

Trial of Square and Round Edge Intraocular Lens on Preventing Posterior Capsule Opacification

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Purpose :

To clarify the effects of square and round edge Intraocular lens (IOL) on preventing posterior capsule opacification (PCO).

Patients and Methods :

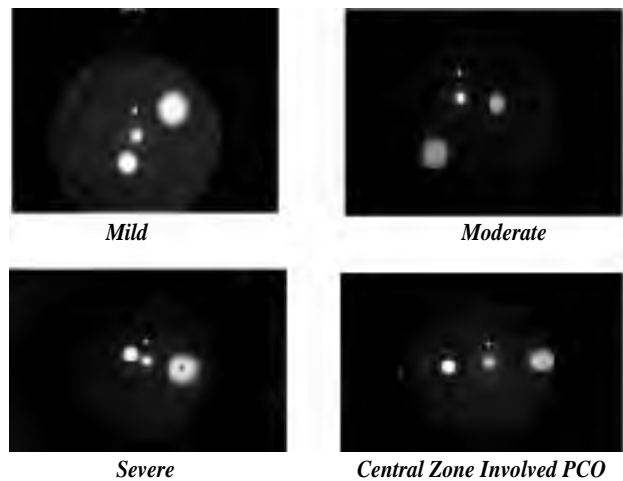
In this study, square edge PMMA IOL was implanted in one eye and round edge PMMA IOL was implanted in fellow eye of 15 patients after Phacoemulsification. Those with Diabetes, Glaucoma, Exfoliation syndrome, Uveitis and Pupil size smaller than 6.0mm, past history of ocular surgery were excluded from study. All patients are between the age of 55 to 60 yrs and received same brand of IOL in the bag with well centered capsulorrhexis covering the IOL optic by one surgeon applying same technique.

Post operatively, all patients received similar routine medications. At different follow-up interval PCO was assessed on morphological scoring systems that are independent of visual acuity testing or other exclusive subjective parameter, like glare, contrast sensitivity and optical aberration. In this morphological scoring system PCO was divided into two broad groups. One is central zone free (Peripheral) PCO and another is central zone involved (Central 5.0mm) PCO. Central Zone free PCO again divided into mild (1 quadrant), moderate (2 quadrant) and severe (> 2 quadrant) involvement of PCO.

Results :

Table shows changes of PCO values in each group at the end of 2 yr follow-up. Evaluating the picture of PCO at the end of two year, it appears that with sharp optic edge IOL, PCO was mostly inhibited, which is less marked in case of round optic edge IOL.

PCO	Square edge IOL	Round edge IOL
Central Zone Free PCO:		
Mild	4	4
Moderate	2	3
Severe	0	1
Central Zone Involved PCO	0	1



Discussion :

Posterior capsule opacification (PCO) is the most frustrating frequent late complication of modern cataract surgery with intraocular lens (IOL) implantation². The rate of PCO is variable reportedly between 3 and 50% during the 1st 5 years after cataract surgery³. The reason for this variation is due to systemic and ocular disease, patients age, surgical methods, IOL materials and designs⁴.

The influence of IOL design on PCO has been extensively described based on experimental work by Nishi¹¹

Schaumberg DA and coauthors report sharp optic edges seem the most significant factor in preventing PCO and currently receiving more attention than IOL material in PCO prevention.

Okihiro Nishi⁹ described the histologic section of an endocapsular IOL that had dislocated posteriorly into the vitreous and was removed showed that migrating lens epithelial cells (LECs) were inhibited at the distinct capsular

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bend created by the Plano-convex optic. They also observed that LECs were inhibited on rectangular shaped well bottoms in culture. All these findings suggest that a capsular bend or angle created by a capsule tensionring or on IOL can induce contact inhibition to LECs and, therefore, reduce PCO. (Fig-1 & Fig-2)



Fig-1



Fig-2

In this study, on the preventive effect of an IOL on PCO, the sharp bend optic IOL created a tight contact between the posterior lens capsule and IOL surface and thus inhibit the migration of LECs towards the centre and central PCO was mostly inhibited which is less marked in case of round optic IOL in this study.

Estimation of PCO on morphological system is not reliable though it is less expensive and less instrument dependent.

Further study is needed with longtime follow-up of at least 5 years and large sample will be helpful to get the information about the role of optic geometry on preventing PCO.

The sharp optic edge exerts greater compression on the posterior capsule and thus enhance the barrier effect provided by this optic geometry.

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Portal Vein Thrombosis in Polycythaemia Vera: A Case Report

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ABSTRACT

Portal vein thrombosis, commonly associated with cirrhosis of liver and thrombophilia, is one of the rare causes of sudden severe abdominal pain. In the absence of non-cirrhotic non-malignant extra hepatic portal vein thrombosis (EPVT), myeloproliferative disease (MPD) should always be considered. We describe a case of Polycythaemia Vera (PV) presenting with severe abdominal pain due to portal vein thrombosis. The patient was prescribed life-long warfarin, beta adrenergic blocker and aspirin. Due to uncontrolled platelet and white blood cell count he was later prescribed cytoreductive therapy.

