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Case Study

Renal Artery Stenosis presenting as Anterior Ischemic Optic Neuropathy (AION) in young

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ABSTRACT

Anterior ischemic optic neuropathy is the most common cause of acute optic neuropathy in older age groups and very rare below 50 years of age. The causes can be non-arteritic (NAION) or arteritic (AION), the latter being associated with giant cell arteritis (GCA or Temporal arteritis). In the majority of cases it is idiopathic, but known risk factors are hypertension (HTN), diabetes (DM), Obstructive Sleep apnea (OSA) and drugs. Visual loss secondary to NAION as a presenting feature of renal artery stenosis is rarely seen. Herein we describe a young male patient who presented with progressive loss of vision in his left eye which was acute in onset. He improved over few weeks with a vision of 20/20(right eye) and 20/80(left eye) and was discharged home.

Keywords: Anterior ischemic optic neuropathy, Blindness, Hypertension, ischemic, Renovascular.

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INTRODUCTION

Secondary hypertension is defined as increased systemic blood pressure (BP) due to an identifiable cause. Only 5–10% of patients suffering from arterial hypertension have a secondary form, whereas the vast majority have essential (idiopathic/primary) HTN.[1] It is not cost effective to search for secondary causes of hypertension in every patient and quite a few times they are missed. The prevalence of secondary HTN and the most common etiologies vary by age group. The most common causes of secondary hypertension are listed in Table 2. In secondary HTN target pressures are easier to achieve than in primary HTN.[2]

Renovascular disease (RVD) is one of the most common causes of secondary HTN below 30 years of age and is present in 10-40% of patients with End stage renal disease (ESRD).[3] The presenting signs and symptoms of renovascular hypertension is generally difficult to control hypertension (Requiring more than 3 Anti HTN drugs), abdominal bruit, accelerated HTN, azotemia, early/late onset HTN (<30yrs/>55yrs) and/or signs of other end organ damage. End organ damage can manifest as cardiac or renal or visual problems. The visual disturbances are mainly restricted to hypertensive changes. NAION as a presenting feature is rare. Herein we describe a case who presented with non-arteritic anterior ischemic optic neuropathy [NAION] and was discharged home with a good recovery in vision.

CASE REPORT

23-year-old male with no co-morbidities or previous hospitalizations, was referred from a nearby ophthalmology hospital with history of sudden onset of unilateral (left eye) progressive painless loss of vision over the last 4 days. He was a smoker but without history of drug abuse. There was no history of fever/ headache/cough/syncope/tinnitus /palpitation/ chest pain/seizures/ breathlessness/increased lacrimation/neurological deficits. On examination, patient was conscious oriented with no neurological deficits with GCS of 15/15, His pulse rate was 84/min and blood pressure was 190/140. ECG showed left ventricular hypertrophy and poor R wave progression. His systemic examination was essentially normal. Ophthalmologic examination revealed a visual acuity of 20/40 parts by Snellen's Visual Acuity Chart in right eye and counting fingers close to face with accurate projection of rays in the left eye. Colour vision was normal in the right eye and absent in the left eye. Right eye pupil was 3mm round, regular and reacting and left eye had a relative afferent pupillary defect (RAPD). Fundus in both eyes showed clear media with blurring of disc margins and disc oedema of up to 4D. Disc was hyperemic with tortuous vessels on disc and in surrounding areas. Generalised arteriolar attenuation was present. No AV crossing changes were seen. Macular and peripapillary folds were seen and foveal reflex was absent, suggestive of severe macular oedema. Patient was diagnosed to have bilateral papilledema with macular star and arteriolar attenuation which was present more in the left eye than in the right eye (Fig 1). Since the patient had sudden visual loss and RAPD, Visual Evoked potential (VEP) was ordered. VEP revealed prolonged P latency and reduced amplitude in both eyes, suggestive of involvement of optic nerves.
In view of RAPD, VEP reports and fundus picture, a diagnosis of anterior ischemic optic neuropathy of both eyes was made.

An initial diagnosis of Hypertensive Urgency was made and started on labetalol infusion. He was started on IV Methylprednisolone 500mg BD for 3 days followed by tapering dose of oral steroids for 11 days.

His initial laboratory reports were as in Table 1. MRI brain was essentially normal. Renal artery Doppler revealed small right kidney with long segment renal artery stenosis(RAS). Nephrologist opined as probably renovasuclar HTN and requested for serum Renin/serum Aldosterone and 24 hours urine protein with creatinine. A 2D echo revealed an ejection fraction (EF) of 58% with concentric left ventricular hypertrophy (LVH) and grade 1 diastolic dysfunction. CT angiography revealed a severe long segment renal artery stenosis. A DTPA scan showed right kidney at 10% and hence angioplasty was deferred.

