complex nephritis. We report on a patient with miliary tuberculosis complicated by immune complex nephritis.

Case report

A 45 year old Indian clerk presented in January 1981 with chest pain. Results of physical examination, electrocardiography, and chest x ray examination were normal; urea concentration was 3.8 mmol/l (23 mg/100 ml) and creatinine 50 μ mol/l (565 μ g/100 ml); urine microscopy showed no abnormality and no protein. He presented again in July 1981 with a three week history of fever, night sweats, and weight loss (4 kg over six months). Results of examination were again unremarkable: blood pressure 150/100 mm Hg; urea concentration 14.6 mmol/l (87.8 mg/100 ml); creatinine concentration 256 μ mol/l (2.9 mg/100 ml); and creatinine clearance 30 ml/min (figure). Urine microscopy showed 40×10^6 red blood cells/l and no white blood cells; urine protein excretion was 1.3 g/24 hours. Chest x ray examination showed right basal and widespread miliary shadowing; results of intravenous pyelography were normal. Sputum, early morning urine, and gastric washings were negative on microscopy for tubercle bacilli. Mantoux testing yielded a negative result (1 in 100). C3 and C4 concentrations were normal. Liver biopsy showed epithelioid cell granulomas with central caseation; occasional tubercle bacilli were shown by Ziehl-Neelsen staining.

Four weeks later *Mycobacterium tuberculosis*, fully sensitive to anti-tuberculosis drugs, was isolated from sputum and liver samples. Renal biopsy after three weeks' treatment with isoniazid, pyrazinamide, and rifampicin showed focal proliferative glomerulonephritis with granular mesangial staining for IgA and IgM; intense staining for C3 was present in the mesangium and around capillary loops. Granulomas were not seen; amyloid stains yielded negative results. Circulating immune complexes were detected by macrogol (polyethylene glycol) assay at 190 μ g/l (normal < 100 μ g/l) but not by Clq binding assay; plasma C3d concentration, a measure of complement activation, was 25.5% (normal < 21.0% of a fully activated serum standard).² Circulating immune complexes and C3d concentrations fell and were normal after three months of antituberculosis treatment. The fever took five weeks to resolve. Prednisolone was added to the treatment after four months (figure).

Clinical course of treatment with antituberculosis drugs.

Conversion: SI to traditional units—Creatinine: 1 μ mol/l $\approx$ 11.3 μ g/100 ml.

Six months after diagnosis a second renal biopsy showed segmental scarring but no hypercellularity of glomerular tufts, and immunofluorescence testing yielded negative results. Fifteen months after diagnosis he was well and his renal function had improved: urea concentration was 6.8 mmol/l (40.8 mg/100 ml); creatinine concentration 167 μ mol/l (1.9 mg/100 ml); creatinine clearance 56 ml/min; 24 hour urine protein 0.2 g; and results of urine microscopy normal. Mantoux test results had become positive at 1 in 1000.

Comment

Patients with acute febrile illnesses may develop proteinuria that disappears on recovery. This "febrile proteinuria" has sometimes been ascribed to a non-specific response to the febrile state. In certain acute infections, however, renal biopsy has shown an immune complex nephritis.³ This does not appear to have been described in patients with tuberculosis.

It has been proposed that there is a clinical and immunological range of tuberculosis analogous to that of leprosy.⁴ Patients reactive to tuberculin and with localised lesions have neither free mycobacterial antibody nor immune complexes in serum; patients who are not reactive but have acute miliary disease have immune complexes and high levels of antibody.⁴ Our patient with immune complex

nephritis appears to be at the "lepromatous" end of the immunological spectrum whereas the patients of Mallinson *et al*¹ with localised reactive disease may be placed at the "tuberculoid" end.

Immune complex nephritis has been reported in patients with lepromatous leprosy.⁵ Such patients are not unlike our patient as they also show evidence of circulating immune complexes and complement activation and are unreactive to lepromin.

Dr M Wansbrough-Jones, St George's Hospital Medical School, kindly performed plasma C3d assays.

¹ Mallinson WJW, Fuller RW, Levison DA, Baker LRI, Cattell WR. Diffuse interstitial renal tuberculosis—an unusual cause of renal failure. *Q J Med* 1981;50(198):137-48.

² Perrin LH, Lambert PH, Mischer PA. Complement breakdown products in plasma from patients with systemic lupus erythematosus and patients with membranoproliferative or other glomerulonephritis. *J Clin Invest* 1975;36:165-76.

