vascular rejection in the