ORIGINAL ARTICLES – Clinical science

Trigeminal sensory loss in orbital disease

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Abstract

A study was undertaken to estimate the incidence of periorbital trigeminal sensory loss with different types of orbital disease and to determine whether the area of loss is related to the location or nature of the underlying condition. A retrospective review was carried out of the clinical case notes of patients identified from the orbital diagnostic database held at Moorfields Eye Hospital. A total of 103 patients (51 male, 52 female), aged between 11 and 84 years (median 51 years), had signs of periorbital trigeminal sensory loss at the time of investigation for orbital disease and in 14/103 (13%) this sensory loss was symptomatic. Sensory loss was due to trauma in eight cases (8%), benign orbital disease in 54 (52%), and malignant disease in 41 cases (40%). The relative frequency of recorded sensory changes is about 9% for malignant tumours, 5% for benign tumours, 4% after orbital trauma, 3% for orbital inflammatory disease, and less than 1% for orbital structural anomalies. Relative corneal hypoaesthesia occurred in 40/103 (39%) cases. Periorbital sensory loss was recorded in about 3% of patients with orbital disease and, in contrast with commonly held views, malignancy was not the most common cause of sensory loss. The affected dermatome is a useful guide to the location of orbital disease, but a poor indication of underlying pathology. Relative corneal hypoaesthesia appears unrelated to the type of disease or to its location within the orbit.

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The function of peripheral sensory nerves may be reduced by compression caused by structural anomalies, inflammation, or neoplasms, or by direct infiltration – with abnormal tissues (such as amyloid or sarcoid), or by malignant infiltration. In some cases, impairment of function may be sufficient to be symptomatic or to cause clinically detectable sensory loss. It is a widely held view that sensory loss in relation to a mass is likely to indicate malignancy and also that malignant disease is one of the commonest causes of sensory loss.

Cutaneous sensory loss with orbital disease might be expected for several reasons. Several branches of the first and second divisions of the trigeminal nerve pass through all quadrants of the orbit and subserve a large area of facial sensation (Fig 1). The osseous orbit allows compression, rather than displacement, of nerves by any abnormal tissues within the diseased orbit and might, therefore, be expected to predispose the nerves to injury or impairment of function.

This paper examines the relative incidence of periorbital sensory loss with different orbital diseases and the dermatome involvement in relation to the position of the disease within the orbit.

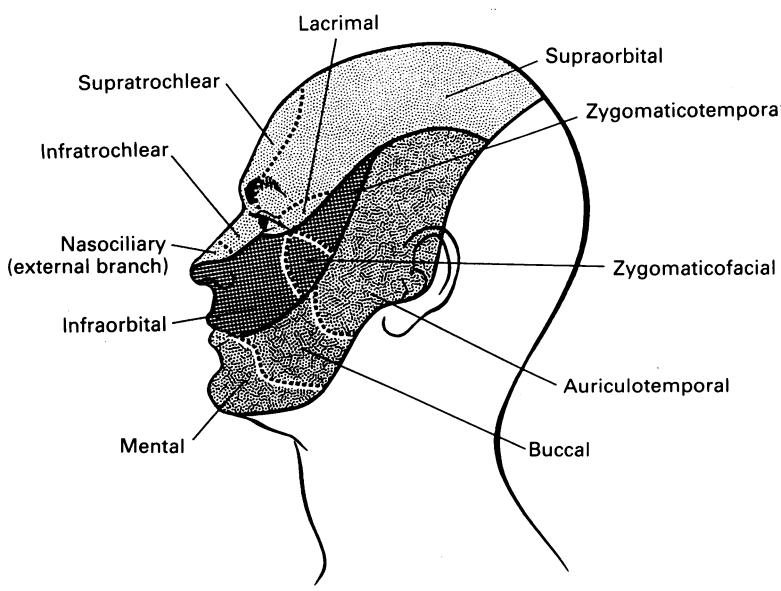

Figure 1 Sensory dermatomes typically subserved by the divisions of the trigeminal nerve (from Rootman[1]).

Methods

Patients with periorbital trigeminal sensory loss were identified from the diagnostic orbital database held in the orbital clinic at Moorfields Eye Hospital. The clinical case notes were reviewed for these patients and details of sensory loss recorded, together with the type and position of their orbital disease. Periorbital sensation for light touch was assessed as part of the clinical examination of almost all patients attending the orbital clinic.

Results

Notes for 103 patients with loss of periorbital cutaneous sensation or corneal sensation were reviewed; these were patients with a difference in light touch or pinprick perception between the two sides of the face, changes conforming to the pattern of appropriate dermatomes (Fig 1). There were 51 males and 52 females, with an age

Table 1 Proportion of patients with different types of orbital disease and clinically detectable sensory loss

Type of orbital lesion	Number of cases
Malignant tumours:	41/103 (40%)
primary lacrimal carcinoma	19
metastasis	11
lymphoma	6
melanoma	3
malignant neurilemmoma	2
Benign tumours:	22/103 (21%)
peripheral nerve sheath tumours	6
sphenoidal wing meningioma	6
optic nerve meningioma	4
lymphoid hyperplasia	4
pleomophic adenoma	2
Inflammatory:	26/103 (25%)
idiopathic	22
dacryoadenitis	2
cellulitis	1
sarcoidosis	1
Structural:	6/103 (6%)
sinus mucoceles	3
vascular anomalies	2
congenital malformation	1
Post-traumatic	8/103 (8%)

range between 11 and 84 years at the time of review (median 51 years, mean 48 years).