Patients BP was under control with oral drugs and his vision started to show signs of improvement. Patient was discharged and over a period of 3 to 4 weeks patient's vision improved to 20/20 N6 in right eye and 20/80 N6 in left eye, with fundus findings of resolving papilledema (Fig 2).

On his last OPD visit 6 months later his left eye vision was 20/40 and his optic disc appeared pale, suggestive of partial optic atrophy. Blood pressure was well under control.

DISCUSSION

NAION, presumably the result of hypoperfusion and infarction of the anterior optic nerve,[4] typically occurs after 55yrs and is rare among youngsters. The annual incidence NAION is estimated at 2.3 to 10.2 per 100, 000 persons aged

50yrs and older and 0.54 per 100,000 for all ages.[5,6] NAION is manifested as isolated, sudden, painless, monocular vision loss with edema of the optic disc. Progressive worsening of vision over a period of a few days or a few weeks, presumably related to worsening ischemia with local compartment syndrome associated with the disc edema is common.[7] Many studies on NAION including the multicenter Ischemic Optic Neuropathy Decompression Trial (IONDT) have excluded patients younger than 50 years because of the presumption that NAION is a rare entity in younger patients. [5]

It has been suggested that when anterior ischemic optic neuropathy occurs in a young patient, it is often the result of an underlying condition, predisposing the patient to vascular insufficiency, such as diabetes mellitus, accelerated hypertension, end-stage renal disease with dialysis, hypotension or anemia.

In patients younger than 50years, diabetes, hypertension and hypercholesterolemia are associated more strongly with anterior ischemic optic neuropathy than in older individuals. Peri-operative ischemic optic neuropathy or anterior ischemic optic neuropathy secondary to hyper-coagulable states also have been described in younger patients. Most anterior ischemic optic neuropathy series of younger patients are small and relatively little is known about this group of patients. [5,6,10] Younger patients with anterior ischemic optic neuropathy have better visual acuity outcomes and higher risk of fellow eye involvement, both of which were seen in our patient.

In one study published in the year 2007 [9] it was concluded that anterior ischemic optic neuropathy in younger patients is not uncommon and represents 25% of anterior ischemic optic neuropathy patients in a tertiary neuro-opthalmic service. Except for giant cell arteritis, ocular and systemic risk factors and associated disorders are similar to those described in older anterior ischemic optic neuropathy patients.

As per ACC/AHA (American College of Cardiology/ American Heart Association) Clinical Practice Guidelines for screening of RAS/RVD, duplex ultrasonography, computed tomographic angiography (CTA), and magnetic resonance angiography (MRA) all receive a class I indication (level B evidence).[8] Angiography, though a gold standard, is invasive and atheromatous embolisation of renal arteries is a possibility with further worsening of renal function.

Our patient presented with progressive loss of vision in with left eye which was confirmed on fundoscopy. Right eye though there was no visual disturbances fundoscopy revealed arteriolar attenuation suggestive of bilateral involvement. CT angiography revealed a long segment stenosis and a DTPA scan showing 10% functioning kidney which meant the kidney was beyond salvage and hence no aggressive interventions were planned.

In conclusion, Renal artery stenosis can present as non-arteritic anterior ischemic optic neuropathy which could be one of the forms of end organ damage. A high index of suspicion, a low threshold for renal Doppler in patients with secondary HTN will help in early diagnosis of renal artery stenosis. Non-arteritic anterior ischemic optic neuropathy, though a rare entity in the young, if detected early can salvage the vision.

Table 1 – Investigations

	INV	**DAY 1**	**DAY 2**	**DAY3**	**DAY4**	**DAY5**	**DAY6**	**DAY7**
1	Hemoglobin (gm/dl)	17.2g/dl						
2	PCV	46.8%						
3	TLC (cells/mm^3)	9530						
4	Plateletcount(cells/mm^3	280000						
5	ESR (mg/dl)	2 mm/hr						
6	Serum Creatinine(mg/dl)	0.89				0.91	0.95	
7	BUN (mg/dl)	13.3				17.7	13.1	
8	Uric Acid (mg/dl)	5.2				5.7	4.7	
9	Sodium(mEq/l)	132	131	134	131	134	134	134
10	Potassium (mEq/l)	2.7	3.21.	3.43	3.64	3.33	3.12	3.01
11	Chloride (mEq/l)	98	88.8	89.8	86	87.4	89.4	89.4

12	Urine Albumin	3+						
13	Urine Sugar	Nil						
14	Urine WBC	1-2						
15	Urine RBC	0						
16	CRP (mg/dl)	0.29						
17	CSF		Normal					
18	Ser Calcium(mg/dl)		9.82					
19	Plasma Renin activity (ng/ml/hour)			36.9				
20	Ser TSH (microIU/ml)			0.482				
21	Aldosterone (ng/dl)			23.6				
23	ANA PROFILE					Normal		

Table -2 BP, blood pressure; Ca2+, calcium; K+, potassium; PO4, phosphate; CT, computer tomography; ARR, aldosterone – renin ratio; Na+, sodium; AF, atrial fibrillation; TSH, thyroid-stimulating hormone; fT4, free thyroxine; fT3, free triiodothyronine.
aPrevalence in hypertensive patients.
bPrevalence in patients with resistant hypertension.

