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Restrictive Strabismus After Retrobulbar Anesthesia

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Abstract: Two rare cases of strabismus resulting from contracture of the extraocular rectus muscles after retrobulbar anesthesia for cataract surgery are described. Clinical signs in both cases suggested that the development of the impaired function of the lateral and superior rectus muscles followed the same pattern: initial stimulation followed by paretic and restrictive stages. Abnormal enlargement of the muscles was identified by computed tomography (CT) and magnetic resonance imaging (MRI). The data indicate that the strabismus was the result of direct injection of anesthetics into the rectus muscle. **Jpn J Ophthalmol 1997;41:23–26**

Key Words: Cataract surgery, restrictive strabismus, retrobulbar anesthesia.

Introduction

The contracture, or overaction, of extraocular muscles after retrobulbar anesthesia is one of the primary differential diagnoses when persistent strabismus occurs after cataract surgery.[6] A myotoxic effect of the anesthetics has been suggested as a cause of transient paresis, with subsequent contracture of its antagonist, in the affected muscle.[2,3] The traumatic aspect of this condition has received much attention.[4] In this report, two rare cases of restrictive strabismus following paralytic strabismus after cataract surgery are described. The enlarged lateral and superior rectus muscles were detected by computed tomography (CT) and magnetic resonance imaging (MRI). Clinical signs suggested that the rectus muscles were stimulated by the injection of anesthetics, then became transiently paretic, and finally became contracted.

Case Reports

Case 1

A 64-year-old woman had phacoemulsification (PEA) of the right eye with implantation of a posterior chamber intraocular lens (PC-IOL). Anesthetic of 4 ml 2% lidocaine with 1:80,000 epinephrine was injected retrobulbarly in the inferotemporal quadrant using a curved tapered needle. An outward deviation of the right eye was observed immediately after injection. No bridle suture was placed and there were no complications during surgery.

On the first postoperative day, the patient complained of horizontal diplopia, worsening on right gaze. Abduction of the right eye was restricted. This diplopia gradually decreased over 2 weeks; however, the patient noticed horizontal diplopia, again, which worsened on left gaze because of limited adduction of the right eye. One year after surgery, she had 15 prism-diopter (PD) exoptropia in the primary position (Figure 1a), and the lateral rectus muscle (LRM) overacted on right gaze. Peak velocity of horizontal (both right and left) saccadic eye movements of the right eye was less than that of the left eye, although both were within normal limits. Forced duction testing confirmed restricted adduction of the right eye. CT showed focal enlargement of the LRM of the right eye (Figure 1b). The diplopia disappeared after a 10 mm recession of the right LRM.

Case 2

A 56-year-old woman had PEA with PC-IOL of the left eye. A retrobulbar block was used, as in Case 1. An upward deviation of the left eye was observed

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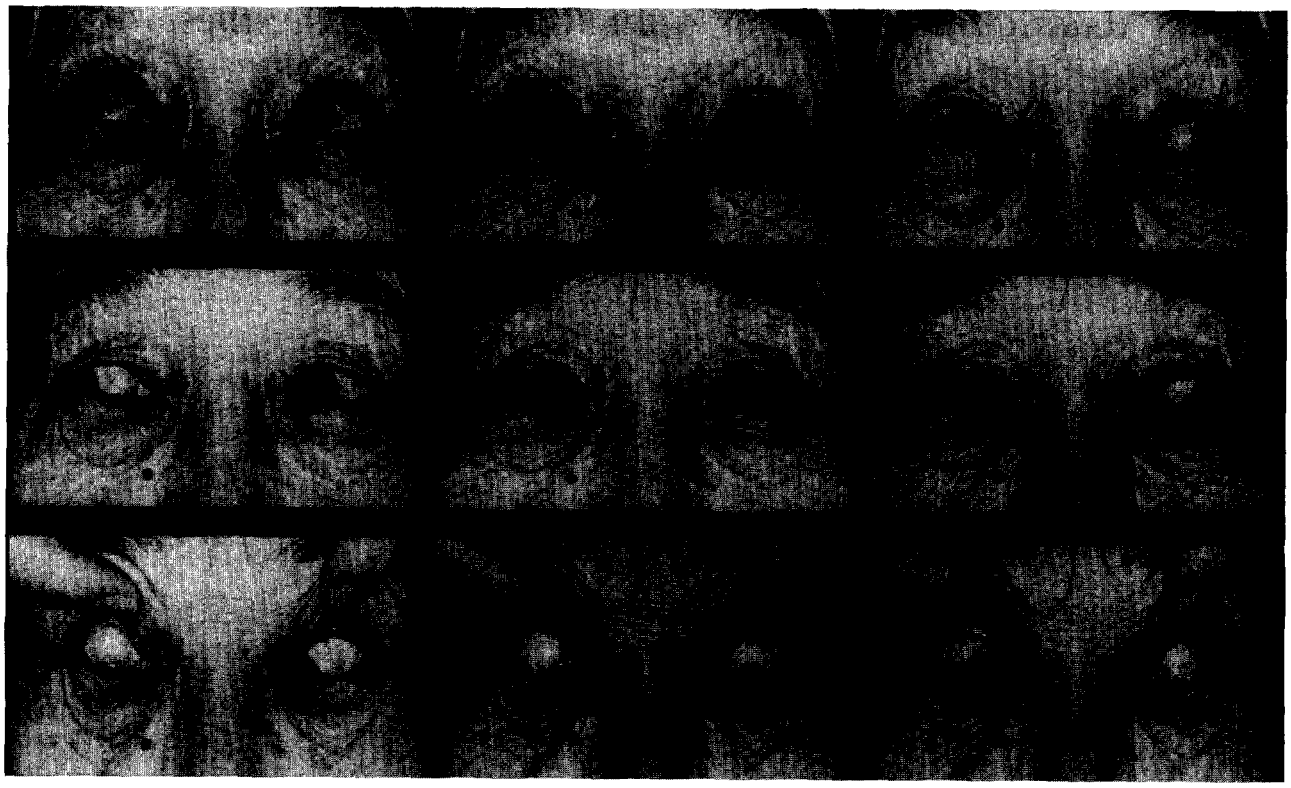

