over the next 5 months, visual acuities remaining at 6/9 in both eyes. The tattoo swelling persisted until surgical excision.

Ocular examination revealed fine keratic precipitates and moderate anterior chamber activity with cells and flare. There was no evidence of vitreous involvement, or retinal vasculitis. Results of routine investigations including a full blood count, erythrocyte sedimentation rate, VDRL and TPHA tests, chest radiograph and sacroiliac joint radiograph were all within normal limits and there were no clinical signs of an underlying systemic disorder.

Excision biopsy of the skin tattoo revealed a 'sarcoid type' allergic granulomatous reaction to the tattoo dye in the upper dermis with an overlying subcorneal pustule (Fig. 2). Following excision, there was no further recurrence of uveitis.

Discussion

Tattoos are composed of pigment containing metallic compounds which may provoke a sensitisation reaction. Metals commonly found in tattoo pigment include mercury (red), chromium and titanium (green), copper (blue) and iron (yellow and brown).[3]

Delayed hypersensitivity reactions to the metallic component may occur, with the histological appearance varying from diffuse lymphohistiocytic infiltrate[3] to pseudolymphomatous reactions,[4] lichenoid reactions[5] and sarcoidal granulomas.[6]

Buechner and associates[7] suggested that helper T cells are important in the formation of sarcoid granulomas by mononuclear phagocytes, and that duration and activity of the disease process may be related to T cell populations.

Hanada and associates[1] reported a case in which symptoms of systemic sarcoidosis and concurrent uveitis developed in a 31-year-old man following extensive tattooing. Histological examination of the skin lesions, regional lymph nodes and lung tissue revealed non-caseating granulomas and, in addition, microscopy of the lung specimens showed fragments of red tattoo granules. They concluded that tattoo pigments were responsible for the sarcoidal granulomas, as all lesions appeared following the tattooing process.

Mansour and Chan[2] recently reported a case of recurrent bilateral uveitis in a 35-year-old man with extensive skin tattoos. Biopsy of the tattoos revealed non-necrotising granulomas surrounding pigment granules. Immunopathology of the lesions during the phase of acute swelling showed nests of infiltrating cells in the dermis, consisting mainly of T and B lymphocytes and macrophages. Ninety per cent of the infiltrating cells stained positive for major histocompatibility complex class 2 antigens. In this case there was a high ratio of B lymphocytes and macrophages with equal numbers of T-helper and T-suppressor cells characteristic of delayed-type hypersensitivity, in contrast to sarcoidosis.[7,8]

Our patient showed no evidence of active sarcoidosis, but 'sarcoid type' granulomas were evident in the tattoo biopsy. It is probable that the concurrent tattoo eruption and uveitis were related to the sensitising nature of the metallic component of the tattoo dye.

A. M. McElvanney
S. M. M. Sherriff

Eye Department
Royal Preston Hospital
Sharoe Green Lane
Preston PR2 4HT
UK

References

1. Hanada K, Chiyoya S, Katabira Y. Systemic sarcoidal reaction in tattoo. Clin Exp Dermatol 1985;10:479–84.
2. Mansour AM, Chan C-C. Recurrent uveitis preceded by swelling of skin tattoos. Am J Ophthalmol 1991;111:515–6.
3. Slater DN, Durrant TE. Tattoos: light and transmission electron microscopy studies with X-ray microanalysis. Clin Exp Dermatol 1984;9:167–73.
4. Blumental G, Okun MR, Ponitch JA. Pseudolymphomatous reaction to tattoos: report of three cases. J Am Acad Dermatol 1982;6:485–8.
5. Taaffe A, Knight AG, Marks R. Lichenoid tattoo hypersensitivity. BMJ 1978;1:616–8.
6. Goldstein A. Histologic reactions to tattoos. J Dermatol Surg Oncol 1979;5:896–900.
7. Buechner SA, Winkelman RK, Banks PM. T-cell subsets in cutaneous sarcoidosis. Arch Dermatol 1983;119:728–32.
8. Chan C-C, Wetzig RP, Palestine AG, Kuwabara T, Nusenblatt RB. Immunohistopathology of ocular sarcoidosis: report of a case and discussion of immunopathogenesis. Arch Ophthalmol 1987;105:1398–402.

Sir,

Coexistent Optic Disc Pit and Ocular Hypertension Misdiagnosed as Glaucoma

Optic disc pit is an uncommon congenital malformation of uncertain embryological origin.[1,2] It is associated with other congenital defects such as retinal dysplasia, retinochoroidal colobomata, ocular vascular anomalies and midline neurological abnormalities.[3,4] Optic disc pits are usually asymptomatic unless associated with macular disease,[3] but unless the examiner is familiar with their appearance they may present a diagnostic challenge.