³ Rainford DJ, Woodrow DF, Sloper JC, de Wardener HE, Griffiths I. Post meningococcal acute glomerular nephritis. *Clin Nephrol* 1978;9:249-53.

⁴ Brostoff J, Lenzini L, Rottoli P, Rottoli L. Immune complexes in the spectrum of tuberculosis. *Tubercle* 1981;62:169-73.

⁵ Iveson JM, McDougall AC, Leatham AJ, Harris HJ. Lepromatous leprosy presenting with polyarthritis, myositis and immune-complex glomerulonephritis. *Br Med J* 1975;iii:619-21.

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Oxygen hazards in divers breathing helium and oxygen mixtures

The technology of hyperbaric life support has advanced over the past two decades with the growing commercial importance of diving in exploration for and production of oil from the sea bed. Related medical practice in the United Kingdom serves probably more than 2000 commercial divers, who work mainly for the North Sea oil industry. British regulations forbid the use of compressed air when diving below 50 m of seawater¹ and impose the need for an artificial atmosphere, typically of 40 kPa (0.4 bar) oxygen with the balance largely helium.² A diving operation is complex and requires teamwork to support divers inside a saturation chamber for 30 days or more. Divers depend on life support technicians, who control their environment and comfort from a console outside the chamber. There is an analogy between life support technicians and anaesthetists, both of whom are responsible for ensuring inter alia that their charges receive an appropriate partial pressure of inspired oxygen.

Case reports

Case 1—When the lock was opened after decompression of a chamber filled with a helium and oxygen mixture two divers were found unconscious on the chamber floor by a life support technician. He too collapsed on entering the chamber, and it was rapidly recompressed with air to 400 kPa (4 bars), with recovery of the occupants. Before the lock was opened air should have been piped into the chamber to displace the helium and oxygen mixture, but a helium valve had been opened before the seal was broken and pure helium, instead of air, had been piped in. The occupants had lost consciousness for some minutes owing to hypoxia, but their position on the floor may have kept them in a layer of helium and oxygen because its density is greater than that of pure helium.

Case 2—At a depth of 190 m of seawater a diver was working outside his diving bell and became alarmed when his colleague inside did not reply on the communications system. On returning to the bell he found his colleague semiconscious and slumped on the floor. He closed the hatch, and the bell was winched to the surface and locked to the deck decompression chamber of the diving support vessel in the usual way. Examination of the bell later showed a faulty tracking regulator in the oxygen supply line, which had resulted in undetected leakage of pure oxygen into the bell through the sensing line. The bellman had lost consciousness while squatting over the

hatch at the bottom of the bell, in which position his face was only inches away from the gas supply nozzle. On regaining consciousness he remembered having felt nausea, and he had bitten his tongue and had a bruise on his forehead. He appeared to have suffered a convulsion as a result of oxygen toxicity,³ as the partial pressure of inspired oxygen could have been nearly 2000 kPa (20 bars). Subsequent questioning excluded a history of epilepsy, and an electroencephalogram did not show any epileptic features.

Comment

The two cases show opposite extremes of partial pressure of inspired oxygen in hyperbaric conditions and could both have been fatal. The hypoxic incident might have led to further injury if the divers had fallen in the steel chamber and would not have happened if the lock had been opened immediately the chamber reached the surface. This would have allowed passive equilibration between the helium and oxygen in the chamber and atmospheric air through the hatch as is conventional practice. The accident highlights the danger of using cylinders containing 100% helium in diving. They are not necessary operationally, and the recommendation that diving gas offshore should be available only as compressed air or as prepared mixtures of helium and oxygen in the appropriate ratios deserves special consideration.⁴ The concept of premixed respirable gas is not new, and the need for ensuring a supply of oxygen is greatest when the administration of a respirable gas mixture is not under constant medical supervision.⁵ In contrast, the hyperoxic episode was due to mechanical failure and shows the importance of thorough maintenance and checking of equipment.

Both cases indicate that there may be an ergonomic need for improved monitoring and display of gas analysis. The use of audio alarms for high and low oxygen concentrations both at the console and in the diving bell would ensure that changes in ambient atmosphere are detected early. There is a good case for such a system becoming mandatory.

I thank the staff of the Institute of Environmental and Offshore Medicine, University of Aberdeen, and Offshore Medical Support for their encouragement.

¹ Anonymous. A guide to the diving operations at work regulations. *Health and safety series booklet HS(R)8*. London: HMSO, 1981. (Regulation 12(i) (g): schedule 6, para 123.)

