Retinal and Choroidal Vascular Occlusion After Fat Injection Into the Temple Area

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Autologous fat injection for soft-tissue augmentation has become an effective and reliable method of restoring volume and correcting the atrophy that accompanies senescence or congenital anomalies and has become increasingly popular in recent years. We present a rare case of acute visual loss after autologous fat injection into the temple area.

A 22-year-old man presented at our outpatient department complaining of the sudden onset of severe vision loss in the right eye while he was receiving the autologous fat injection into the ipsilateral temple area. The patient had undergone autologous fat transplantation from the abdomen into the temple region in a department of plastic surgery 20 days previously for the treatment of soft-tissue deficiency of the right side as a result of right-sided craniofacial microsomia. From a review of the history, the operation was performed under local anesthesia, and nerve blocks (infraorbital, mental, and supraorbital blocks) were administered around the area to be transferred using 2% lidocaine combined with 1:100,000 epinephrine. Twenty days after onset, he was referred to our department. On physical examination, the patient’s level of consciousness was normal, without aphasia. Neurologic examination was normal except that he had no light perception in the right eye. External and anterior segment evaluation revealed an afferent pupillary defect in the right eye. Right fundus examination revealed that the color of the retina was normal, but there were atrophic macular spots, yellow flecks, and cotton wool spots around the macula, attenuated retinal arterioles, and optic disc atrophy (Figure 1). Fundus fluorescein angiography showed a delay in arteriovenous transit time (14 s), narrowed and blocked arterioles in the perifoveolar regions, and perfusion defects of the choroidal circulation accompanied by late leakage in the posterior pole (Figure 2). Ocular coherence tomography imaging revealed retinal thinning, and the marked decrease in retinal thickness was shown numerically and by conventional color (Figure 3). The cross-sectional imaging of ocular coherence tomography showed that the margin of each inner retinal layer was difficult to differentiate, and the decreased thickness was most obvious in the inner layer, whereas the outer layer thickness was little affected. In addition, structural damage from the limiting membrane line to the pigment epithelium layer line and intraretinal cystic changes were seen within the foveal pit region (Figure 4). Based on these clinical features, a diagnosis of chorioretinal infarction was made. Because the patient presented late in the course of the disease, he was given only high-dose oral multivitamins for 1 month. He was then followed up over 3 months, during which no improvement of vision was noted.

The present case demonstrates a chorioretinal infarction caused by autologous fat injection. Central retinal artery occlusion is an ocular emergency, because it is a stroke of the eye caused by obstruction of the central retinal artery, usually by a thrombus or embolus that results in painless, disabling vision loss. Choroidal infarction is extremely rare, although certain clinical appearances have been attributed to ciliary vessel occlusion.

It is believed that sharp instrumentation and high-injection pressures could introduce fat into the vasculature. The force of the injection is sufficient to cause retrograde flow into the ophthalmic arterial system. Subsequent forward flow into the ophthalmic arterial distribution, including the central retinal and posterior ciliary arteries, exposes the retinal and choroidal microvasculature to the risk of embolization. In addition, in unusual cases, some individuals have a branch between the external carotid artery and the ophthalmic vessels. Thus, it is also likely that fat droplets passed via the collateral artery to the ophthalmic arterial system.

Another factor in our case was the use of epinephrine combined with lidocaine for anesthesia. Adjunct use of a vasoconstrictor can reduce the risk of hemorrhage and congestion but can potentially cause vasospasm of the retinal and optic nerve circulation, thereby increasing the risk of occurrence of occlusion and hypoxia.

Severe visual loss after a general surgical procedure can be a devastating complication for both surgeon and patient. Because fat injection has become increasingly popular in recent years, physicians should be aware of such a possibility so that patients could be forewarned and thus present within a few hours rather than 1 day or more after visual loss.

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Disclosures
None.

References


Figure 1. Examination of the right fundus revealed that the retina had regained its normal color but left the atrophic macular spots, yellow flecks around the macula (red circle), the cotton wool spots (blue circle), attenuated retinal arterioles (red arrows), and optic disc atrophy (blue arrow).

Figure 2. Fundus fluorescein angiography showed narrowed and blocked arterioles with emboli in the perifoveolar regions (red arrows), demonstrating perfusion defects of the choroidal circulation (red circle).

Figure 3. Ocular coherence tomography imaging revealed retinal thinning, and the marked decrease in retinal thickness was shown numerically and by conventional color. ILM indicates internal limiting membrane; and RPE, retinal pigment epithelium.

Figure 4. A cross-sectional retinal image showed that the margin of each inner retinal layer (red arrow) was difficult to differentiate, and the decreased thickness of the inner layer was more obvious than that of the outer layer (blue arrow). The intraretinal cystic changes were seen within the foveal pit region (yellow arrow).
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