

Branch Retinal Artery Occlusion: A Complication of Iron-Deficiency Anemia in a Young Adult with a Rectal Carcinoid

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— We report a case of branch retinal artery occlusion (BRAO) in a patient with iron-deficiency anemia. Various ophthalmological and laboratory studies were performed. A 32-year-old man had a sudden decrease of vision in his left eye to counting fingers at 30 cm two days ago. The left fundus showed a cherry-red spot and milky-white edema, except for the upper temporal region of the macula, and an optic disc malformation. Fluorescein angiography revealed leakage from the disc and a slightly delayed filling time in the left eye but an arterial filling defect was not noted. The differential diagnosis in this young patient includes polycythemia, hypercoagulopathy, coagulation abnormalities, trauma, hypertension, and autoimmune diseases such as systemic lupus erythematosus. Laboratory examinations revealed no abnormalities except for iron-deficiency anemia. The patient was treated with stellate ganglion block, hyperbaric oxygen, and ferrous sulfate. His visual acuity never recovered to better than 0.08. He had a coincidental rectal carcinoid and the tumor was excised surgically. No metastasis was observed. BRAO can be a complication of anemic retinopathy and can lead to severe visual loss without early medication.
——— branch retinal artery occlusion; iron deficiency anemia; rectal carcinoid
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The ocular complications of anemia include hard exudates, cotton-wool patches, flame-shaped hemorrhages, and Roth spots in the fundus. These are all signs of anemic retinopathy. A MEDLINE search extracted three papers on cases with a central retinal vein occlusion (CRVO) in iron-defi-

ciency anemia (Kirkham et al. 1971; Kacer et al. 2001; Shibuya et al. 1995). However, retinal artery occlusion (RAO) has not been reported to be a complication of iron-deficiency anemia in young adults (Matsuoka et al. 1996).

RAOs are relatively common in patients over

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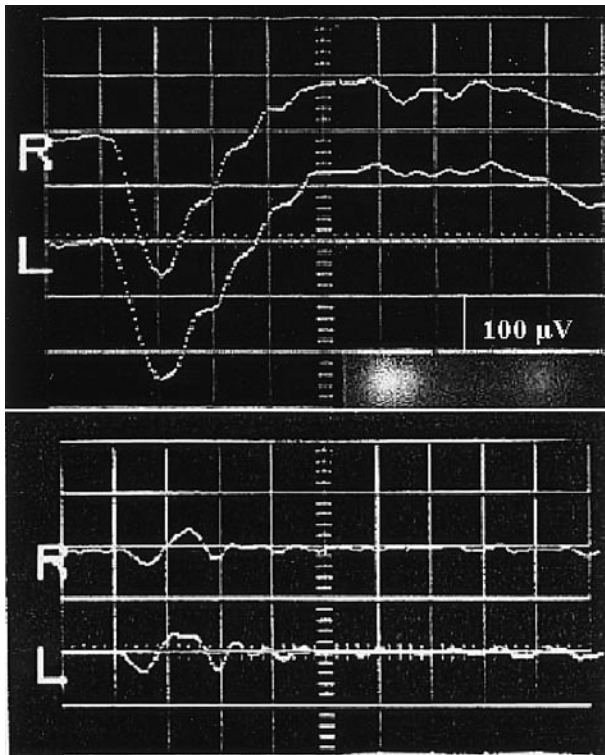


Fig. 2. Leading edges of full-field electroretinograms for two eyes (Upper). Extracted oscillatory potentials (Lower).

40 years of age, and have also been reported in young patients as a complication of hemoglobin sickle cell disease. RAOs are difficult to treat, although urokinase therapy, acetazolamide, hyperbaric oxygen, and stellate ganglion block have been used.

CASE REPORT

A 32-year-old man had a sudden decrease of vision in his left eye without ocular trauma on June 21, 2003 and was admitted on June 23. He had not received any medication during the 2 days. His family history was noncontributory, and his best-corrected visual acuity was 1.5 in the right eye and counting finger (CF) at 30 cm in the left eye. His intraocular pressures were normal, and slit-lamp examination revealed no inflammation in the anterior chamber or vitreous, in both eyes. No rubeosis irides was noted, and his other ocular history was unremarkable.

The right fundus appeared normal, but the left fundus showed a cherry-red spot and milky-