Key word: Polycythaemia, portal vein thrombosis

Case Report:

A 26 year old youngman presented with the complaints of sudden severe abdominal pain for 4 days in August, 2007. It was dull aching in nature, associated with nausea and vomiting not accompanied by haematemesis and melaena. He was afebrile and gave no history of dark urine, headache, seizure, loss of consciousness, generalized itching or erythromelalgia.

Physical examination revealed congested lower palpebral conjunctiva without facial plethora. He was non icteric. Spleen was palpable 10 cm from left costal margin in non-tender soft abdomen. Ascites was absent. Initial investigations and treatment with anticoagulation was done in the dept. of Gastroenterology in collaboration with the dept of Haematology and Vascular Surgery.

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At presentation lab investigation revealed Hb(Haemoglobin) 16.5g/dl, WBC 11.0 x10⁹/L, ESR 10 mm1st hour, Hct(Haematocrit) 0.51, Platelet 350 x10⁹/L, serum bilirubin 1.8 g/dl, serum total protein 8.1 g/dl, and albumin 4.5g/dl.

The first USG (Ultrasonogram) done on September 9, 2007 (Fig-1) showed liver with homogenous echo texture with increased periportal echogenicity and echogenic structures noted within the portal vein along with cavernous formation. Duplex scan confirmed the echogenic structure to be thrombus in the lumen of main portal trunk causing partial obliteration (Fig-2). Mean velocity within the cavernous was 30.6 cm/sec. Thrombus was still present within the portal vein after anticoagulation(Fig-3), however, cavernous velocity was increased (35.6 cm/sec). Endoscopy upper GIT showed grade I esophageal varices with duodenal erosion. Later on he was treated with warfarin 5 mg and Propranolol 20 mg Daily. Two months later a follow up Color Doppler showed re-canalization of portal vein, a normal hepatic vein flow and development of collateral vessels in hilar and peripancreatic region along with moderate splenomegaly (18.5 cm).

Differential diagnoses:

The diagnosis of acute EPVT was confirmed by USG. Causes of abdominal pain like appendicitis, pancreatitis, cholecystitis, cholangitis and inflammatory bowel diseases were excluded by history, examination and USG. Liver function tests including the viral markers and renal function were also normal. Amongst other causes of systemic prothrombotic states were inherited thrombophilia, MPD, so our differential diagnoses were inherited thrombophilia and MPD.

Coagulation screening test were done PT(Prothrombin time) 12.4s, APTT(activated partial thromboplastin time) 35.5s, TT (Thrombin time) 12s, Fibrinogen 200mg/dl were found to be normal. ATIII (Anti-thrombin III, 118.6%) and APCR (Activated protein C resistance, 2.45) were found negative, protein C (121.7%) and protein S (76.4%) were also normal. Ham's test, sucrose lysis test and sickle test were negative (Table-I).

Arterial blood gas analysis was normal. Bone marrow aspiration study revealed panmyelosis with predominant

erythroid and megakaryocytic hyperplasia. Finally, we requested JAK2 kinase mutation study and FISH for bcr-abl in a reference laboratory abroad. The patient was found to be positive for 2343G-T mutation in JAK 2 gene resulting in amino acid change Valine in 617 phenyl alanine. FISH for bcr-abl was negative.

Discussion of Pathology:

Polycythaemia Vera is a clonal haemopoietic chronic myeloproliferative stem cell disorder involving hyperplasia of all three major cell lineages. The main pathological feature is expansion of total red cell mass with independent of erythropoietin, elevation in platelets and neutrophils^{17,18}. The median age of onset is 55-60 years¹ with cases under the age of 20 being very rare [18]. The JAK2 V617F mutation increasing the proliferative capacity of cells is present in all erythropoietin-independent erythroid colonies

Table 1 : Molecular analysis for inherited thrombophilia results were as follows:

Mutations analyses	Patient DNA	Result
Factor V 1691	Factor V 1691G/G	Normal
Factor II 20210	Factor II 20210 G/G	Normal
MTHFR677	MTHFR677C/C	Normal

Clinical diagnosis:

