MVL between the ETDRS immediate treatment arm and our patients ($P = 0.11$). No patient underwent fluorescein angiography.

Comment

The recent UK audit of diabetic maculopathy treatment found that 9.2% of patients doubled their visual angle by 9 months.[2,3] Our outcomes were more positive, possibly as a result of these being newly diagnosed cases. This was achieved without fluorescein angiography. There is little evidence that patients having fluorescein angiograms have better acuity outcomes than individuals treated on clinical grounds alone. A randomized controlled trial to conclusively demonstrate visual benefit would be prohibitively expensive.[6] Could other noninvasive methods of assessing macular morphology such as optical coherence tomography be used to target laser treatments in the early stages of the disease?

At present, UK audit standards for diabetic maculopathy treatment require assessment of waiting times and access to treatment but not of the visual outcome. This audit demonstrates that measurement of visual outcomes can be achieved, and are essential for the assessment of retinal services, from screening to treatment.

References

1 Early Treatment of Diabetic Retinopathy Study Group. Study 1. Photocoagulation for diabetic macular oedema. *Arch Ophthalmol* 1985; **103**: 1796–1806.
2 Bailey CC, Sparrow JM, Grey RH, Cheng H. The National Diabetic Retinopathy Laser Treatment Audit. I. Maculopathy. *Eye* 1998; **12**(Part 1): 69–76.
3 Bailey CC, Sparrow JM, Grey RH, Cheng H, The National Diabetic Retinopathy Laser Treatment Audit. II. Proliferative retinopathy. *Eye* 1998; **12**(Part 1): 77–84.
4 Bailey CC, Sparrow JM, Grey RH, Cheng H, The National Diabetic Retinopathy Laser Treatment Audit III. Clinical outcomes. *Eye* 1999; **13**(Part 2): 151–159.
5 Bourne RR, Rosser DA, Sukudom P, Dineen B, Laidlaw DA, Johnson GJ *et al*. Evaluating a new logMAR chart designed to improve visual acuity assessment in population-based surveys. *Eye* 2003; **17**(6): 754–758.
6 Kylstra JA, Brown JC, Jaffe GJ, Cox TA, Gallemore R, Greven CM *et al*. The importance of fluorescein angiography in planning laser treatment of diabetic macular edema. *Ophthalmology* 1999; **106**(11): 2068–2073.

NE Sinclair, A Booth, A Clover and R Newsom

Southampton Eye Unit, Southampton Hospitals NHS Trust, Southampton SO16 6LD, UK

Correspondence: NE Sinclair,
Tel: +44 (0) 1794 514977;
Fax: +44 (0) 1794 514977.
E-mail: sinclne@hotmail.com

Eye (2006) **20,** 1471–1472. doi:10.1038/sj.eye.6702361; published online 12 May 2006

Sir,

Horner's syndrome: an atypical presentation in a child with neurofibromatosis type 2

Atypical manifestation of neurofibromatosis type 2 (NF2) in a 10-year-old boy was recently reported in *Eye*.[1] I would like to present another 10-year-old child with the atypical presentation of Horner syndrome. Although a diagnosis of NF2 is most often made in adults, both cases highlight that onset is also possible in childhood. Further, eye manifestations may be the presenting symptoms.

Case report

Seven years ago, a 10-year-old girl was referred for ophthalmic opinion as part of a general investigation because of heredity of NF2 in her father and half-brother. At that time there was no multidisciplinary follow-up program of relatives of NF2 patients in our hospital and her eyes had not previously been examined. Audiology examination had not revealed any vestibular schwannomas. She had no eye complains. Visual acuity was normal (6/6) both eyes. The anterior segment was normal in her right eye, but there was a posterior subcapsular opacity in her left eye, not affecting the visual acuity. No Lisch noduli were found. Horner's syndrome was noted in the left eye with a moderate ptosis and an anisocoria, most prominent in dark. Both fundi were normal. She had no strabismus.

