AIM

SCOPE
Aim and Scope

Improvement in the medical management of diabetes mellitus during the last 50 years has increased the life expectancy of a large number of diabetic patients. This increase has resulted in a significant rise in diabetic complications, including diabetic cataract and diabetic retinopathy. These complications are leading causes of visual dysfunction and blindness in patients with diabetes mellitus. About 10-15% of patients undergoing cataract surgery are diabetic, and this number is increasing (Gilvarri, Eustace, 1982).

Diabetic cataract is the rapidly progressive one that is found in younger persons (under 40 years of age). There is no relationship between the duration and severity of diabetes and the onset and progress of the cataractous changes. However as suggested by Vogt (1967) it could be provoked prematurely by diabetes. It is usually bilateral when the diabetes is not well controlled, changes in blood glucose levels causes changes in refractive power by as much as 3 to 4 diopters. This results in blurred vision. Such changes do not occur when the disease is well controlled (Vaughan et al., 1994).

Despite extensive research, the mechanism responsible for diabetic cataract formation is still unknown. Although modern cataract extraction and IOL (Intra ocular lens) implantation have become a routine procedure with a low complication rate in senile cataract, the decision to remove the cataract in patients with diabetes involves many considerations. Cataract extraction in diabetic patients may lead to several complications during surgery as well as during the postoperative period. Proliferative retinopathy may develop rapidly after cataract surgery despite relatively short duration
and acceptable metabolic control of diabetes. That’s why the researchers should work
towards developing new strategies of rational drug design to prevent or delay cataract.

Many studies of human cataractous lenses demonstrate that
cataractogenesis is a multifactorial process and in most cases oxidative stress is thought
to play a major role in cataract formation.

The increasing global incidence of diabetic cataract emphasis the need to
understand the various biochemical alterations and mechanism involved in the
pathogenesis of diabetic cataract.

Oxidative stress is thought to play a major role in cataract formation,
Oxidative stress either as the primary event or secondary to risk factors like aging and
smoking, is one of the predominant factors that lead to cataract. A major mode of
damage to lens proteins involves oxidative reactions (Balasubramanian, 2002; Ziegler
et al., 1985; Young, 1991). For this reason, the possible role of antioxidants in delaying
the onset or progression of age-related cataract has gained considerable interest.
Endogenous defense mechanism which protect the lens against oxidative damage include
compounds like glutathione, ascorbate and antioxidant enzymes like catalase, superoxide
dismutase, glutathione reductase, glutathione peroxidase (Varma et al., 1984; Zigman et
al., 1979; Mc Cay, 1985). But with increasing age, the levels of those protective enzymes
are known to decline in the human eye (Garland et al., 1988; Berman, 1991).
Supplementation with antioxidants thus appears to be an attractive possibility to delay the
onset of age-related cataract. It has been estimated that a delay in cataract formation of
approximately 10 years would reduce the cataract surgical burden by perhaps 45%
(Kupfer, 1984)
In view of these observations, the purpose of this study was to determine the activities of antioxidants such as GSH, ascorbic acid, protein oxidation, calcium and antioxidant enzymes such as GPx, GR, GST, AR, SD in diabetic and nondiabetic cataractous lenses and to designate the role of these enzymes in the development of diabetic cataract.