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CASE REPORT

Damage to the inferior oblique muscle branch of the oculomotor nerve: a complication during orbital fat decompression

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Abstract
Purpose To present patients who suffered damage to the inferior oblique muscle branch of the oculomotor nerve during orbital fat decompression.
Methods This study was a retrospective chart review of all patients who underwent orbital decompression surgery between April 2009 and June 2016 by the authors.
Results Among 414 sides from 226 consecutive patients who underwent orbital decompression, the inferior oblique muscle branch was injured in two sides (0.5%) of two patients. Both patients showed hypotropia and incyclotropia immediately after surgery. Within 6 months of injury, ocular deviation on primary gaze had mostly resolved after conservative treatment. None of the patients underwent strabismus surgery. Postoperative computed tomographic images demonstrated that the affected branch was indistinct 3–4 mm posterior to the inferior oblique muscle.
Conclusions This report indicates that injury to the inferior oblique muscle nerve branch can occur at a point posterior to the inferior oblique muscle during orbital fat decompression; however, the resulting ocular deviation improves considerably within 6 months of injury.

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Keywords Inferior oblique muscle branch · Oculomotor nerve · Orbital fat decompression · Hypotropia · Incyclotropia

Introduction

Damage to the inferior oblique muscle branch of the oculomotor nerve has not been reported as a complication of orbital decompression, which may lead to postoperative diplopia. The occurrence of postoperative diplopia following orbital decompression has been attributed to the following: (1) misalignment of the extraocular muscles (EOMs); (2) direct injury to or enhanced tension of the EOMs; and (3) scarring of the EOMs or orbital tissues around the EOMs [1–4].

The inferior oblique muscle branch of the oculomotor nerve extends anteroposteriorly in the inferolateral quadrant of the orbit [5]. As this quadrant is occupied by the greatest volume of fat as compared to the other quadrants [6], sufficient fat removal from this quadrant is recommended to obtain a satisfactory decompressive effect [6]. However, excess removal may cause injury to the inferior oblique muscle branch.

In this report, we have described two patients who suffered from injury to the inferior oblique muscle branch of the oculomotor nerve during orbital fat decompression.

Materials and methods

This study was a retrospective chart review of all patients who underwent orbital decompression surgery between April 2009 and June 2016 by the authors. The following patient data were collected: sex, age, thyroid status, surgical side, concomitant bony decompression procedure, volume of orbital fat removal from the inferolateral quadrant, side of the injury, treatment for ocular deviation, results of pre- and postoperative extraocular muscle motility, and results of pre- and postoperative Hertel exophthalmometry. EOM motility was evaluated using the Hess chart, binocular single-vision field (BSVF) assessment, and the angle of ocular deviation on synoptophore examination.

Computed tomographic (CT) studies were performed using a high-speed scanner (Aquillion 64; Toshiba, Tokyo, Japan). Contiguous 1-mm coronal images were obtained using a soft tissue window algorithm (width: 400; level: 20).

Results

Patient demographic and clinical data are summarized in Table 1. Among 414 sides from 226 patients, two sides (0.5%) from two patients were included in this study.

One patient (Patient #1) was a 29-year-old woman with right inferior oblique paresis after balanced orbital decompression. Orbital fat was removed via the lateral approach. The inferior oblique muscle was not exposed intraoperatively.

Although Patient #1 presented with slight underaction of the right inferior oblique muscle on right supero-nasal gaze on Hess chart preoperatively (Fig. 1a), the BSVF was considerably wide (Fig. 1b), and neither hypotropia nor cyclotropia was detected on synoptophore examination (Table 1). However, immediately after orbital decompression surgery, the patient developed hypotropia of 4 degrees and incyclotropia of 5 degrees (Table 1). These parameters decreased to 0 and 2 degrees, respectively, after oral vitamin B12 administration for a period of 6 months. (Table 1). The patient still exhibited residual underaction on right supero-nasal gaze on Hess chart (Fig. 1c), and diplopia on left temporal and supero-temporal gazes (Fig. 1d); however, strabismus surgery was not performed as the synoptophore examination revealed no deviation on primary gaze.

Preoperative coronal CT images of Patient #1 clearly demonstrated the inferior oblique muscle branch on both sides (Fig. 1e). However, the right branch was indistinct on a postoperative coronal CT image taken 3 mm posterior to the inferior oblique muscle on the affected side (Fig. 1f).

The other patient (Patient #2) was a 42-year-old woman with right inferior oblique paresis after deep lateral orbital wall decompression. Orbital fat was removed via the lateral approach. The inferior oblique muscle was not exposed intraoperatively.

