Correspondence

Incidence of optic disc haemorrhages in chronic simple glaucoma and ocular hypertension

Sir, How often do optic disc haemorrhages help us estimate the visual prognosis for patients with elevated intraocular pressure? If optic disc haemorrhages truly do precede retinal nerve fibre layer defects and do signal increased risk of progression of visual field loss,1 we as ophthalmologists need to know just how often we can expect to find this sign in our patients. In his article2 Professor John Gloster is to be congratulated for the presentation of data from such a large number of photographs. There are, however, several serious problems with the article’s methodology that limit application of its conclusions to all patients with elevated intraocular pressure.

Professor Gloster looked for optic disc haemorrhages in 1829 photographs of optic discs taken of 617 eyes of 320 patients with glaucomatous field defects and cupping, and of 284 eyes of 169 patients with at least one IOP measurement greater than 21 mmHg and who had no field defects, and he concluded that: (1) at least one-third of eyes (not patients, as the abstract states) affected by glaucoma show disc haemorrhages at some time or another; (2) 17% of glaucoma patients had disc haemorrhages; (3) there is a rising incidence of haemorrhage as the vertical cup:disc ratio increases; (4) there is a lower incidence of haemorrhages in ocular hypertension than in chronic simple glaucoma; and (5) more haemorrhages were found in eyes with lower IOPs than with higher IOPs.

As is common in such studies Professor Gloster’s data are retrospective and not population-based, which, by his own admission, limits the accuracy and generalisability of his conclusions. He also did not have a control group. He simply looked at some of the disc photos of some of the people in the general ophthalmological population who had an intraocular pressure greater than 21 mmHg. It is not surprising that, as the number of photographs per patient increased, the incidence of disc haemorrhages per patient increased, because the decision to order disc photographs is inherently biased toward selecting those patients with positive or at least questionable findings by ophthalmoscopy. Professor Gloster states only that photographs were taken ‘of a substantial proportion of the patients seen in the Glaucoma Unit,’ a referral centre. He excluded an unspecified number of photos in which he was not sure that he could rule out small disc haemorrhages, again introducing a selection bias. Haemorrhages may be easier to rule in than rule out. Were the patients whose poor-quality photos were excluded different somehow from the rest of the glaucomatous population? One might hypothesise that, for example, chronic pilocarpine use might sometimes have inhibited pupilary dilatation and clear photography.

Neither the number of photographs taken of the patients’ discs per examination, the interval between examinations, nor the number of follow-up visits per patient were standardised or controlled for. Moreover, the patients apparently were on various therapeutic regimens. It is quite possible that patients with poorly controlled IOPs, progressive field loss, previous haemorrhages, or greater cupping would have more frequent check-ups and more photographs taken. The total number of eyes with disc haemorrhage divided by the number of eyes photographed is 60/617, or 9.7%. Unless Professor Gloster is drawing his conclusions from the 10 eyes that were photographed 7 times each, how then can he make the statement, ‘it is probable that at least one-third of affected eyes showed [optic disc haemorrhages] at some time or another’? As for relating maximum intraocular pressure recorded to the incidence of optic disc haemorrhage, in which he concluded that ‘the incidence of haemorrhage was greater in eyes with lower intraocular pressures than in eyes with higher pressures,’ he did not study the IOPs recorded at the time the photography was done but rather before therapy was begun, ‘in some instances . . . several years before the photograph was taken.’ It is well known that glaucoma patients can present at any stage in their disease, and that initial pressure does not correlate with field loss at 5-year follow-up. Patients vary considerably in their response to glaucoma therapy, which in Professor Gloster’s article is not even specified, raising the question whether a lower incidence of haemorrhages is related to higher IOP or to some other factor correlated with initial (presenting) IOP.