Case Report

A 60-year-old man presented with left central visual clouding. He had no significant past history and no relevant family history. The visual acuities were 6/6, the anterior segments and gonioscopy were unremarkable, but the intraocular pressures were 24 mmHg right and 26 mmHg left. The right fundus and disc were normal, but an inferotemporal excavation of the neural rim of the left disc was noted which was thought to represent glaucomatous cupping. Central perimetry revealed an apparent left arcuate scotoma with a normal right field. Left primary open angle glaucoma and right ocular hypertension were diagnosed and treatment was commenced with guttae timolol 0.25% to both eyes.

However, during follow-up, the disc appearance was reviewed and thought to be an optic disc pit 0.28 disc dia-

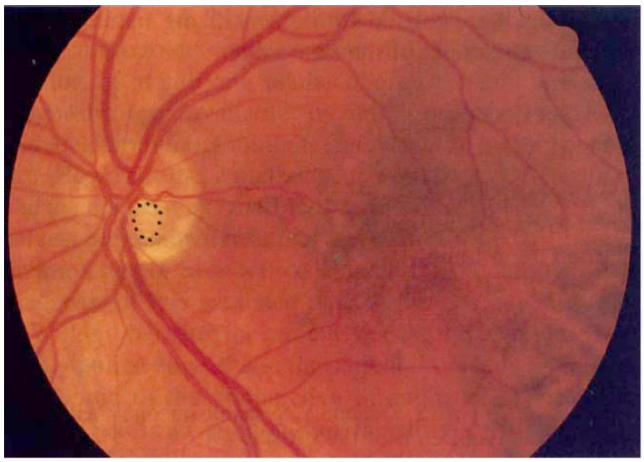

Fig. 1. *Fundus photograph of the left eye shows an inferotemporal oval optic disc pit (margin outlined).*

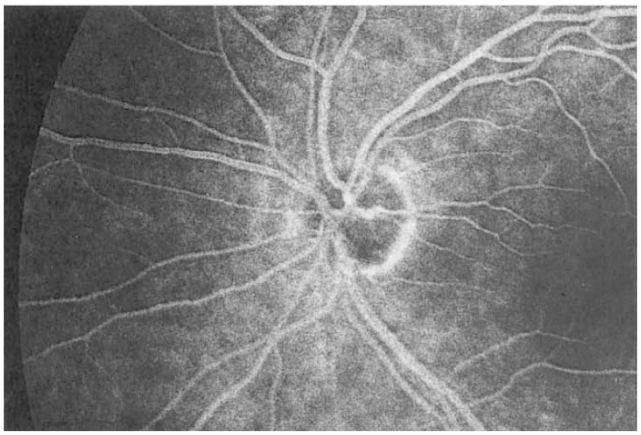

Fig. 2. Fluorescein angiogram of the left eye reveals a hypofluorescent area in the inferotemporal disc corresponding to the optic disc pit. Hypofluorescence was maintained throughout the run. The macula appears normal.

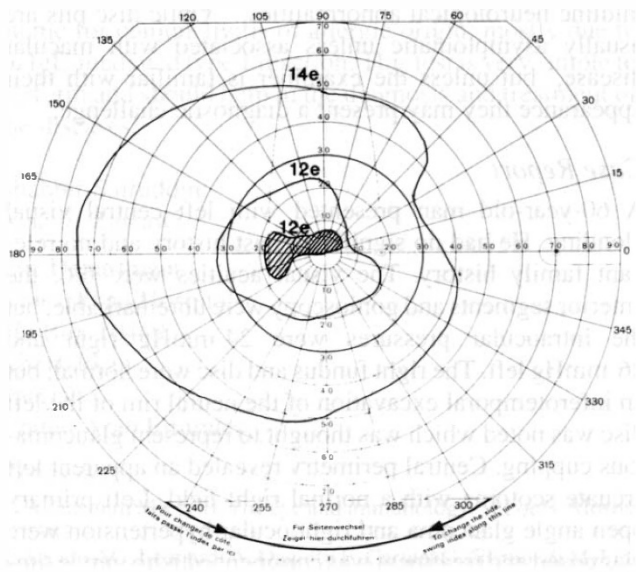

Fig. 3. *Goldmann perimetry of the left eye showing an enlarged blind spot connected to a centrocaecal scotoma. There is also a constriction of the peripheral nasal field.*

meters wide and 3 dioptres deep with a normal macula (Fig. 1). Fluorescein angiography confirmed this and highlighted the pit as an area of inferotemporal hypofluorescence (Fig. 2). Careful Goldmann perimetry revealed a centrocaecal scotoma and enlarged blind spot with a nasal step (Fig. 3). Computed tomography of the head was normal. The diagnosis was revised to ocular hypertension and during follow-up the disc appearance and visual fields have not changed.

