Posterior Ischemic Optic Neuropathy Following Percutaneous Nephrolithotomy

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Purpose: To report a case of posterior ischemic optic neuropathy (PION) following percutaneous nephrolithotomy (PCNL).

Case Report: A 57-year-old man with history of diabetes mellitus, hyperlipidemia and mild anemia underwent PCNL for treatment of nephrolithiasis. He noticed painless visual loss in both eyes immediately after the procedure. Visual acuity was light perception, however ophthalmologic examinations were unremarkable and the optic discs were pink with no swelling. Visual fields were severely affected, but neuroimaging was normal. Within three months, visual acuity and visual fields improved dramatically but the optic discs became slightly pale.

Conclusion: This is the first report of PION following PCNL. PION is a rare cause of severe visual loss following surgery. Severe blood loss, hypotension, anemia and body position during surgery are the most important risk factors. Ophthalmologists, urologists and anesthesiologists should be aware of this condition and this rare possibility should be considered prior to surgery.

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INTRODUCTION

Posterior ischemic optic neuropathy (PION) or retrobulbar ischemic optic neuropathy is a type of optic nerve infarction which occurs posterior to the lamina cribrosa. This condition can lead to severe and sometimes bilateral visual loss. It is a rare entity and may occur: (1) after different types of non-ocular operations such as radical neck dissection and spine surgery; (2) as a complication of giant cell arteritis; and (3) in the setting of systemic vasculopathies without inflammation (non-arteritic).[1] Other conditions associated with PION include posterior-draining dural cavernous sinus fistula with arterial steal, blepharoplasty, laparoscopic nephrectomy, endoscopic sinus surgery, hemodialysis, migraine, herpes zoster ophthalmicus and polyarteritis nodosa.[2-9] Herein, we report a case of bilateral PION after percutaneous nephrolithotomy (PCNL) which to our knowledge has not been previously reported.

CASE REPORT

A 57-year-old man was referred with severe bilateral visual loss following PCNL. He had history of diabetes mellitus type II and hyperlipidemia for five years as well as mild anemia but did not smoke. He had undergone cataract

extraction in both eyes under retrobulbar anesthesia during the past year. The patient was on treatment with metformin for glycemic control.

The patient had undergone PCNL for nephrolithiasis on the right side under general anesthesia 24 hours before referral. Soon after surgery, the patient complained of painless loss of vision in both eyes with no other ophthalmic symptoms. On initial examination, visual acuity was light perception (LP) in both eyes, relative afferent pupillary defect (RAPD) was negative and on slitlamp examination, the patient was pseudophakic bilaterally. Intraocular pressure was in the normal range in both eyes. On fundus examination, both discs were pink with sharp borders and the only remarkable finding was a linear splinter hemorrhage in the superotemporal area of the macula in the right eye (Fig. 1).

The PCNL operation had lasted for two hours during which the patient had been in prone position with the face turned to the left. No significant blood loss or systemic hypotension had occurred during the procedure; according to the anesthesiology record, systolic blood pressure never dropped below 120 mmHg. Preoperative laboratory tests were as follows: fasting blood sugar 217 mg/dl, hematocrit 36.4%, hemoglobin 12 g/dl, red blood cell count $4.1\times10^6/mm^3$ and erythrocyte sedimentation rate 59 mm/hr.

Visual fields were obtained 48 hours postoperatively when visual acuity improved to counting fingers at 3 meters in both eyes and revealed a dense superior arcuate defect in the right eye and severe generalized depression in the left. The reliability indices were good and global indices were as follows: right eye, PSD= 9.29 and MD= -14.8; left eye, PSD= 6.61 and MD= -26.92 (Fig. 2). Orbital and brain CT scans were normal.

Seventy-two hours later, visual acuity gradually improved and reached 20/40 and 20/80 in the right and left eyes respectively. Anterior segment examinations, IOP and funduscopy were the same as the initial examination. After two months, visual acuity further improved to 20/30 in both eyes and mild disc pallor was seen in both eyes. Ishihara color vision tests were normal (10/10) bilaterally, and visual fields improved dramatically (Fig. 3).