The girl was investigated by the paediatric neurologists, and a magnetic resonance imaging (MRI) scan revealed an intraspinal/thoracic tumour with an extension from CVI to ThII and extrapleurally left side (Figure 1). The tumour was operated in two sessions when she was 11 years old. Histological examination showed neurofibroma. Minor vestibular schwannomas were detected at 15 years of age and are followed up, but still not operated at the age of 17 years.

The girl is followed up regularly by audiologist, paediatric neurologist, and ophthalmologist. Her posterior subcapsular cataract remains similar and she still has a normal visual acuity in both eyes.

NF2 is an autosomal, dominantly inherited disease and is associated with mutations in the NF2-gene, localized to chromosome 22.[2] It is characterized by bilateral

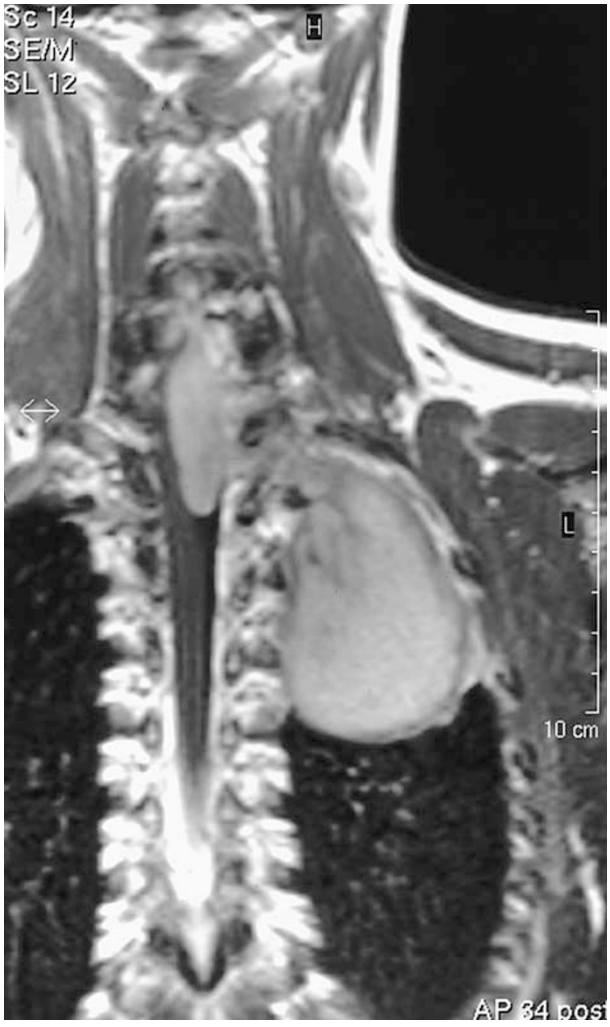

Figure 1 Thoracic MRI showing an intraspinal/thoracic tumour with an extension from CVI to ThII and extrapleurally left side (with permission of the patient).

vestibular schwannomas, that is, 'acoustic neuromas'. Other central nervous tumours may also occur, such as meningeomas of the brain, schwannomas of other cranial nerves, spinal nerve roots, and peripheral nerves and gliomas.[3,4]

Various eye manifestations are described in patients with NF2, the most common being cataracts that are described in up to 80%.[5,6] Cataracts often have an early onset and may even be congenital.[3] Other ophthalmologic manifestations include retinal hamartomas and epiretinal membranes, choroidal hamartomas, optic meningeomas and gliomas, conjunctival neurofibromas, eye muscle pareses, and strabismus.[3,5–7] Reduced corneal sensibility owing to fifth nerve lesions and corneal opacities owing to facial nerve palsy may also occur. Lisch noduli are, however, rare.

Comment

A case of Horner syndrome in a patient with NF1 was previously reported in *Eye*.[8] To my knowledge, however, this patient is the first reported case of NF2 presenting with Horner syndrome. As spinal tumours are frequent in NF2, this is an ophthalmologic manifestation that ought to be kept in mind.