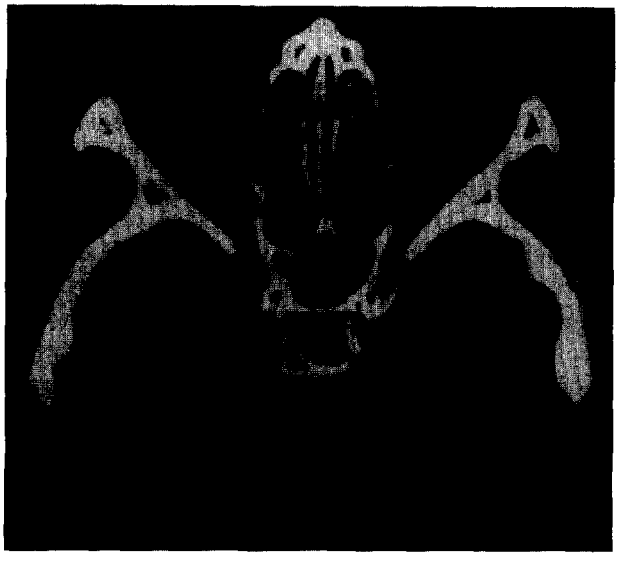

Figure 1. (**A**: above) Nine diagnostic positions showing esotropia and overaction of right lateral rectus muscle (Case 1). (**B**: below) CT scan focal enlargement of right lateral rectus muscle (Case 1).

immediately after the injection. No bridle suture was placed. There were no complications during surgery.

On the first postoperative day, the patient complained of vertical diplopia; supraduction of the left eye was restricted. This diplopia soon resolved, but a few weeks after surgery, she noticed a vertical diplopia, which worsened on downward gaze. The recurrent diplopia increased for about 3 months. Examination with a major amblyoscope revealed 16 PD left hypertropia with 6 PD esotropia in the primary position (Figure 2a). Forced duction testing revealed restricted infraduction of the left eye. MRI showed focal enlargement of the superior rectus muscle (SRM) of the left eye (Figure 2b). The left hypertropia and diplopia disappeared in all fields of gaze after a 7 mm recession of the left SRM.