DISCUSSION

The history, clinical course and paraclinical data in this patient are compatible with PION after PCNL which to the best of our knowledge has not been previously reported. In the course of ischemic optic neuropathy, the optic nerve is first damaged by ischemia followed by axonal necrosis. Depending on the affected segment of the optic nerve, ischemic optic neuropathies are subdivided into anterior ischemic optic neuropathy (AION) and PION. Although the underlying disorder in both types is insufficient blood supply, the pathophysiological mechanisms are not the same because of different vascular supplies in the anterior and posterior segments of the optic nerve.[10,11]

The optic nerve, anterior to the entry point of the central retinal artery, is only supplied by a pial capillary plexus which receives blood from central retinal and ophthalmic arteries.[12] At the lamina cribrosa, it is supplied by a vascular plexus, named the circle of Zinn-Haller, which receives blood from three sources including choroidal, short posterior ciliary, and meningeal arteries.[12,13] The posterior segment of optic nerve is only supplied by a posterior capillary plexus few branches of which penetrate the optic nerve substance causing the blood supply in the posterior segment of the optic nerve to be poorer than the anterior part. There are also some other differences between PION and AION. Structural anomalies such as small scleral canals and crowded optic nerve heads with small cups which are known risk factors for non-arteritic AION, have not been recognized as a risk factor for POIN. In cases with PION, Sadda et al[1] reported only 4% with such a structural configuration.

The three types of PION include: (1) non-arteritic; (2) arteritic; and (3) surgically induced (perioperative), shock induced or secondary to hemorrhage due to other causes.[1,14] Diagnostic criteria for PION include: (1) sudden onset of

decreased vision (ranging from no light perception to 20/20) and/or visual field defects, occurring immediately or shortly after reversal of general anesthesia; (2) positive RAPD (unless there is bilateral disease or there has been previous optic neuropathy in the fellow eye); (3) normal optic nerve head appearance at the onset of visual loss without swelling or peripapillary bleeding; (4) optic atrophy after 6-8 weeks; and (5) lack of compressive, toxic or inflammatory disorders.[1,14-16]

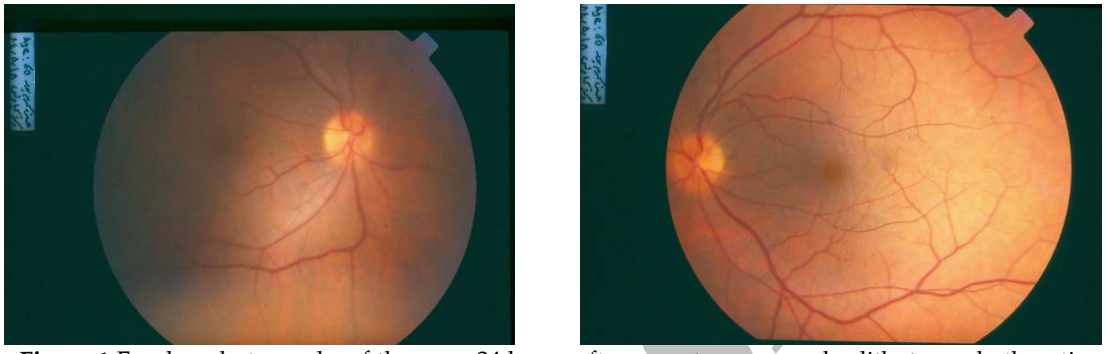

Figure 1 Fundus photographs of the eyes, 24 hours after percutaneous nephrolithotomy; both optic disks appear normal.

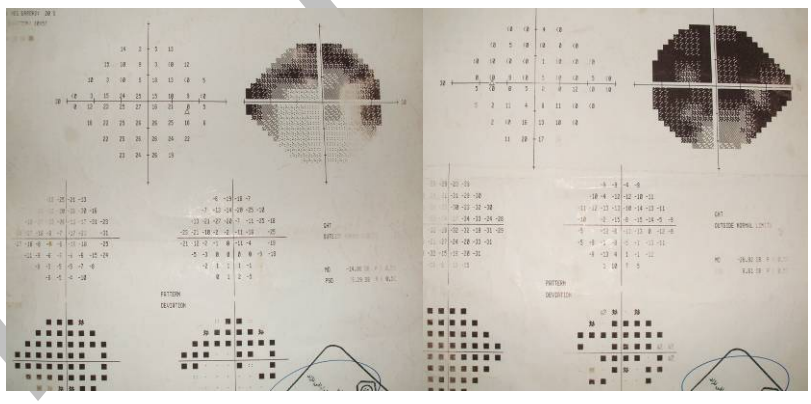

Figure 2 Visual fields of the patient 48 hours after percutaneous nephrolithotomy; a superior arcuate defect together with generalized depression is seen in the right eye and severe generalized depression is evident in the left eye.