Patients with NF2 are most often diagnosed as adults, but 10% have their onset before 10 years.[3] NF2 must therefore be considered as a differential diagnosis also in children. In 13% of patients with NF2, eye manifestations were the presenting symptom.[9] This underlines the importance of including ophthalmologic examination in multidisciplinary screening programs of first-degree relatives of patients with NF2 as well as in follow-up of patients with established NF2. Eye examinations are particularly important for early diagnosis, which may contribute to a better outcome after surgery for vestibular schwannomas. Further, early detection and treatment of eye manifestations may also lead to prevention of amblyopia.

References

1 Bosch MM, Mironov A, Killer HE. Atypical presentation of neurofibromatosis type 2 in a boy. *Eye* 2005; **19**: 705–706.
2 Rouleau GA, Wertelecki W, Haines JL. Genetic linkage analysis of bilateral acoustic neurofibromatosis to a DNA marker on chromosome 22. *Nature* 1987; **329**: 246–248.
3 Evans DG, Huson SM, Donnai D, Neary W, Blair V, Newton V *et al*. A clinical study of type 2 neurofibromatosis. *Q J Med* 1992; **84**(304): 603–618.
4 Parry DM, Eldridge R, Kaiser-Kupfer MI, Bouzas EA, Pikus A, Patronas N. Neurofibromatosis 2 (NF2): clinical characteristics of 63 affected individuals and clinical evidence of heterogeneity. *Am J Genet* 1994; **52**(4): 450–461.
5 Bouzas EA, Freidlin V, Parry DM, Eldridge R, Kaiser-Kupfer MI. Lens opacities in neurofibromatosis 2: further significant correlations. *Br J Ophthalmol* 1993; **77**(6): 354–357.
6 Ragge NK, Baser ME, Klein J, Nechiporuk A, Sainz J, Pulst SM *et al*. Ocular abnormalities in neurofibromatosis 2. *Am J Ophthalmol* 1995; **120**(5): 634–641.
7 Evans DG, Birch JM, Ramsden RT. Paediatric presentation of type 2 neurofibromatosis. *Arch Dis Child* 1999; **81**(6): 496–499.
8 Cackett P, Vallance J, Bennett H. Neurofibromatosis type 1 presenting with Horner's syndrome. *Eye* 2004; **19**: 351–353.
9 Parry DM, McCollin MM, Kaiser-Kupfer MI, Pulaski K, Nicholson HS, Bolesta M *et al*. Germ-like mutations in the neurifibromatosis 2 gene: correlations with disease severity and retinal abnormalities. *Am J Hum Genet* 1996; **59**(3): 529–539.

G Holmstrom

Department of Ophthalmology, University Hospital, Uppsala University, Uppsala 75185, Sweden

Correspondence: G Holmstrom,
Tel.: +46 18 6115180;
Fax +46 18 507455.
E-mail: gerd.holmstrom@ogon.uu.se

Eye (2006) **20,** 1472–1474. doi:10.1038/sj.eye.6702363;
published online 28 April 2006

Sir,

Rapid regression of disc neovascularization in a patient with proliferative diabetic retinopathy following adjunctive intravitreal bevacizumab

Bevacizumab (Avastin, Genentech Inc., San Francisco, CA, USA) is a recombinant humanized monoclonal IgG1 antibody that inhibits human vascular endothelial growth factor (VEGF). It has been administered intravitreally in VEGF-mediated diseases such as choroidal neovascularization[1] and central retinal vein occlusion.[2] VEGF plays a major role in mediating neovascularization in eyes with proliferative diabetic retinopathy (PDR).[3] We describe a patient who had dramatic regression of retinal neovascularization 1 week following adjunctive intravitreal bevacizumab.

Case report

A 29-year-old insulin-dependent diabetic male patient had recurrent vitreous hemorrhage (VH) OD for 8 months, despite pan-retinal photocoagulation (PRP). On presentation, his vision was 20/25-2 OD and 20/20-1 OS. Fundus examination showed intraretinal hemorrhages in four quadrants, moderate PRP, and no macular oedema OU. There was mild VH, and florid new vessels on the disc (NVD) OD. There was $\frac{1}{2}$ disc area of NVD, and NVE OS. Over the next 2 weeks, further PRP was performed OU, the VH cleared, and vision improved to 20/20 OD.

