Intraocular Pressure and Hemodialysis

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Dear Sir,

We were very interested in the article by Gafter et al. [1], whose study was designed to evaluate intraocular pressure (IOP) changes during 4-hour hemodialysis sessions in uremic patients receiving chronic hemodialysis treatment. These authors reported that their patients had a low IOP and hemodialysis did not increase it significantly. Thus they concluded that the risk of severe IOP rise following hemodialysis is only a remote possibility. We report here our experience in a group of 34 patients with end-stage renal failure enrolled in a chronic hemodialysis program. Dialysis took place 3 times weekly for 4–5 h. IOP readings were taken before, during (approximately 2 h after start) and within 30 min after dialysis using a Schiötz tonometer. The average IOP in uremic patients before dialysis was 14.9 ± 1.4 mm Hg and its average increase was 1.2 mm Hg during dialysis and 0.6 mm Hg after the end of the session. These data confirm, at least in part, the findings of Gafter et al. [1]. Indeed, in our patients IOP both before and after hemodialysis was less than the mean IOP observed in control subjects (16.5 mm Hg). However, the analysis of the IOP changes during the dialysis session led us to identify three groups of patients on the basis of the different tonometric behavior. Group 1 consisted of 25 patients in which IOP did not change significantly. Group 2 consisted of 6 patients (18%) in which there was a significant increase of IOP (13.5 ± 0.3 mm Hg before, 23.2 ± 1 mm Hg 2 h after start, 18.1 ± 2 mm Hg after dialysis). Group 3 consisted of 3 patients in which IOP significantly decreased during hemodialysis. During the dialysis session group 2 patients often developed complications such as headache, nausea and vomiting and in some cases these symptoms were observed in association with the rise in IOP. Goni-oscropy revealed in these patients a narrow angle between pupil and lateral cornea. In contrast, this angle appeared normal in patients of group 1 and 3. The mechanism leading to the rise of IOP during hemodialysis is still controversial. It has been reported that during dialysis there is an increase in aqueous humor formation [2]. Our findings suggest that this does not induce a significant change in IOP in patients with normal outflow capacity. However, if there exists an outflow obstruction the rise becomes significant. In group 2 patients, the administration of acetazolamide (500 mg/day orally) resulted in preventing intradialytic increase of IOP or associated symptoms, particularly headache. Nevertheless, acetazolamide caused in all patients a metabolic acidosis which induced us to interrupt the therapy after approximately 6–8 days.

In conclusion our findings suggest some considerations: (1) a significant rise of IOP during hemodialysis is not a remote possibility but is, in our experience, relatively frequent (18% of patients). (2) The state of IOP should be considered when planning long-term hemodialysis. (3) The possibility of an obstruction of aqueous humor outflow should be kept in mind in patients showing a significant increase of IOP during dialysis. (4) Acetazolamide therapy must be used cautiously in these patients since it may produce a severe metabolic acidosis as we have recently reported [2, 4]. (5) Uremics with excessive intradialytic increase of IOP may have to be protected by the administration of drugs, other than acetazolamide, such as pilocarpine or mannitol.

References

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