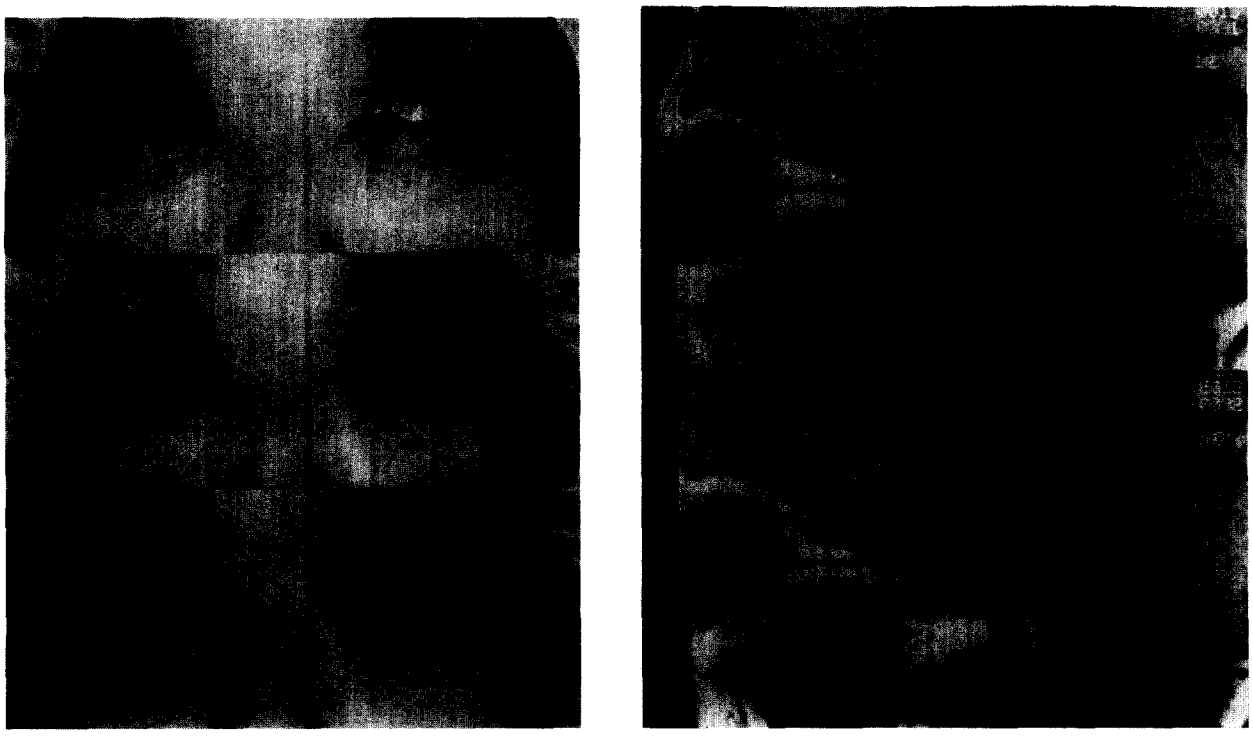

Figure 2. (**A**: left) Left hypertropia and overaction of left superior rectus muscle in up, primary, and down gaze (Case 2). (**B**: right) Saggital MRI focal enlargement of left superior rectus muscle (top: right eye; bottom: left eye) (Case 2).

Discussion

In both cases presented here, the anesthesia was administered by the same surgeon. The clinical signs of impairment followed similar patterns. Immediately after the retrobulbar injection, the eye deviated toward the enlarged rectus muscle. The affected muscle was paretic during the early postoperative period, with gradual recovery. A few weeks after surgery, the muscle became restrictive, as the forced duction testing confirmed. Direct injury to the muscle by the injection of anesthetics appeared to be the most likely cause of this ophthalmoplegia. Placement of a bridle suture might cause strabismus after cataract surgery;[5] however we had not used bridle sutures in these cases.

Many cases of hypertropia and superior rectus muscle overaction of the involved eye following cataract surgery have been reported.[2,3,6] Most of these were not associated with restrictive ophthalmoplegia; rather, a temporary paralysis of the inferior rectus muscle resulting in a contracture or strengthening of the antagonistic superior rectus muscle is suggested.

Our patients resemble those described by Hamed,[4] who experienced restrictive ophthalmoplegia with an enlarged rectus muscle; we do not know if his patients also noticed the transient diplopia from paralysis of the rectus muscle that affected our patients. Munoz[7] described a case of inferior rectus muscle overaction after peribulbar anesthesia was administered inferiorly. He hypothesized that the myotoxicity led to the contraction of the affected muscle.

The 3-phase pattern of the development of impairment in our cases points to direct injury of the muscle by retrobulbar anesthetic administration. Deviation of the eye, observed immediately after the retrobulbar injection, would be caused by the hyperreactivity of the muscle to the trauma of needle penetration and the mass of the anesthetic agent. Transient paralysis of the affected rectus muscle after surgery may have resulted from muscle damage from the myotoxicity of the anesthetic. The paralysis was gradually succeeded by muscle contracture,[7] possibly resulting from fibrotic changes within the muscle; diplopia in the opposite direction finally developed.

Porter reported on experimental retrobulbar anesthesia with bupivacaine hydrochloride in monkeys;[8] morphologic changes produced in the extraocular muscle were mild and no functional disorder was indicated. Carlson et al described massive lesions that

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regularly developed from injection of local anesthetics into the rectus muscles of monkeys.[9] The enlarged portion of the muscle shown by CT or MRI in our two cases is the site of the anesthetic injection.

Deep insertion of the retrobulbar block needle makes successful anesthesia available, but the risk of extraocular muscle injury increases because the rectus muscles converge in a narrow posterior orbital space. The anesthetist must use extreme care with direction and depth in placement of the needle point during retrobulbar anesthesia in order to prevent contracture-producing injury to the extraocular muscle.

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