

## **Steroid Induced Glaucoma in Patients of Spring Catarrh: A Retrospective Study**

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### **Summary**

*A retrospective study of 530 cases of spring catarrh using topical steroids revealed that 7% (48 patients) developed a rise in intra-ocular pressure to levels above the normal limit of 22 mm Hg. Spring catarrh in our part of the world is of a severe mixed variety causing intense symptoms of irritation and itching as well as severe corneal lesions often threatening the patient's sight. Most cases cannot be controlled by simple anti-allergic/decongestant therapy. Very often they require a potent topical steroid, instilled several times a day. However due to ignorance of the patient, prescription of steroids by quacks, easy and free availability of steroidal preparations on the market and poor and irregular follow-up, many patients continue to use such drugs without any check of intra-ocular pressure. Quite a number of these patients may end-up with steroid induced complications especially glaucoma. In this study 7% of patients either presented or came back with signs and symptoms of steroid induced glaucoma. The male to female ratio remained the same as for the spring catarrh (5:1). Glaucoma in many patients occurred after having used steroids for more than 15 years and resulted in marked visual loss. It is therefore stressed that steroid induced*

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*glaucoma, which is initially an entirely reversible condition, should be continuously looked for in all patients using steroids. Where possible, non-pressure elevating steroids should be used. If potent steroids must be used, the possible side effects should be explained to the patients and a regular and careful follow-up of pressure maintained.*

## **Introduction**

Steroids were first appreciated in 1920, synthesized in 1937 and thereafter made available for clinical use<sup>22</sup> in 1948. Since then a large number of synthetic steroids of varying potencies have been prepared and used in many life-and sight-saving situations. This almost brought a revolution in the field of Medicine. Steroids have since been used as potent anti-inflammatory, anti-allergic and immunosuppressive agents. But soon, a darker side to this picture emerged; steroids were found to cause several serious systemic as well as ocular complications. Prominent amongst the ocular complications were posterior sub-capsular cataracts, super-added bacterial, viral and fungal infections, corneal sloughing leading to perforation and glaucoma. This last mentioned complication is of special interest and importance as due to its silent onset, it often passes un-noticed by both patient and doctor, unless specifically sought for by regular and careful follow-up of intra-ocular pressure and visual fields. It is made all the more important by the fact that this conditions, in the early stages, is entirely reversible simply by complete withdrawal of the offending agent. However, if allowed to persist for a prolonged period of time, irreversible damage due to glaucomatous optic atrophy occurs.

## **Material and Methods**

Between 1982–1984 we conducted a study on 530 patients of spring catarrh who presented at the department of Ophthalmology, Khyber Hospital, Peshawar. All of them were either using steroids or were subsequently put on steroids. We checked pressure at the time of presentation and subsequently on follow-ups. 48 cases (7%) either presented to us with glaucoma following prolonged use of steroids or subsequently developed a rise in pressure. These patients were initially put on the medical management of chronic simple glaucoma with replacement of steroids by non-steroidal drugs for the symptomatic relief of spring catarrh e.g. anti-histaminics, decongestants etc.

Those patients in whom we were unable to control pressure by these means, underwent trabeculectomy as a last resort to preserve their vision. No conclusions regarding hereditary or other predisposing factors were made as most of these patients came from far flung areas and were unable to provide us with a scientifically accurate family history nor were we able to examine any of their relatives.

## Results

Out of 530 patients of spring catarrh using steroids, 48 (7%) developed a rise of intra-ocular pressure. The male to female ratio was the same for both spring catarrh and steroid induced glaucoma (5 males to every female). The majority of patients fell into the age-group 11-25 years (Table I). The duration of use of steroids varied from a few months to many years but the maximum presented within 15 years of the onset of treatment (Table II). The number of patients with a history of personal or familial atopy was 25% in patients of steroid induced glaucoma as compared to patients of spring catarrh as a whole in whom it was 35%.

TABLE I  
STEROID INDUCED GLAUCOMA  
AGE OF PATIENTS AT THE TIME OF PRESENTATION

S. No.	Age Group years	No. of Patients	Percentage
1.	0-5	—	—
2.	6-10	1	2.08
3.	11-15	13	27.09
4.	16-20	18	37.50
5.	21-25	10	20.83
6.	26-30	3	6.25
7.	31-35	—	—
8.	36-40	3	6.25
		Total:-	48
			100.00

TABLE II  
STEROID INDUCED GLAUCOMA  
DURATION OF USE OF STEROIDS AT PRESENTATION

S. No.	Duration in years	No. of Patients	Percentage
1.	0-5	14	29.17
2.	6-10	21	43.75
3.	11-15	9	18.75
4.	16-20	1	2.08
5.	Unknown.	3	6.25
		Total:-	48
			100.00

38.5% of patients presented with a visual acuity of less than 6/24. (Tables III and IV). At the time of presentation, steroids were stopped for all patients and the medical management of chronic simple glaucoma was instituted. The use of non-steroidal drugs for the symptomatic relief of spring catarrh e.g. anti-histaminics and decongestants etc. were started. On this regimen 75% of patients were controlled, however in 25% (12 patients) the pressure remained elevated and as a last resort trabeculectomy had to be performed to preserve whatever sight remained.