Several types of orbital diseases caused detectable sensory loss (Table 1): malignancy was present in 41/103 (40%) cases, benign tumours in 22 (21%) cases, orbital inflammatory disease in 26 (25%) cases, and structural lesions (six cases) or preceding trauma (eight cases) in a minority of patients. For the period of review, these rates represented 9% of cases of malignancy recorded in the orbital database, 5% of benign tumours, 3% of orbital inflammations, 4% of post-traumatic cases, and less than 1% or orbital structural lesions (Table 2).

Table 2 The relative incidence of periorbital sensory loss with different classes of orbital disease, together with the proportions that are either symptomatic or associated with pain

		Sensory loss	
Type of orbital lesion	Proportion of cases (%)	Symptomatic (%)	With pain (%)
Malignancy	41/469 (9)	6/41 (15)	21/41 (51)
Benign tumours	22/471 (5)	–	4/22 (18)
Inflammatory	26/956 (3)	4/26 (15)	23/26 (88)
Structural	6/988 (<1)	2/6 (33)	2/6 (33)
Post-traumatic	8/186 (4)	2/8 (25)	4/8 (50)
Overall	103/3070 (3·3)	14/103 (14)	54/103 (52)

Table 3 Dermatomes affected in 88 patients with cutaneous periorbital sensory loss; the proportions indicate the number with sensory loss in an area appropriate to the major site of their disease

Neural dermatomes involved				Proportion with involvement appropriate to principal site of orbital lesion
Lacrimal	Alone			9/10
	and supraorbital	Alone		5/5
		and supratrochlear	Alone	4/4
			and infratrochlear	1/1
			and infraorbital	1/1
		and zygomatics		1/1
	and zygomatics		Alone	2/2
			and infraorbital	1/1
Supraorbital	Alone			13/13
	and supratrochlear		Alone	7/8
			and infratrochlear	4/5
			and infraorbital	2/2
	and infraorbital			1/1
Supratrochlear				4/4
Infraorbital	Alone			14/18
	and infratrochlear			3/3
	and zygomatics			2/2
Zygomatics				0/1
All areas				6/6

Table 4 Incidence of corneal hypoaesthesia in relation to the type of orbital disease

Area of trigeminal sensory loss	Nature of orbital disease	
	Benign disease	Malignant disease
Corneal hypoaesthesia only	9/15 (60%)	6/15 (40%)
Corneal and cutaneous hypoaesthesia	16/25 (64%)	9/25 (36%)
Cutaneous hypoaesthesia only	37/63 (59%)	26/63 (41%)

The sensory loss was symptomatic (with numbness) in only 14/103 (13%) patients: in 2/8 (25%) cases after trauma, 6/54 (11%) with benign conditions, and 6/41 (15%) with malignant tumours (Table 2). The proportions with symptomatic sensory loss are not significantly different with malignancy (6/41 cases) or with benign diseases (8/62 cases; $\chi^2=0{\cdot}08$, $p>0{\cdot}9$). Sensory loss was accompanied by pain – so-called 'anaesthesia dolorosa' – in 54/103 (52%) patients, being most common with inflammatory disease (23/26 cases; 88%) and with malignancy (21/41 cases; 51%) (Table 2).

Cutaneous sensation was reduced in 88/103 (86%) patients, the other 14% having only corneal hypoaesthesia, with the ophthalmic division of the trigeminal nerve being most frequently affected; in 63/88 (73%) patients several dermatomes were affected (Table 3). The area of hypoaesthesia was appropriate for the disease location in 48/51 (96%) patients with involvement solely of the ophthalmic division and appropriate for disease location in 16/21 (76%) with maxillary division involvement solely (Table 3). In each of six patients with widespread cutaneous hypoaesthesia, there was extensive disease affecting the apex of the orbit.

Forty (39%) patients had relative corneal hypoaesthesia (in isolation or with cutaneous loss) and the incidence of corneal sensory loss is similar with benign or malignant disorders (Table 4). Of 15 patients with corneal hypoaesthesia solely, only six (40%) had intraconal disease.

Discussion

Trigeminal sensory loss with orbital disease is relatively uncommon, occurring in only about 3% of cases in this series. Such loss was most frequent with malignant tumours (in 9% of orbital malignancies) but, because of the relative infrequency of malignancy among orbital diseases, benign conditions (tumours, inflammation or structural anomalies) were the most common cause of sensory loss (52% caused by benign disease, 40% malignancy, and 8% post-traumatic). In contrast with commonly held views, orbital malignancy is not the most common cause of periorbital sensory loss.

Sensory loss is rarely symptomatic and only 14 patients reported numbness. Just over half of the patients, however, experienced pain with their orbital disease – most of them having either inflammatory disease or malignancy (88% and 51% of which cases were painful, respectively).

Several branches of the trigeminal nerve may be affected by orbital disease (Table 3), especially those of the ophthalmic (first) division which has

a large number of branches passing through the orbit (Fig 1). In most cases (91%) the extent of cutaneous sensory loss is indicative of the position of the orbital disease, with the lacrimal, supraorbital, or supratrochlear nerves being affected by disease along the orbital roof and the zygomatic and infraorbital nerves being affected by diseases along the orbital floor (Table 3). Disease at the orbital apex or the superior orbital fissure may cause hypoaesthesia affecting several, or even all, of the periorbital dermatomes.

Relative corneal hypoaesthesia appears unrelated to the nature of the underlying orbital disease – as with cutaneous hypoaesthesia (Table 4). In contrast with cutaneous sensory loss, however, corneal loss appears to be unrelated also to the position of the disease within the orbit. Of 15 patients with purely corneal hypoaesthesia, only six (40%) had intraconal disease – a disease location where impairment of the nerves subserving corneal sensation might be expected.

1 Rootman J. *Diseases of the orbit. A multidisciplinary approach.* 1st ed. Philadelphia: J B Lippincott, 1988: 9.