Flashing lights in thyroid eye disease: a new symptom described and (possibly) explained

Jenny J Danks, Richard A Harrad

Abstract

Background—Some patients with restrictive thyroid ophthalmopathy, referred for consideration of extraocular muscle surgery, complained of flashing lights in the superior visual field on upgaze. The frequency was assessed and the pathogenesis of this previously unreported symptom explored.

Methods—30 patients were recruited, all of whom had tight inferior recti and were in the burnt out phase of thyroid eye disease. They were directly questioned regarding any symptoms of photopsia and their records were examined with respect to disease status and treatment, ocular motility, intraocular pressure, retinal status, and surgical intervention. Magnetic resonance imaging (MRI) and cine MRI scans were reviewed for evidence of globe compression. The frequency of symptoms was compared with an age and sex matched control group.

Results—Three patients spontaneously complained of flashing lights. A further nine patients had this symptom when directly questioned. 18 patients had no symptoms. None of the 33 control patients had symptoms on direct questioning. Sagittal MRI and cine MRI failed to demonstrate globe compression by the inferior rectus muscle even in cases that showed an intraocular pressure rise in upgaze.

Conclusion—A new symptom of flashing lights in upgaze has been identified in thyroid eye disease patients with tight inferior recti. It is suggested that the lights are likely to be phosphenes as a result of either compression of the globe by a tight inferior rectus or traction on the insertion of the inferior rectus. The small amount of globe compression required to produce phosphenes seems to be beyond the resolution limit of MRI.

Thirty patients were recruited, six males and 24 females, with an average age of 55 years (range 26–80 years). All had restrictive thyroid ophthalmopathy with inferior rectus muscle disease and with tight inferior recti, in order to establish whether there is a relation between the symptoms and the presence of tight muscles.

Materials and methods

Thirty consecutive patients referred for consideration of extraocular muscle surgery in the dry or burnt out phase of thyroid eye disease were recruited. All patients had restrictive thyroid ophthalmopathy affecting the inferior rectus muscle. Patients were identified retrospectively from operative, outpatient, and radiological records. Controls were age and sex matched oculoplastic patients without intraocular disease.

Thyroid eye disease had been diagnosed on the basis of clinical presentation, restrictive ophthalmopathy, and radiological evidence of extraocular muscle enlargement. All patients had biochemical evidence of thyroid dysfunction in the past. In addition, the degree of inflammatory activity on short tau inversion ratio (STIR) sequence magnetic resonance imaging (MRI) was documented. Ocular motility assessment included Hess charts, fields of binocular single vision and uniocular fields of fixation. Intraocular pressure measurements in primary position and upgaze were noted where available. The nature of any immunosuppressive treatment and surgical intervention was documented.

Twenty patients had radiological imaging, 19 with MRI and 11 cine MRI, including patients with spontaneous phosphenes and some with raised intraocular pressure on upgaze. Three scans were performed specifically to examine globe compression. Cine MRI with longer exposure was performed in an attempt to improve image resolution. All scans were examined for evidence of globe compression, particularly on upgaze.

Patients who spontaneously complained of flashes of light were questioned as to the nature of their symptoms. Other patients were directly questioned for the occurrence of such symptoms. They were asked to describe any current or previous symptoms and to note any flashing lights when attempting to look up behind closed lids. Control patients were asked the same leading questions as study patients. All patients with positive symptoms had a retinal examination.

Results

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involvement. Range of upgaze was assessed as 0–70% of normal in the worse affected eye. Control patients comprised eight males and 25 females, with an average age of 55 years (range 31–81 years). None had any evidence of thyroid eye disease or intraocular pathology.

Treatment of the thyroid patients had included immunosuppression during the wet or active phase of the disease in 19 patients. Three patients underwent orbital decompression for compressive optic neuropathy. Seventeen patients had adjustable extraocular muscles after stability for at least 6 months. This comprised 10 inferior rectus recessions after stability for at least 6 months. Three medial rectus recessions and four patients had multiple muscles recessed. Of the symptomatic patients, seven had no muscle surgery, one was preoperative, and four were postoperative. Of the asymptomatic patients, five had no muscle surgery and 13 were postoperative.

Three thyroid patients spontaneously complained of flashing lights. A further nine patients had such symptoms on direct questioning. Eighteen patients had no symptoms despite direct questions. None of the 33 control patients had symptoms of flashing lights; two had floaters secondary to posterior vitreous detachments.

The lights reported by patients in the superior field on upgaze or eye closure are likely to represent this phenomenon. On lid closure, tight inferior recti are likely to exert pressure on the globe and traction on the muscle insertion with the upward movement of the eye, known as Bell’s phenomenon. We postulate this mechanism for our patients symptoms. The elevation of intraocular pressure in upgaze, is similarly, due to tight inferior rectus muscles. That there was no statistical correlation between the presence of phosphenes and increased intraocular pressure in upgaze or degree of muscle restriction, is not surprising, as all patients had restriction of upgaze which was theoretically capable of producing symptoms. It is possible that some patients had forgotten their symptoms and, in some, the degree of restriction had been altered by surgery. It is perhaps relevant that only four out of 12 (33%) of the symptomatic patients had had muscle surgery, compared with 12 out of 17 (71%) asymptomatic patients.

We attempted to demonstrate compression of the globe on imaging with cine MRI. No distortion of the globe could be seen. Only a small amount of pressure is required to produce phosphenes and it is likely that the resolution of the images is not sufficient to demonstrate globe compression in these cases.

In summary, flashing lights in thyroid eye disease are likely to represent phosphenes as a result of mechanical distortion of the sensory elements of the retina produced by tight muscles. We report a new symptom of flashing lights in thyroid eye disease which we attribute to compression of the inferior retina by the tight inferior rectus muscle or traction on the insertion of the inferior rectus.
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