TABLE III  
STEROID INDUCED GLAUCOMA AND VISUAL ACUITY

No. of eyes with steroid induced glaucoma	=	96
No. of eyes with poor visual acuity	=	37
a. with poor visual acuity $\leq 6/60$	=	29
b. with poor visual acuity $> 6/60$ but $\leq 6/24$	=	8

TABLE IV  
CAUSES OF DECREASED VISION IN STEROID INDUCED GLAUCOMA

Causes	V.A. $\leq 6/60$ (No. of Eyes)	V.A. $> 6/60$ but $\leq 6/24$ (No. of Eyes)	Total (No. of Eyes)
Glaucomatous changes only	18	4	22
Glaucomatous changes with cataracts	11	4	15
Total:-	29	8	37

## Discussion

Since 1950, when a number of keen observers noted a rise in intra-ocular pressure in patients using steroids<sup>1,2</sup>, it has now been well-established that the use of steroids may cause a rise in intra-ocular pressure. It was after Francois description in 1954 of a patient of spring catarrh whose pressure fell to normal after cessation of steroid therapy<sup>3</sup> that the use of steroids could be exactly accounted for as the cause of raised intra-ocular pressure. This however did not go unchallenged since some observers did not find any significant rise in normal patients.<sup>4</sup>

Since then there have been numerous reports of steroid induced glaucoma in humans and experimental animals<sup>5, 6, 7, 8</sup>. It was noted that steroids affect the intra-ocular pressure more in glaucoma patients and their relatives<sup>9, 10</sup> and patients with congenital anomalies of the angle<sup>8</sup>. This is especially true for patients with open angle glaucoma<sup>9</sup>. A rise in intra-ocular pressure in patients with narrow angle glaucoma has also been reported<sup>10</sup>.

Cupping of the disc and defects in visual fields are directly related to the amount of rise in intra-ocular pressure and not to the duration of raised pressure<sup>11</sup>. They may occur with remarkable rapidity, sometimes even within one month of topical application<sup>12</sup> thus emphasizing the need for repeated examination and prompt action.

Steroid induced glaucoma is similar to chronic simple glaucoma in its mechanism of reduced facility of outflow and damage to the optic nerve. It appears to be genetically determined but its exact pathophysiology is still not clear. The magnitude of pressure elevation depends on the potency, concentration, frequency of applications and length of time of administration of steroids. A common feature of this condition is that it is generally reversible, for after cessation of steroids the pressure tends to fall sometimes rapidly and sometimes after one or two months<sup>13, 14, 15</sup>. In our study however we had a different experience as in 25% of cases the pressure did not fall to normal, months after cessation of steroids and the use of pressure decreasing therapy. In these patients further deterioration forced us to resort to trabeculectomy as a sight saving procedure. If the rise of pressure is allowed to persist, irreversible damage is caused and as we have seen, it may even lead to complete blindness.

Most of these patients were using steroids for several years, the majority presented within 15 years of onset of therapy. However 6 patients reported after 15 years which shows that the Ophthalmologist prescribing steroids must be vigilant all the time and all such patients must have frequent and regular follow-up of intra-ocular pressure as long as they are using steroids. This is not easy in a country like ours where great distances may separate the patient and his Ophthalmologist and therefore, following symptomatic relief, most patients may not report for follow-up until quite late. The intense irritation, caused by spring catarrh and relieved by steroids, usually prompts the patients to continue the use of steroids even after the Ophthalmologist has ceased to prescribe them. This is caused by the free availability of these drugs on the market enabling anyone to buy them. This is also true of those who have very easy access to steroids like doctors, nurses, pharmacists and paramedical staff. Such patients need special care. Special care is also needed in glaucoma patients both chronic simple and angle closure glaucoma, glaucoma suspects and their relatives, high myopes, diabetics and patients with Krukenburg spindles<sup>16</sup>.

In view of the prevailing situation in the country, patients of allergic and inflammatory ocular diseases should be given a trial of non-steroidal preparations like sodium cromoglycate<sup>17</sup>, aspirin<sup>18</sup>, anti-histaminics and decongestants. If they still require steroids then the new synthetic preparations like Medrysone and Fluorometholone,<sup>19, 20</sup> claimed to have minimal pressure elevating effects should be prescribed; care must be exercised as some patients may show a significant rise even with these drugs. If such therapy fails to control the condition, only then may potent steroids be prescribed but under strict supervision of an Ophthalmologist with careful and regular follow-ups. To improve the follow-up, patients should be explained all the possible side effects of steroids and encouraged to seek advice as promptly as possible.

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