Patient #2 exhibited normal preoperative extraocular muscle motility on Hess chart (Fig. 2a), normal BSVF (Fig. 2b) and had neither hypotropia nor cyclotropia on synoptophore examination (Table 1). However, immediately after orbital decompression surgery, the patient displayed 8 degrees of hypotropia and 8 degrees of incyclotropia (Table 1). These parameters decreased to 2 and 0 degrees, respectively, after oral vitamin B12 administration for 6 months (Table 1). The patient still exhibited residual underaction of the right inferior oblique muscle on supero-nasal gaze on Hess chart (Fig. 2c) and diplopia on left temporal and supero-temporal gazes (Fig. 2d). Similarly, the patient did not undergo strabismus surgery as the synoptophore examination revealed no deviation on primary gaze.

The inferior oblique muscle branch was clearly illustrated bilaterally on preoperative coronal CT images of Patient #2 (Fig. 2e). However, the right branch was indistinct on a postoperative coronal CT image taken 4 mm posterior to the inferior oblique muscle on the affected side (Fig. 2f).

Discussion

Damage to the inferior oblique muscle branch of the oculomotor nerve during orbital fat decompression was demonstrated in two patients. This complication has not been previously described, although surgeons are urged not to injure directly the inferior oblique muscle itself during orbital fat decompression [2, 7, 8].

Damage to the inferior oblique muscle branch can be caused by direct trauma by the surgical instrument, indirect injury by excess traction on the nerve during orbital fat removal, or indirect injury by compression

Table 1 Demographic and clinical data of patients with inferior oblique muscle injuries

Patient #	1	2
Age	29	42
Sex	F	F
Thyroid status	Euthyroid	Euthyroid
Surgical side	B	B
Concomitant bony decompression		
R	Orbital fat removal following balanced orbital decompression	Orbital fat removal following deep lateral orbital wall decompression
L	Orbital fat removal following balanced orbital decompression	Deep lateral orbital wall decompression
Volume of orbital fat removal (mL)		
R	1.5	1.2
L	0.9	0
Location (side) of injury	R	R
Treatment for ocular deviation	Oral vitamin B12 (1500 μg/day)	Oral vitamin B12 (1500 μg/day)
Ocular deviation (degree)		
Hypotropia		
Preoperative	0	0
Immediately after surgery	4	8
Postoperative 6 months	0	2
Incyclotropia		
Preoperative	0	0
Immediately after surgery	5	8
Postoperative 6 months	2	0
Hertel exophthalmometry (mm)		
Preoperative	25.0	17.0
Postoperative 6 months	19.0	13.0

F female, *B* bilateral, *R* right, *L* left

due to orbital edema and/or hematoma. Direct injury to the inferior oblique muscle presents a similar limitation pattern of EOM motility to indirect injury. However, our patients exhibited considerable resolution of the injury-related inferior oblique paresis, indicating that the injury must have been due to an indirect cause [9]. In addition, the anterior approach via a conjunctival or subciliary incision to the orbital floor carries a much higher risk of direct injury to the inferior oblique muscle because it is located in the anterior orbit [10, 11] and is thereby easily exposed during fat removal [2, 7, 8]. However, orbital fat removal was done via the lateral approach in both patients, in which the inferior oblique muscle was not intraoperatively exposed. This makes direct nerve injury as an unlikely cause of limited EOM motility.

The infraorbital groove and orbital floor are accessible landmarks in estimating the location of the inferior oblique muscle branch. The branch is located just above the infraorbital groove, posterior to the