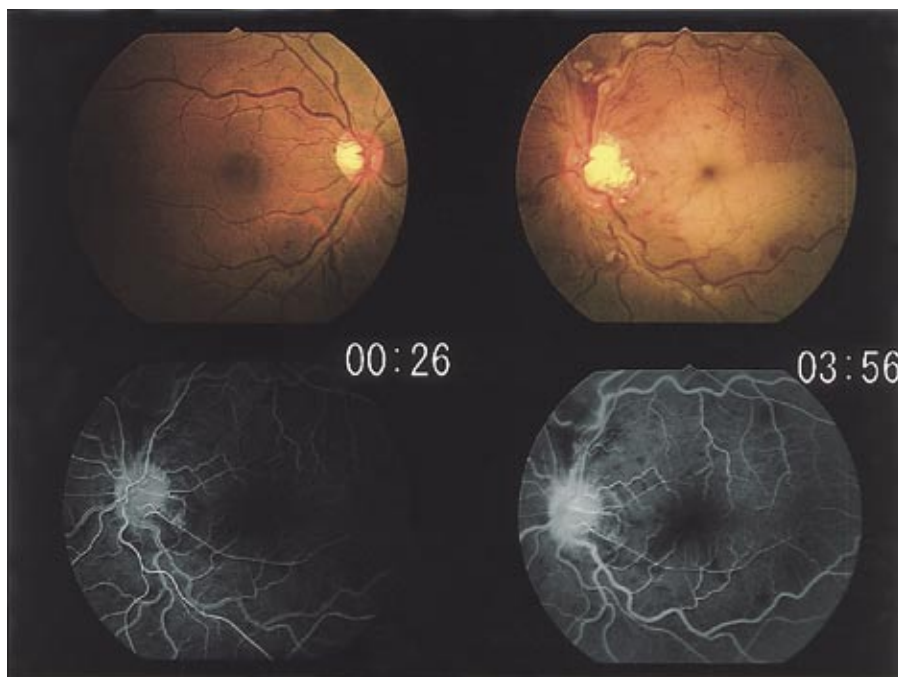


Fig. 1. Color fundus photograph (Top right) of left eye showing a cherry-red spot, milky-white lesion of macula, multiple intraretinal hemorrhages, and mild venous dilation. Fluorescein angiographic photograph (Bottom) of left eye showing leakage from the disc, slightly delayed filling time but no clear arterial filling defect with normal choroidal filling.

TABLE 1. *Laboratory findings*

	Patient	Normal Range in our lab.
Red blood cell count	325×10 ⁴ /μl	428 - 566×10 ⁴ /μl
Hemoglobin	6.4 g/100 ml	13.6 - 17.4 g/100 ml
Hematocrit	23.10%	41.7 - 52.5%
Corpuscular vol.	71.1 fl	87.8 - 102.4 fl
Corp. hemoglobin	27.7 pg	31.3 - 34.4 pg
Erythrocyte sedimentation rate	14 mm	<10 mm
White blood cell count	2200 /μl	3200 - 9600 /μl
Seg	44%	31 - 73%
Eosi	5%	0 - 7%
Baso	1%	0 - 3%
Lymp	42%	18 - 51%
Mono	8%	1 - 12%
Platelet count	25.2×10 ⁴ /μl	15.5 - 34.7×10 ⁴ /μl
Serum iron	10 μg/100 ml	39 - 185 μg/100 ml
Ferritin	5.2 μg/liter (June 30th)	20 - 300 μg/liter
Total iron binding capacity	539 μg/100 ml	256 - 424 μg/100 ml
CRP	0 mg/100 ml	0 - 0.2 mg/100 ml
GOT	27 IU/liter	12 - 30 IU/liter
GPT	17 IU/liter	8 - 35 IU/liter
Blood urea nitrogen	10 mg/100 ml	8 - 20 mg/100 ml
Creatine	0.7 mg/100 ml	0.4 - 1.0 mg/100 ml
Total protein	6.9 g/100 ml	6.5 - 8.2 g/100 ml
Albumin	4.5 g/100 ml	4.2 - 5.3 g/100 ml
Thrombo test	82%	70.1 - 999.9%
Hepaplastin test	84%	70.1 - 999.9%
Activated partial thromboplastin time	30.9 second	28 - 40 second
Antithrombin 3	127.00%	86 - 138%
Fibrinogen	187 mg/100 ml	210 - 380 mg/100 ml
D-dimer	0.4 mg/100 ml	0 - 0.3 mg/100 ml

white edema throughout the fundus except for the upper temporal macula region. There were also multiple intraretinal hemorrhages and mild venous dilation (Fig. 1, upper). The edema appeared to be caused by BRAO and not by complete central RAO (CRAO). Embolic materials were not seen. The left eye also has an optic disc malformation.

Fluorescein angiography revealed disc leakage and a slightly delayed filling time in the left eye but an arterial filling defect was not noted (Fig.

1, lower). The choroidal filling was normal.

Static perimetry showed a central scotoma in his left eye. The oscillatory potentials of the electroretinograms (ERGs) were reduced in both eyes as in CRAO (Fig. 2). Other abnormalities were not detected.

He had no heart disease, diabetes, or hypertension. From the laboratory findings, he was diagnosed as having iron-deficiency anemia (Table 1). The results of other tests including autoimmune disease factors (rheumatoid factor and anti-

nuclear antibody), blood pressure, chest x-ray, electrocardiography, ultrasonography of the neck, and computed tomography of the brain and orbits were negative or within the normal range.

The patient was treated with stellate ganglion block, hyperbaric oxygen, and ferrous sulfate. One week later, his visual acuity recovered to 0.04 and remained unchanged for one month later. The Gastroenterology Department found a tumor in his rectum which may cause bleeding. No metastasis was detected and tumor markers of carcinoembryonic antigen (CEA) and carbohydrate antigen 19-9 (CA19-9) were within the normal range. The fundus appeared almost normal but intraretinal hemorrhages were still present. His anemia improved gradually. Transanal partial excision of the tumor was performed on August 25, 2003. Histopathological examination revealed a carcinoid tumor with potential malignancy. Four months later, his visual acuity recovered to 0.08.

The differential diagnosis in this young patient includes polycythemia, hypercoagulopathy, coagulation abnormalities, trauma, hypertension, and autoimmune diseases, such as systemic lupus erythematosus. Laboratory examinations revealed no relevant abnormalities except for iron-deficiency anemia.

Although the pathogenesis of the BRAO was not determined, microvascular retinal occlusions may have occurred just as other vascular occlusions in patients with iron-deficiency anaemia (Hartfield et al. 1997). The retinochoroidal circulation can be disturbed temporarily in patients with abnormalities of the blood components. The malformation of left optic disc might have contributed to the ischemic changes. The right eye appeared to be normal with good visual acuity but

may also have had a temporary ischemic accident as demonstrated by the reduced ERGs.

A case of incomplete CRAO has been reported in a 13-year-old girl with iron-deficiency anemia following urokinase and ferrous sulfate therapy. She had a good recovery of visual acuity because of the early detection and treatment (Matsuoka et al. 1996). Our case has indicated that BRAO can be a complication of anemic retinopathy and can lead to severe visual loss without early medication.

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