choroidal lesions with blurred vision in the right eye due to macular oedema. This gradually improved, though visual acuity remained somewhat reduced due to chronic posterior uveitis. In August 1974 chest radiographs showed peripheral lung mottling and her maintenance dose of corticosteroid was increased to 10 mg daily.

Discussion

Heerfordt's syndrome is an unusual manifestation of sarcoidosis consisting of parotitis with chronic or subacute uveitis and often complicated by cranial nerve pareses, usually of the facial nerve. Originally described in 1909,[1] it was not recognised as a manifestation of sarcoidosis until 1937. The complete picture is rare. Scadding[2] had no cases of the complete syndrome in his series, and Greenberg *et al*,[3] reviewing 388 cases of sarcoidosis, reported only 8 with uveoparotitis and only one with a facial palsy. Familial association in sarcoidosis is well recognised.[4] In 13 instances of sarcoidosis affecting identical twins the manifestations of the disease in each twin pair tended to be similar.[5] This seems to be true of less closely related subjects. Interestingly, both our cases presented almost simultaneously with a most uncommon form of sarcoidosis and followed very similar courses.

[1] Heerfordt, C F, *Albrecht v Graefes Archiv für Ophthalomolgie*, 1909, **70**, 254.
[2] Scadding, J G, in *Sarcoidosis*. London, Eyre and Spottiswoode, 1967.
[3] Greenberg, G, *et al*, *British Medical Journal*, 1964, **2**, 861.
[4] Research Subcommittee of British Thoracic and Tuberculosis Association, *Tubercle*, 1973, **54**, 87.
[5] Selroos, O, *et al*, *American Review of Respiratory Diseases*, 1973, **108**, 1401.

Department of Thoracic Medicine, Battle Hospital, Reading
N J C SNELL, MB, BS, medical registrar
A J KARLISH, MD, FRCP, consultant physician

Tear fluid lysozyme concentration: guide to practolol toxicity

By the end of 1974 the Committee on Safety of Medicines had reported 187 cases of diminished tear secretion and conjunctivitis or corneal damage associated with long-term practolol therapy.[1] Wright, reporting toxic effects of practolol, stated that several out of 27 patients had low levels of tear lysozyme.[2] We report here the tear fluid lysozyme concentrations in 30 patients who had been taking practolol over a long period.

Patients, methods, and results

Of the 30 patients examined four had taken practolol for 6 months, the remaining 26 had taken it for at least a year. Ten showed signs of ocular toxicity and 20 showed no signs. Eight of the 10 toxic patients had been off practolol for periods varying from 1 to 12 weeks (average 6·4 weeks). We assayed the lysozyme concentration in the tear fluid of both eyes of all the patients by our quantitative method with calibrated standards,[3] the measurements being in units of activity/μl.

All the 10 toxic patients had tear lysozyme concentrations below normal in one or both eyes (see table). In one patient (case 2) lysozyme was absent. In 3 of the 10 patients (cases 1, 3, 7) the concentration in one eye was about three times that of the other, and in one patient (case 4) it was 9 times. These differences are outside normal limits of variation between the two eyes.[3]

Of the 20 patients with no signs of toxicity 15 had normal tear lysozyme concentrations (see table). One (case 19) had low concentrations in both eyes. Three (cases 11, 14, 26) had low concentrations in one eye, but the differences in concentrations between the two eyes were within normal limits. In one patient (case 17) the initial concentrations were 201 U/μl right and 57 U/μl left. A month later they were 16 U/μl and 25 U/μl respectively—a striking fall. A month after that (in January) the patient developed signs of toxicity and practolol was stopped. In May the concentrations had risen to 40 U/μl and 45 U/μl, and in July to 120 U/μl and 60 U/μl.

We also examined two patients with sclerosing peritonitis due to practolol but with no signs of ocular toxicity. Both had abnormally low levels of tear lysozyme (45 U/μl and 55 U/μl, and 30 U/μl and 36 U/μl respectively). One, in whom lysozyme levels fell further, later developed signs of ocular toxicity.

Lysozyme concentrations in tear fluid in 30 patients on long-term practolol treatment

Case No	Age	Sex	Lysozyme concentrations (U/μl)		
			Right eye	Left eye	Lowest normal for age
			Toxic patients		
1	47	F	45* 63†	18* 21†	59
2	52	F	—	—	56
3	54	M	81	33	55
4	57	F	54	6	54
5	58	F	12	9	54
6	62	F	12	24	51
7	64	M	78	24	50
8	67	F	33	10	49
9	68	M	48‡ 21§	42‡ 27§	48
10	75	F	15	6	45
			Non-toxic patients		
11	16	M	91	57	72
12	41	F	93	75	61
13	43	M	68	98	60
14	48	F	57	48	58
15	48	M	114	153	58
16	48	F	66	57	58
17	50	F	201	57	56
18	52	M	243	177	56
19	56	F	42	45	54
20	57	F	70	65	54
21	59	F	116	118	53
22	61	F	123	96	52
23	63	F	57	62	51
24	65	M	108	213	50
25	66	F	87	60	49
26	72	M	39	63	46
27	74	F	93	141	45
28	75	F	102	108	45
29	77	F	234	168	43
30	79	M	99	51	42

*Three weeks after practolol stopped.
†Seven weeks after practolol stopped.
‡One week after practolol stopped.
§Seven weeks after practolol stopped.

Comment

The tear lysozyme concentration could be useful as a screening test for toxicity in patients taking practolol, since the level may fall before signs of ocular toxicity appear. We are continuing this work.

We thank Mr P Wright for his advice and help, Dr D G Fleck for his continued support, and Mr J D Pescod for his technical help.

[1] Committee on Safety of Medicines, *Adverse Reactions Series*, 1975, No 11.
[2] Wright, P, *British Medical Journal*, 1975, **1**, 595.
[3] Mackie, I A, and Seal, D V. Paper awaiting publication.

Public Health Laboratory and St George's Hospital, London SW17
I A MACKIE, MB, DO, clinical assistant, department of ophthalmology
D V SEAL, MB, DIPBACT, registrar

Guillain-Barré syndrome in acute HBs Ag-positive hepatitis

In acute viral hepatitis (AVH) several neurological complications have been described. The central[1] and the peripheral[2] nervous systems may be affected, but the Guillain-Barré syndrome (GBS), characterised by progressive symmetrical pareses, sensory loss, and a raised spinal fluid protein level with normal cell count, is extremely rare in the course of AVH.[3] We report here on a patient who developed hepatitis B and GBS and in whom we were able to define accurately the relation between the onset of hepatitis B and the onset of GBS.

Case report

The patient was a 21-year-old nurse seronegative for hepatitis B surface antigen (HBsAg) in a haemodialysis unit with HBsAg (subtype adw)-positive patients. The clinical course of her disease is summarised in the figure. On