² End E. The use of new equipment and helium gas in a world record dive. *Journal of Industrial Hygiene and Toxicology* 1938;20:511-20.

³ Donald KW. Oxygen poisoning in man. *Br Med J* 1947;i:667-72, 712-7.

⁴ James PB. Shallow water blackout at a thousand feet. *Pressure* 1980;9:8-9. (Newsletter of the Undersea Medical Society.)

⁵ Tunstall ME. Implications of premixed gases and apparatus for their administration. *Br J Anaesth* 1968;40:675-82.

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Chalazions: the frequency of spontaneous resolution

A chalazion is a "chronic inflammatory granuloma caused primarily by the retention of the secretion of a tarsal gland." Few published reports have mentioned its clinical course. We have always suspected that some would resolve without surgery, and indeed Duke-Elder and MacFaul stated that "in some early cases [the chalazion] may be absorbed and disappear."¹ A recent industrial dispute in the National Health Service caused a backlog to develop in our minor operations list, giving us an opportunity to review retrospectively the course of 87 chalazions.

Patients, methods, and results

We studied patients who had presented to our department during the dispute (May to November 1982) with one or more chalazions. They had

been asked to wait until the end of the dispute for incision and curettage. Sixty nine patients had been listed for surgery: 54 had one chalazion, 12 had two, and three had three. We reviewed the notes to see how many chalazions had resolved before surgery was undertaken.

Twenty two chalazions (25.3%) in 20 patients resolved, and 50 (57.5%) in 44 patients were incised and curetted. Eleven patients (with 15 chalazions (17.2%)) did not attend when requested and did not reply to a letter requesting the reason. It is probably safe to assume that in some of these the chalazions had resolved spontaneously.

The mean ages in the groups were similar (from 37.0 to 39.5 years), with an overall age range of 13-68 years. The duration of the complaint was measured as the time from the onset of symptoms to either surgery or confirmation that the lesion had resolved. This was a mean of 6.6 months

Duration of 72 chalazions

Duration (months)	Chalazions	
	Resolved (n = 22)	Incised and curetted (n = 50)
< 2	4	5
2-4	5	12
4-6	6	14
6-8	2	7
≥ 8	5	12

(range 1-28) in those who underwent surgery and 5.4 months (range 1.5-12) in those whose chalazions resolved.

Patients whose chalazions resolved were questioned about how much conservative treatment they had applied. Of the 10 who replied, nine had used a topical antibiotic or hot bathing, or both. None had followed an intensive regimen, most using drops or ointment two or three times a day for one to two weeks with only occasional application of heat.

The timing of surgery depended only on the duration of the complaint at the end of the industrial dispute. The table shows the course of events.

Comment

At least 22 chalazions (25%) and (including those of non-attenders) possibly 37 (43%) out of 87 resolved within a mean of six months from their onset, with only minimal conservative treatment. This is interesting in the light of three recent studies in which 50-90% success rates were obtained with more elaborate conservative treatments (intensive antibiotics, hot bathing, or intralesional corticosteroids).²⁻⁴

There was apparently no way of predicting from the information recorded in our notes which chalazions would resolve. The duration of the complaint was not a reliable guide to the likelihood of spontaneous resolution in the next two months (table). The decision whether patients with chalazions require referral to an eye department for surgery may therefore be based on the degree of discomfort from the lesion and the patient's attitude towards possible surgery, which is quick but uncomfortable. Many patients would probably prefer to wait at least six months before resorting to an operation and might therefore be managed by their general practitioner, perhaps with a trial of antibiotics and hot bathing. Those patients, however, who would rather be rid of their chalazion quickly by surgery may reasonably be referred early. Any atypical chalazion should be referred to exclude malignancy.

We thank the consultant staff of our unit for permission to study their patients and the secretarial staff for their invaluable help.

¹ Duke-Elder WS, MacFaul PA. *System of ophthalmology*. Vol XIII. London: Henry Kimpton, 1974:242-4.

² Bohigian GM. Chalazion: a clinical evaluation. *Ann Ophthalmol* 1979;11:1397-8.

³ Perry HD, Serniuk RA. Conservative treatment of chalazia. *Ophthalmology (Rochester)* 1980;87:218-21.

⁴ Pizzarello LD, Jakobiec FA, Hofeldt AJ, Podolsky MM, Silvers DN. Intralesional corticosteroid therapy of chalazia. *Am J Ophthalmol* 1978;85:818-21.

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