Secondary cause	Prevalence[a]	Prevalence[b]	History	Screening	Clinical findings	Laboratory findings
Obstructive sleep apnoea	>5–15%	>30%	Snoring, daytime sleepiness, morning headache, irritability	Screening questionnaire; polysomnography	↑ neck circumference; obesity; peripheral oedema	Not specific
Renal parenchymal disease	1.6–8.0%	2–10%	Loss of good BP-control; diabetes; smoking; generalized atherosclerosis; previous renal failure; nocturia	Creatinine, ultrasound of the kidney	Peripheral oedema; pallor; loss of muscle mass	↑ Creatinine, proteinuria; ↓ Ca^{2+}, ↑ K^+, ↑ PO_4
Renal artery stenosis	1.0–8.0%	2.5–20%	Generalized atherosclerosis; diabetes; smoking; recurrent flush pulmonary oedema	Duplex, or CT, or MRI, or angiography (drive by)	Abdominal bruits; peripheral vascular disease;	Secondary aldosteronism: ARR →; ↓ K^+; ↓ Na^+
Primary aldosteronism	1.4–10%	6–23%	Fatigue; constipation; polyuria, polydipsia	Aldosterone–renin ratio (ARR)	Muscle weakness	↓ K^+; ARR ↑
Thyroid disease	1–2%	1–3%	*Hyperthyreoidism*; palpitations, weight loss, anxiety, heat intolerance; *Hypothyreodism*; weight gain, fatigue, obstipation	TSH	*Hyperthyreodism*: tachycardia, AF; accentuated heart sounds; exophthalmus; *Hypothyreodism*; Bradycardia; muscle weakness; myxoedema	*Hyperthyreodism*: TSH↓; fT4 and/or fT3 ↑; *Hypothyreodism*: TSH ↑; fT4 ↓; cholesterol ↑
Cushing's Syndrome	0.5%	<1.0%	Weight gain; impotence; fatigue; psychological changes; polydypsia and polyuria	24 h urinary cortisol; dexamethasone testing	Obesity, hirsutism, skin atrophy, Striae rubrae, muscle weakness, osteopenia	24 h urinary; cortisol ↑; Glucose↑; Cholesterol ↑; K^+↓
Phaeochromocytoma	0.2–0.5%	<1%	Headache; palpitations; flushing; anxiety	Plasma-metanephrines; 24 h urinary catecholamine	The 5 'Ps': paroxysmal hypertension; pounding headache; perspiration; palpitations; pallor	metanephrines ↑
Coarctation of the aorta	<1%	<1%	Headache; nose bleeding; leg weakness or claudicatio	Cardiac ultrasound	Different BP (≥20/10 mmHg) between upper–lower extremities and/or between right–left arm; ↓ and delayed femoral pulsations; interscapular ejection murmur; rib notching on chest Rx	Not specific

Kaplan's, Clinical hypertension, Tenth Edition, 2010, Lippincott Williams & Wilkins, p. 363