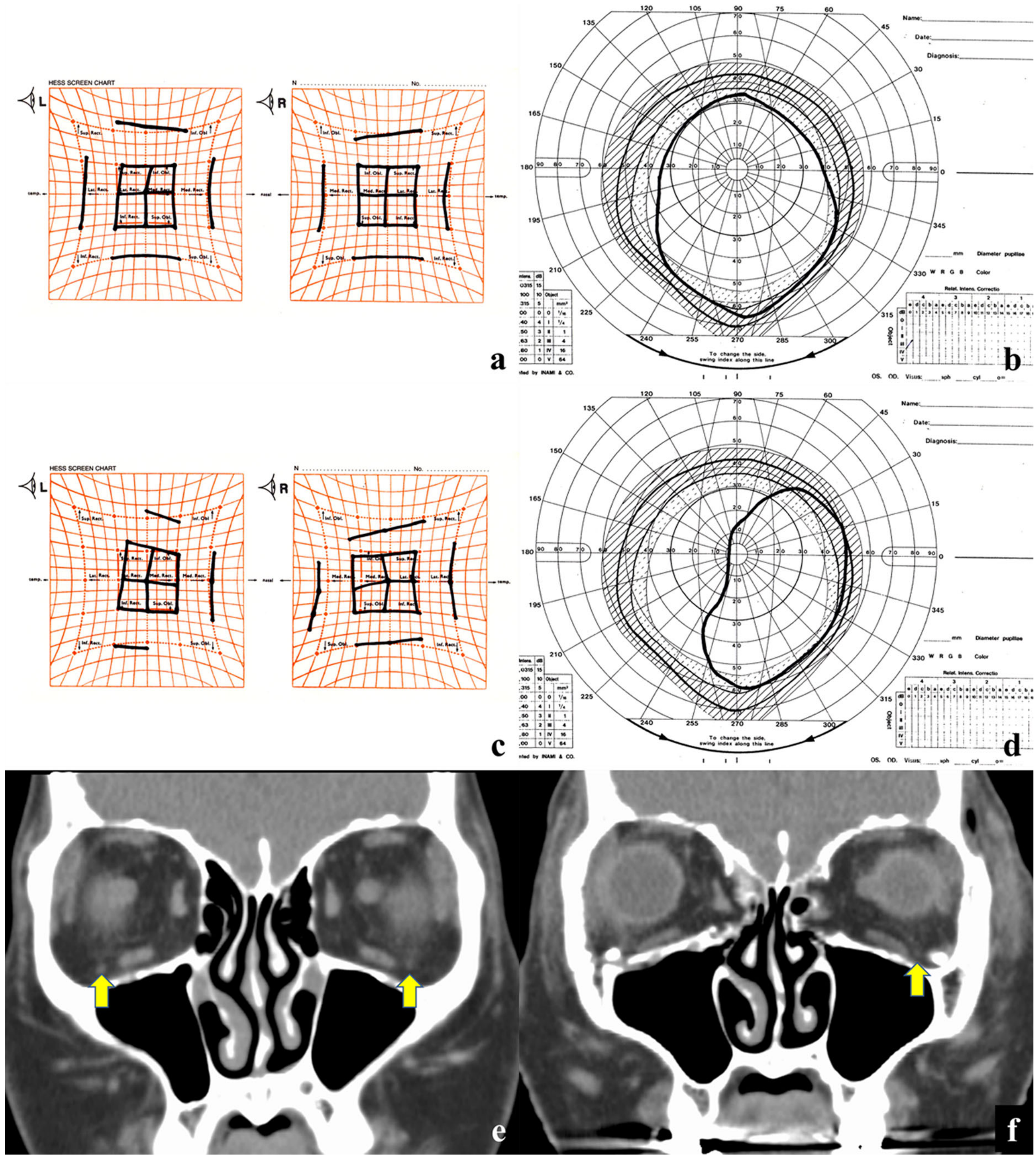

Fig. 1 Patient #1. **a**. Preoperative Hess chart. **b**. Preoperative binocular single-vision field (BSVF). **c**. Hess chart at 6 months post-surgery. **d**. BSVF at 6 months post-surgery. **e**. Preoperative coronal computed tomographic (CT) image. The inferior oblique muscle branch of the oculomotor nerve is visible on both sides (arrows). **f**. Postoperative coronal CT image. The inferior oblique muscle branch of the oculomotor nerve is visible on the left side (arrow), but indistinct on the right side

posterior pole of the globe; and 1 mm above the orbital floor, anterior to the posterior pole of the globe [5]. Exposure of these landmarks prior to orbital fat removal may be a useful approach in preventing branch injury; however, the positional relationships between the inferior oblique muscle branch and these