Polycythaemia Vera with portal vein thrombosis and portal hypertension

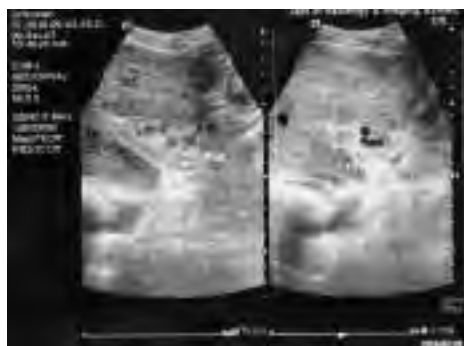


Figure 1: Initial sonographic finding



Figure 2: Color Doppler portal venous system



Figure 3: Follow Up Doppler after anticoagulation

in PV. The mutation is present in 90-95 % of PV patients and in approximately 50 percent of Essential Thrombocytosis and IMF (Idiopathic Myelofibrosis) patients¹. Mutation in the gene for Janus Kinase 2 (JAK2V617F) on chromosome 9p24 (4-7) has gained popularity as a noninvasive diagnostic tool²⁻⁴. The major symptoms related to hypertension and vascular abnormalities are caused by the increased red cell mass. An episode of venous or arterial thrombosis such as deep vein thrombosis, MI or stroke may be the first manifestation in about 25% cases. Mesenteric, hepato-portal or splenic vein thrombosis should always raise suspicion of PV as a possible cause¹⁸.

WHO definition of absolute erythrocytosis as haemoglobin levels >18.5 g/dL (haematocrit > 55.5%) in a man or >16.5 g/dL (haematocrit > 49.5%) in a woman has been questioned by several authorities for its low efficiency (It identified absolute erythrocytosis in only 35% of male PV patients and 63% of the females) and high false positive rate (14% in male and 35% in female)⁵. The clinical course of polycythaemia vera (PV) is marked by a high incidence of thrombotic complications; fibrotic and leukaemic disease transformations are additional causes of morbidity and mortality⁶. Increasing age and a history of vascular events have consistently proven to be independent predictors of thrombosis in patients with PV⁷. Leukocytosis has been identified as risk factor for thrombotic events specially MI (Myocardial infarction)⁷⁻⁸. No study to date has demonstrated a significant correlation between platelet number or function and thrombosis⁹⁻¹⁰.

Discussion of management:

Patient treated with aspirin 75mg, warfarin and propranolol. The patient was treated with aspirin 75mg, warfarin and propranolol and was advised monthly follow up. At the first follow up visit, lab values were Hb 15 g/dl, Hct 0.45, WBC 14 x10⁹/ L, Platelet 500 x10⁹/ L and INR 2.0. Since then he had been under regular follow up and the parameters were within control. A CBC on May, 2008, showed increased number of basophils (5%), nucleated red cells and platelet 700 x10⁹/ L. In next visit platelet and WBC count continued

to rise with persistent basophilia and nucleated red cells in film. At that time he complained of erythromelalgia, redness of sole and palm and intense itching. A cyto-reductive drug, Hydroxyurea (HU) 500 mg twice daily was added, dose of Warfarin increased to 6.25 mg daily and follow up was recommended after two weeks. Later on total count came down to 11×10^9 /L, platelet count to 300×10^9 /L and Hct 0.39. He was kept on HU at same dose for another 2 weeks and then it was gradually tapered to a maintenance dose of 500mg daily. Other previous medications were continued at same doses during that period.

In treating acute EPVT, our target is recanalizing the obstructed veins and preventing extension of the thrombus. The current recommendation is oral anticoagulation for at least 3 months aiming to keep level INR of 2.0-3.0¹¹. Special attention to clinical and laboratory monitoring is needed because of potentially greater bleeding risk and unpredictable drug influences in MPD patients¹². Life-long warfarin prophylaxis has been advocated for patients with abdominal venous thrombosis¹³. No study showed critical benefit of beta blocker or endoscopic therapy for oesophageal varices in EPVT patients, however, we recommended this patient beta blocker.

Phlebotomy is the mainstay of treatment provided the patient can tolerate with a target haematocrit of 0.45¹⁴. Low dose aspirin reduces the risk of thrombotic events as such lower incidences of cardiovascular death, myocardial infarction, stroke, and major venous thromboembolism are observed⁶. The recommended daily dose is 75 mg¹⁹.

HU, the most widely used cytoreductive drug, reduces both haematocrit and platelet count which makes it the drug of choice in high-risk patients¹⁵. The initial dose of HU is 15 to 20 mg/kg/day and maintenance dose should be administered to keep haematocrit at response levels without reducing WBC count values $<3 \times 10^9$ /L⁸. In one study it was found that HU alone did not enhance the risk of leukemia in comparison with patients treated with phlebotomy only. During the same period, the risk was significantly increased by exposure to P³², busulfan, or pipobroman. Appropriate cytoreduction with the goal to optimize the control of the blood cell counts is recommended in all patients with acute vascular events¹⁶.

USG after on June 30, 2008 (While on regular warfarin and propranolol therapy) showed persistent splenomegaly (about 17 cm) and persistence of periportal echogenic shadow and also collateral channel around porta hepatis. Maintenance of INR was maintained within the recommended level with some degree of difficulty because of the inherent bleeding tendency associated with Polycythaemia Patient also needed a high degree of motivation for continuing treatment.

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