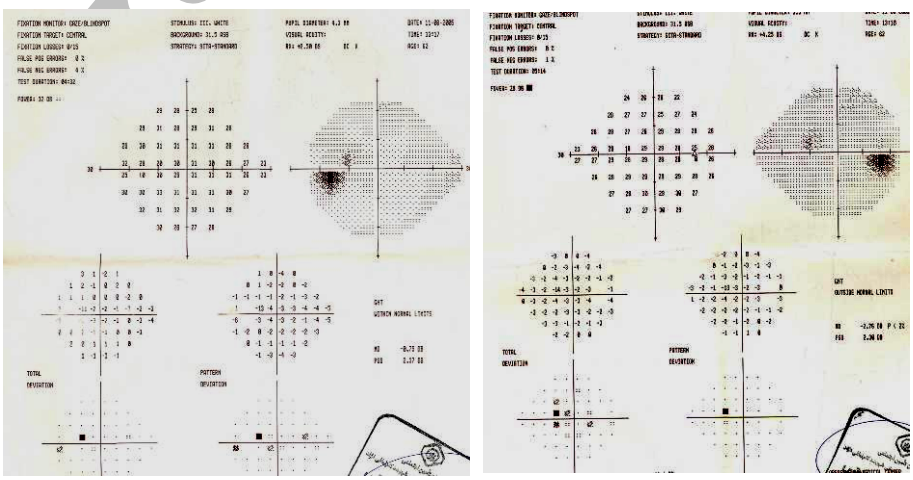

Figure 3 Visual fields of the patient, two months after percutaneous nephrolithotomy.

All three types of PION demonstrate color vision disturbances by pseudoisochromic plates in 70% (63-79%), however, normal color vision has been reported in 7% of surgically induced PION and 15% of the non-arteritic type. Different visual field defects such as altitudinal, central, arcuate, paracentral, cecocentral and generalized depression have been reported. The most common visual field defect was altitudinal in one study[1] and central in another.[15] Patients age has been reported from 18 to 88 years. Bilateral forms are more common in surgically induced and arteritic types (50%), which is in contrast to the non-arteritic type (21%).[1]

There are usually systemic vascular risk factors in patients with non-arteritic PION. The arteritic type almost always occurs in the setting of giant cell arteritis, although it can rarely accompany other types of vasculitis. Perioperative PION usually occurs after prolonged spine surgeries (50%), in which the patients are often younger and have no vascular diseases, but in cases following orthopedic, abdominal, heart and ocular procedures, there usually are other risk factors such as hypertension, diabetes mellitus, smoking, hypercholesterolemia, congestive heart failure, coronary artery disease and cerebrovascular accident, resembling non-arteritic PION.[1]

In post-surgical PION, bilateral involvement is common and visual loss is more severe. In a series of patients with post-surgical PION, 58% had visual acuity of hand motions or worse which remained unchanged in 44% with long-term follow up. Corresponding figures for non-arteritic PION were 26% and 17%, respectively and 42% had visual acuity of 20/60 or better in long-term follow up.[1]

In our patient, initial visual acuity was as poor as LP, however significant improvement to 20/30 was observed after two months. This might be due to the relatively short duration of surgery (2 hours), lack of massive bleeding or significant systemic hypotension during the procedure and adequate homodynamic rehabilitation after surgery. Consequently, a large number of axons regained their normal blood supply and probably few of them underwent ischemic necrosis.

Major risk factors for perioperative PION are persistent pre- or intraoperative anemia, hypovolemia and hypotension during surgery, and facial swelling and edema.[17,18] It is suggested that direct pressure on the globe due to face-down position can be a possible cause, however this position seems to precipitate AION or central retinal artery occlusion rather than PION. In a series of 72 cases of PION reported by Sadda et al[1] no patient had the latter risk factor, however, 82% had a systemic vascular risk factor. Our patient also had hyperlipidemia and diabetes mellitus.