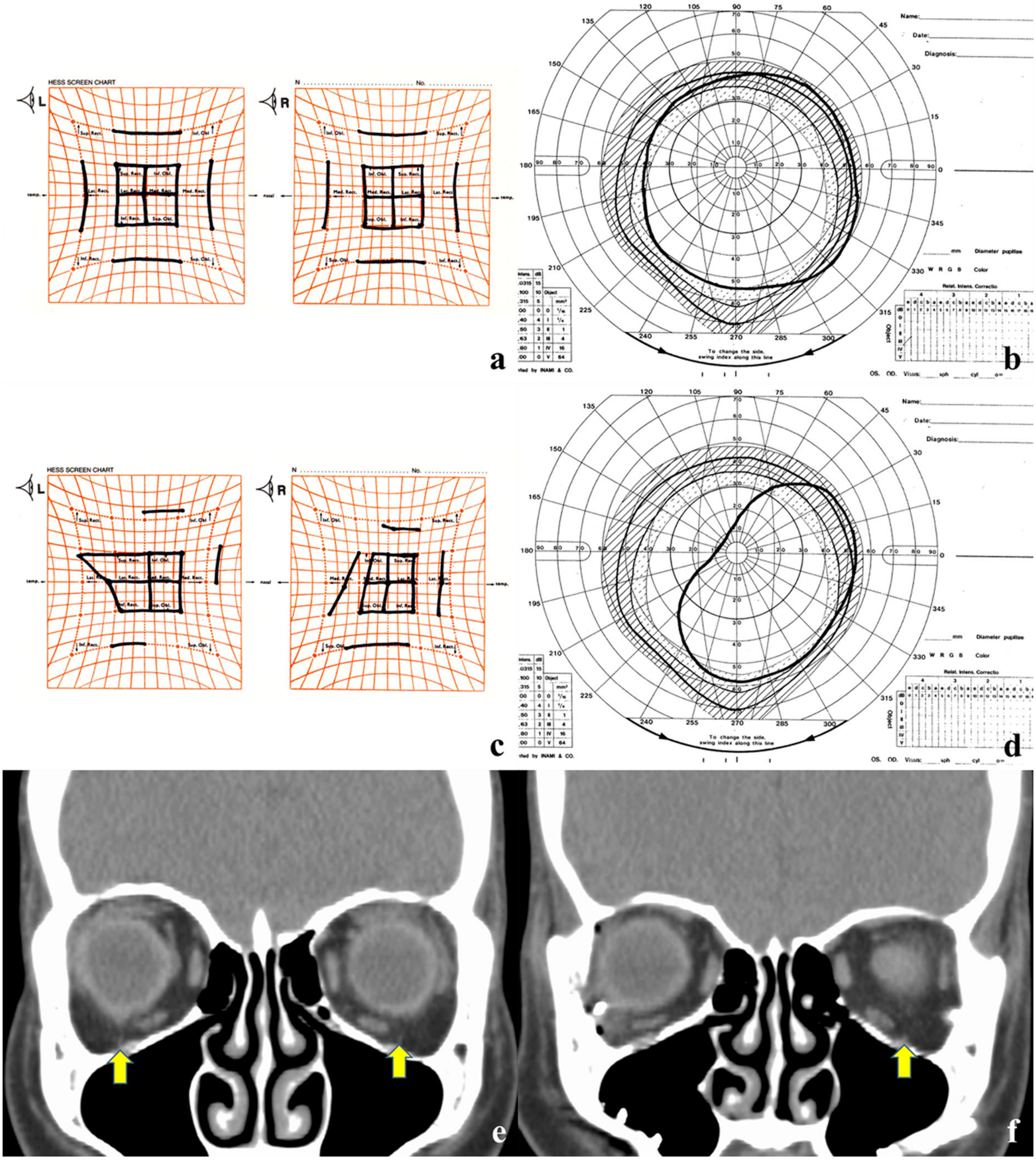

Fig. 2 Patient #2. **a**. Preoperative Hess chart. **b**. Preoperative binocular single-vision field (BSVF). **c**. Hess chart at 6 months post-surgery. **d**. BSVF at 6 months post-surgery. **e**. Preoperative coronal computed tomographic (CT) image. The inferior oblique muscle branch of the oculomotor nerve is visible on both sides (arrows). **f**. Postoperative coronal CT image. The inferior oblique muscle branch of the oculomotor nerve is visible on the left side (arrow), but indistinct on the right side

structures may change during orbital fat decompression via the lateral approach or preceding deep lateral orbital wall decompression [12]. In addition, prior exposure of the infraorbital groove and the orbital floor can also result in damage to the inferior oblique muscle branch.

We previously reported the orbital fat volume in the inferolateral quadrant measured using CT images [12]. As the volume of orbital fat removed can be measured using a syringe during surgery [2, 12], knowledge of the orbital fat volume in the inferolateral quadrant can serve as a marker for maximum fat decompression without causing injury to the inferior oblique muscle branch.

Minimal ocular deviation on primary gaze was detected at 6 months post-surgery in those who did not undergo strabismus surgery. This finding indicates that considerable resolution of the injury-related inferior oblique paresis can be expected after conservative treatment [9]. Strabismus surgery, therefore, is only necessary when there is insufficient patient recovery.

In conclusion, we have reported two cases of injury to the inferior oblique muscle branch of the oculomotor nerve during orbital fat decompression. The resulting ocular deviation improved considerably within 6 months post-surgery after conservative medical management.

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Compliance with ethical standards

Conflicts of interest The authors have no conflicting interests related to this manuscript.

Human Participants and/or Animals; Informed consent This study was approved by the Institutional Review Board (IRB) of our institution (2016-H273) and adhered to the 1964 Declaration of Helsinki tenets. The IRB granted a waiver of informed consent for this study based on the ethical guidelines for medical and health research involving human subjects established by the Japanese Ministry of Education, Culture, Sports, Science, and Technology and by the Ministry of Health, Labor, and Welfare. The waiver was granted since the study was a retrospective chart review, not an interventional study, and it would be difficult to obtain consent from patients who had been treated several years prior. Nevertheless, at the request of the IRB, we published an outline of the study, available for public viewing, on the Aichi Medical University website. This also provided patients the opportunity to decline participation; however, none of the patients declined to participate. Personal identifiers were removed from the records prior to data analysis.

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