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References

Sir, Mr David P. Sutton has raised several points about the data presented and the conclusions drawn therefrom in my paper on ‘Incidence of optic disc haemorrhages in chronic simple glaucoma and ocular hypertension.’ He recognises that I made it clear that the study was retrospective and was not population-based. The limitations of such studies are well known, and include the possibility that unintended processes of selection make the results and conclusions unrepresentative of the clinical condition which we think we are studying. Mr Sutton cites several possibilities. He refers to an unspecified number of photographs which were excluded because I was not sure that I could ‘rule out disc haemorrhages,’ although the paper actually mentions the number of photographs in which a reasonably confident opinion could be given ‘as to the presence or absence of a disc haemorrhage.’ In other words, when a photograph was discarded it was because I had failed to make up my mind not only about the possible presence but also about the possible absence of a haemorrhage, and it is to be hoped that I succeeded in my attempt to give equal consideration to these 2 possibilities. Mr Sutton suggests that long-
continued use of pilocarpine might have inhibited pupillary dilatation and clear photography, and this of course did sometimes happen. In some cases the problem was overcome by cessation of pilocarpine for a longer period before a subsequent attempt at photography, and I gained no impression of a grossly different photographic success rate in patients on miotics. On the other hand it is possible to argue that patients on nonmiotic therapy are more likely to be those who have lens opacities, and consequently they will be the ones less likely to give clear photographs. Some doubt must remain on this sort of problem, but whether such uncertainties can be completely avoided is another matter; for example, glaucoma patients with more than a certain degree of cataract will always be excluded from this sort of study, so some selection is inevitable. Mr Sutton is quite right in saying that such factors as the interval between photographs and the number of visits were not standardised. This was the point of my comment on p. 455 that 'photography at regular intervals' should be aimed for in any future study.

With regard to the observed overall incidence of disc haemorrhages in glaucoma, it is to be expected that this will rise as the number of observations increases. Unfortunately, as was pointed out, the data were insufficient to allow accurate extrapolation to determine the incidence which could be expected with a very large number of observations on each patient. Nevertheless, consideration of the results of Table 1, for example by inspection of a simple graph, suggest that an overall incidence of around 30% is a reasonable minimum. It could easily be more. In this connection it is interesting that, in the study by Bengtsson et al., to which Mr Sutton makes reference, somewhat similar results were presented as a graph of frequency versus number of observations, showing that 2/3 of glaucoma patients presented a disc haemorrhage within 4 visits, and it was stated that the hypothesis of disc haemorrhages occurring in all cases could not be rejected, although it was not proved. This effect of frequency of observation upon apparent incidence clearly has to be borne in mind when comparing groups of patients, and an attempt was made to do this. In Table 3, which related the incidence of haemorrhages to the cup:disc ratio, the average number of times eyes were photographed at various sizes of cup:disc ratio was given. This was also so for the relationship with level of intraocular pressure (Table 5). Mr Sutton says that 'I did not study the IOPs recorded at the time photography was done.' I did not really think that there was much point in doing this because of the varying preparation of patients for photography. Those on miotic therapy usually had their drops stopped, some for longer than others, those on nonmiotic therapy continued their treatment, some had acetazolamide for a day or two before photography, these variations being partly dependent upon previous response of the IOP to temporary cessation of treatment. In any case the level of IOP at photography probably bore no relationship to that at the time the haemorrhage occurred. The maximum IOP which had been recorded for each patient was chosen as the one by which cases could be divided into groups, because this method resembled the traditional division into 'low tension glaucoma' and 'chronic simple glaucoma,' but avoided the arbitrary choice of a dividing pressure level.

It does seem that the occurrence of disc haemorrhages in chronic simple glaucoma is worthy of more study. In particular one would like to know if haemorrhages really do occur at some time or another in all patients, whether they are part of the process leading to glaucomatous disc damage or merely one of the results thereof, and just what prognostic importance they have and so on. I hope that my paper indicated some of the advantages and disadvantages of basing investigations on photography, and I believe that it gave an idea of the scale of the investigation needed to get more accurate information than that which is available to us so far.

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Reference

Herpes zoster ophthalmicus

Sir, In a recent article by Dr S. Lightman et al. it was stated that as a result of a retrospective analysis of 1000 cases of herpes zoster ophthalmicus the great majority of the patients were 'healthy and therefore do not have diminished immunity.' The authors base these claims on the strength of a full blood count, differential white cell count and film, liver function tests, and electrophoresis, blood sugar, and chest x-ray. No attempt was made to assess quantitative immunoglobulin levels, and T cell function was not assessed.

As the paper is entitled a 'Medical review' of herpes zoster ophthalmicus, why was an account of family history of compromised immunity or the patient's past immunisation history not documented? A physical examination was not mentioned.

This paper highlights the danger of reaching conclusions based on retrospective analysis.

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Reference

Sir, Active viral infection is associated with an alteration of T cell function, and assessment of T cell function in our patients on presentation with active herpes zoster would give no indication of the patient's basal immune status. The follow-up of our patients was for an average of 2 years, during which time no further infective episodes occurred, and therefore there was no medical indication for expensive immunological investigations. Family histories were taken from all patients and revealed no evidence of compromised immunity.

Since the most common age range of our patients was 50–70 years, immunisation histories are likely to be inaccurate, and most patients had difficulty recalling whether or not they had chicken-pox.