Five weeks after PRP, vision decreased to 20/40 OD. The examination OD showed florid NVD, new preretinal haemorrhage and VH, with PRP from the arcades to the ora. The NVD OS had regressed and vision was stable. Fluorescein angiography (Figure 1) showed extensive leakage from NVD OD.

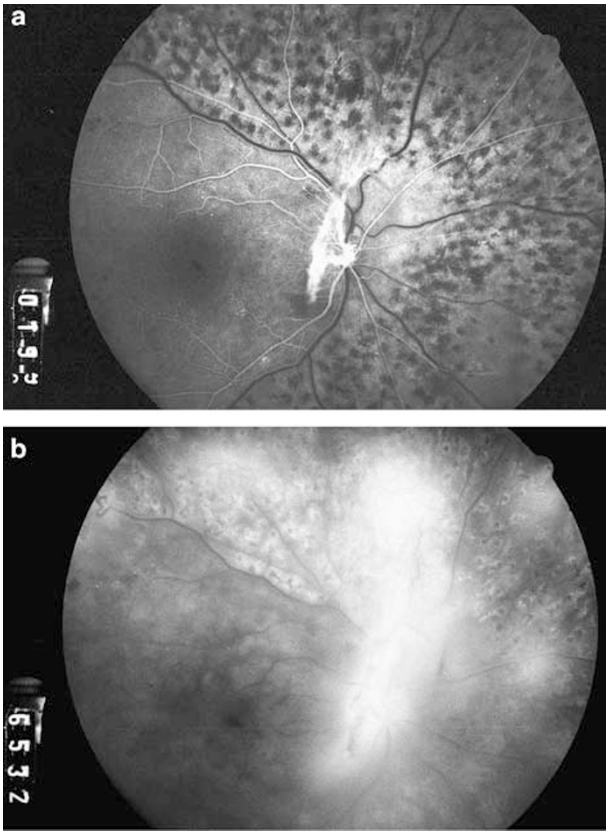

Figure 1 Fluorescein angiogram OD showing leakage from neovascularization of the disc. There is a full PRP. (a) Early phase, (b) late phase.

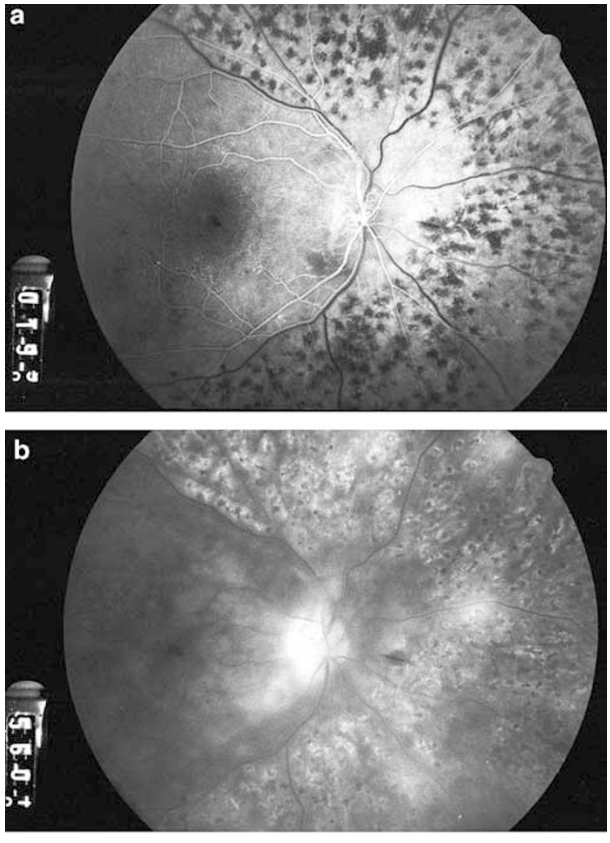

Figure 2 Fluorescein angiogram OD showing regression and cessation of leakage from neovascularization of the disc, 1 week after intravitreal bevacizumab. (a) Early phase, (b) late phase.