Discussion

Optic disc pits are usually small, single, unilateral, well-circumscribed depressions in the inferotemporal optic nervehead and may be oval, circular or triangular.[1,4] The width ranges from 0.1 to 0.7 disc diameters, the depth from 0.5 to 25 dioptres (average of 5 dioptres) and the colour varies from black to grey-olive to yellow.[1,4] The affected disc is often larger and may show peripapillary chorioretinal atrophy.[4] Fluorescein angiography shows early hypofluorescence and, in 50% of cases, a late hyperfluorescence, particularly in those with macular disease.[4]

Optic disc pits usually present with the maculopathy found in 60% of these patients.[1,4] Classically, serous retinal detachment of the macula occurs but macular oedema, cystic retinal changes, haemorrhages, full-thickness or partial-thickness holes and mottled retinal pigment epithelial changes are recognised.[1,4] Theories proposed for the origin of the subretinal fluid include: a connection with the cerebrospinal fluid or liquefied vitreous through the pit; leakage from pit or choroidal blood vessels; an abnormal development of papillomacular nerve fibres with a lowered resistance to later insults; traction from glial elements in the pit floor.[5,6] Laser photocoagulation has been used in treatment with inconsistent results.[5,6]

Although usually asymptomatic, 60% of those with a disc pit and a healthy macula have some visual field defect, including arcuate scotoma, enlarged blind spot, central, paracentral and centrocaecal scotomata, localised peripheral constrictions and steps and sectorial defects.[4] These may not be consistent with the size and location of the pit and may represent an anomaly of the retinal nerve fibres.[7]

The differential diagnosis includes glaucoma, optic nerve tumour, optic neuritis and optic atrophy. There have been 7 patients cited in passing in the literature in whom glaucoma was suspected.[4,7–9] In these cases the diagnosis of glaucoma was excluded by the lack of raised intraocular pressure, including during phasing and after provocation testing, and the failure of progression of field defects with time. In our patient confusion arose because of coexisting ocular hypertension. However, the field disturbance was not typically glaucomatous. This patient was unusual in that the field defect was symptomatic despite an absence of macular involvement. Optic disc pit is a clinical diagnosis and differentiation of this appearance from glaucomatous optic disc cupping in the occasional confusing patient requires the vigilance and awareness of all clinical examiners within whose remit disc assessment may fall.

M. Hingorani, FRCOphth
D. Mooney, MRCOphth
A. Singh, FRCS

Department of Ophthalmology
North Middlesex Hospital
Sterling Way
London N18 1QX
UK

References

1. Kranenberg EW. Crater-like holes in the optic disc and central serous retinopathy. Arch Ophthalmol 1960;64:912–24.
2. Brown GC, Ausberger JJ. Congenital pits of the optic nerve head and retinochoroidal colobomas. Can J Ophthalmol 1980; 15:144–6.
3. Ferry AP. Macular detachment associated with congenital pit of the optic nerve head. Arch Ophthalmol 1963;70:346–57.
4. Brown GC, Shields JA, Goldberg RE. Congenital pits of the optic nerve head. II. Clinical studies in humans. Ophthalmology 1980;87:51–65.
5. Theodossiadis G. Evolution of congenital pit of the optic disk with macular detachment in photocoagulated and non-photocoagulated eyes. Am J Ophthalmol 1977;84:620–31.
6. Gass JDM. Serous detachment of the macula secondary to congenital pit of the optic nervehead. Am J Ophthalmol 1969; 67:821–41.
7. Petersen HP. Pit or crater-like holes in the optic disc. Acta Ophthalmol (Copenh) 1958;36:435–43.
8. Neame H. Congenital hole at the optic disc: report of ten cases with especial reference to visual field defects. Trans Ophthalmol Soc UK 1947;57:109–32.
9. Rosen E. Crater-like holes in the optic disc. Br J Ophthalmol 1948;32:465–78.

Sir,

Primary Orbital Lymphoma Presenting as Epiphora

We present a case of epiphora caused by compression of the nasolacrimal sac by a primary orbital lymphoma. This is both a rare cause of a common presenting symptom and an unusual presentation of such a tumour. The history, management and histology are described and their implications discussed.