Considering the fact that there is no definite treatment for PION, preventive measures are vital because of the high rate of bilateral involvement, severity of the condition and the possibility of irreversible visual loss. The prognosis of arteritic and postoperative PION is usually poor which is in contrast to non-arteritic PION.[1,16]

In conclusion, although PION is rare, surgeons and anesthesiologists should be aware of this potentially blinding condition which may occur following heart and spine surgeries and any procedure with massive hemorrhage and blood loss. The case presented in this report is the first following PCNL. The possibility of PION should be considered in individuals with perioperative risk factors. Preventive measures such as modification of systemic vascular risk factors and hemodynamic stability before, during and after any major procedure are vital to decrease the risk of this condition.

REFERENCES

1. Sadda SR, Nee M, Miller NR, Biousse V, Newman NJ, Kouzi S. Clinical spectrum of posterior ischemic optic neuropathy. *Am J Ophthalmol* 2001;132:743-750.
2. Hashimoto M, Ohtsuka K, Suzuki Y, Hoyt WF. A case of posterior ischemic optic neuropathy in a posterior-draining dural cavernous sinus fistula. *J Neuroophthalmol* 2005;25:176-179.
3. Kordic H, Flammer J, Mironow A, Killer HF. Perioperative posterior ischemic optic

neuropathy as a rare complication of blepharoplasty. *Ophthalmologica* 2005;219:185-188.
4. Metwalli AR, Davis RG, Donovan JF. Visual impairment after laparoscopic donor nephrectomy. *J Endourol* 2004;18:888-890.
5. Huang TW, Liu CM, Cheng PW, Yang CH. Posterior ischemic optic neuropathy following endoscopic sinus surgery. *Otolaryngol Head Neck Surg* 2003;129:448-450.
6. Buono LM, Foroozan R, Savino PJ, Danesh-Meyer HV, Stanescu D. Posterior ischemic optic neuropathy after hemodialysis. *Ophthalmology* 2003;110:1216-1218.
7. Lee AG, Brazis PW, Miller NR. Posterior ischemic optic neuropathy associated with migraine. *Headache* 1996;36:506-510.
8. Kothe AC, Flanagan J, Trevino RC. True posterior ischemic optic neuropathy associated with herpes zoster ophthalmicus. *Optom Vis Sci* 1990;67:845-849.
9. Saraux H, Le Hoang P, Laroche L. Anterior and posterior acute ischemic optic neuropathy related to poly-arteritis nodosa (author's transl). *J Fr Ophthalmol* 1982;5:55-61.[Abstract]
10. Hayreh SS. Posterior ischemic optic neuropathy. *Ophthalmologica* 1981;182:29-41.
11. Hayreh SS. The optic nerve head circulation in health and disease. *Exp Eye Res* 1995;61:259-272.
12. Isayama Y, Hiramatsu K, Asakura S, Takahashi T. Posterior ischemic optic neuropathy. I. Blood supply of the optic nerve. *Ophthalmologica* 1983;:86:197-203.
13. Olver JM, Spalton DJ, McCartney AC. Microvascular study of the retrolaminar optic nerve in man: the possible significance in anterior ischemic optic neuropathy. *Eye* 1990;4:7-24.
14. Kelman SE. Ischemic optic neuropathies. Walsh & Hoy's clinical Neuro-Ophthalmology. 5th Ed Philadelphia, Pennsylvania: Williams & Wilkins; 2005: 370-374.
15. Hayreh SS. Posterior ischaemic optic neuropathy: clinical features, pathogenesis, and management. *Eye* 2004;18:1188-1206.
16. Buono LM, Foroozan R. Perioperative posterior ischemic optic neuropathy: review of the literature. *Surv Ophthalmol* 2005;50:15-26.
17. Dunker S, Hsu HY, Sebag J, Sadun AA. Perioperative risk factors for posterior ischemic optic neuropathy. *J Am Coll Surg* 2002;194:705-710.
18. Lee AG. Ischemic optic neuropathy following lumbar spine surgery; case report. *J Neurosurg* 1995:83:348-349.