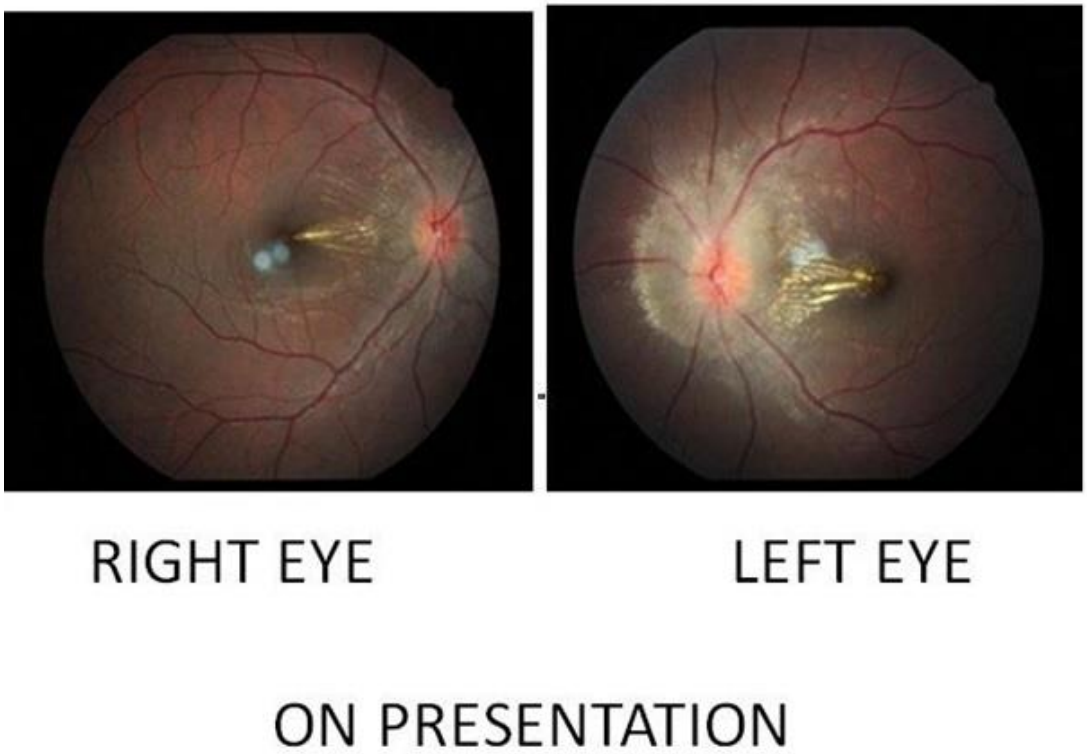

Figure 1 – Fundoscopy on initial admission showing bilateral papilledema with macular star and arteriolar attenuation - left eye more than right eye.

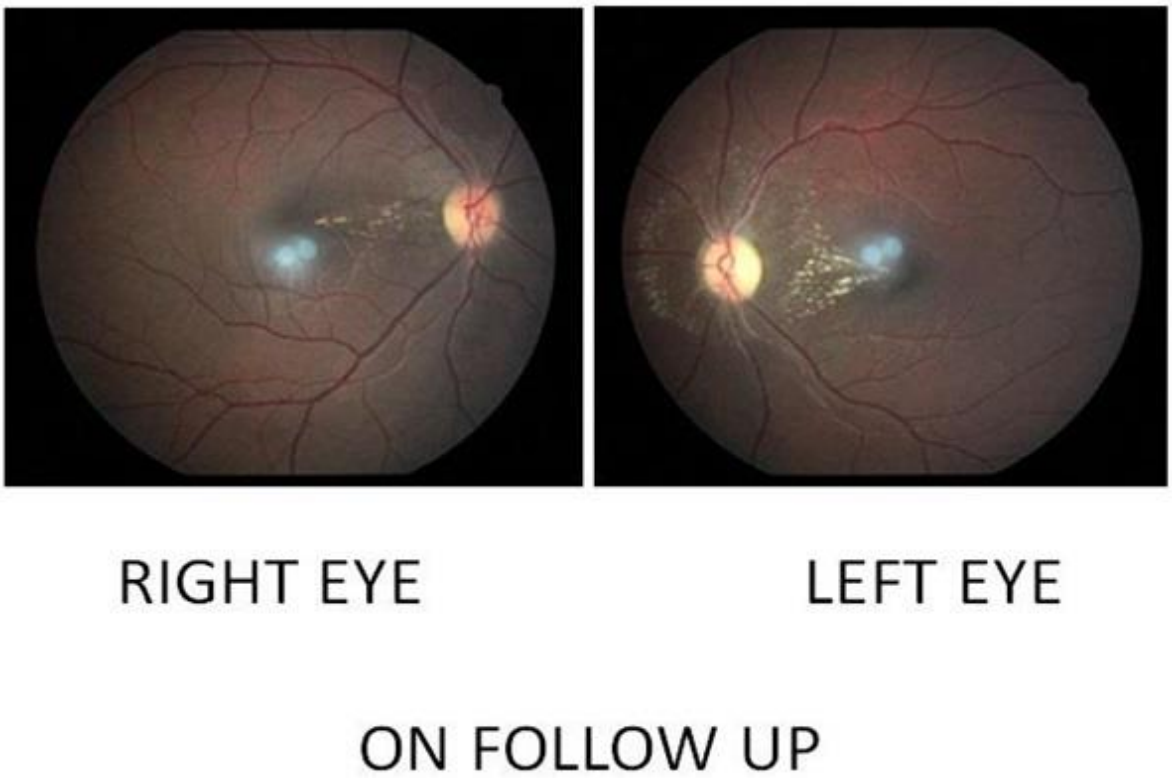

Figure 2 – Fundoscopy at discharge showing resolution of initial findings.

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