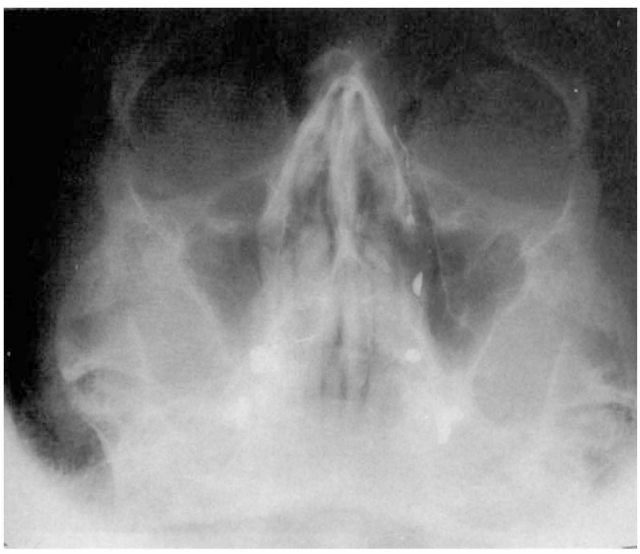

Fig. 1. *Dacryocystogram showing a patent and medially compressed left nasolacrimal sac.*

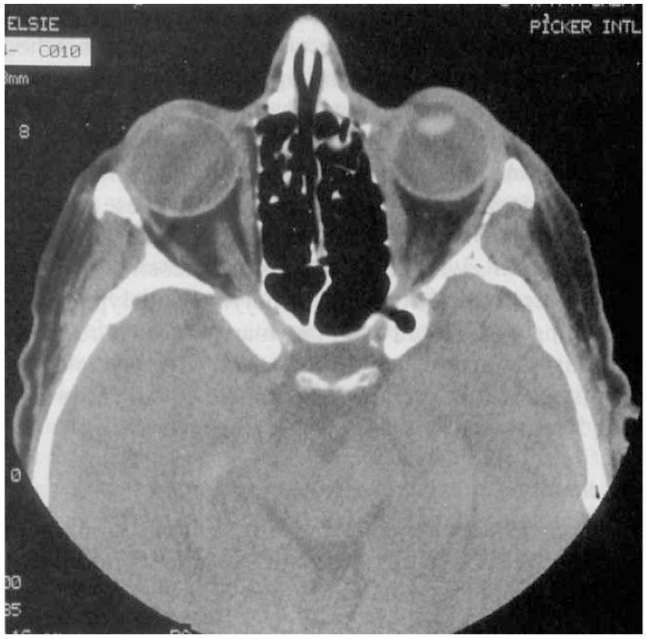

Fig. 2. *CT scan showing a left-sided orbital tumour extending over the orbital rim.*

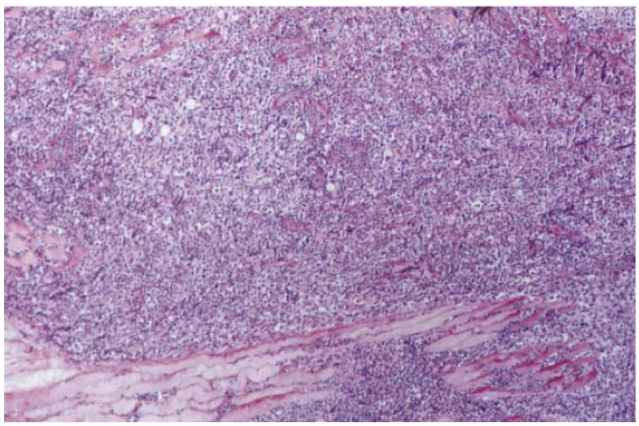

Fig. 3. *Histology shows a mass of lymphoid cells infiltrating between muscle fibres from the orbital tissue. (Haematoxylin & eosin, × 160).*

Case Report

A 68-year-old woman presented with a 6 month history of epiphora from the left eye and a 6 week history of a mass at the medial canthus. Examination confirmed the presence of a firm, non-compressible swelling extending over the medial orbital rim, which exhibited no reflux. The patient was otherwise asymptomatic and had no other medical problems.

A dacryocystogram was performed which demonstrated a patent but medially compressed nasolacrimal sac (Fig. 1), and a CT scan showed that this was due to compression by an expanding orbital tumour (Fig. 2). This lesion was then biopsied under a general anaesthetic by dividing the overlying skin and fibres of orbicularis to reveal its surface from which samples were taken.

Macroscopically the two biopsied tissue fragments, the larger 5 mm in diameter, consisted of muscle and connective tissue with a white infiltrate. Microscopically both