

## INTRODUCTION

Keratoconus (KC) is a non inflammatory, progressive, degenerative disorder characterized by thinning & steepening of the cornea & is usually bilateral (*Agrawal, 2009*).

Its incidence in the general population is reported to be about one in 2000 (*Rabinowitz, 1998*). However, Incidences of one in 600 to one in 420 seem to be more logic, regarding the current diagnostic facilities (*Caporossi et al., 2006*).

Two chief mechanisms for the development of KC have been put forward. One purposed that ectasia is closely associated with tissue degradation (*Kenney et al., 2000*), whereas the other suggested that it's due to slippage between collagen fibrils (*Meek et al., 2005*) & Weakened of the interfibrillar glue, then lamella or (collagen bundles) would have the potential to tear apart (*Agrawal, 2009*).

The central & inferior regions of the cornea are likely to be affected preferentially (the main region of cone formation), since interlamellar cohesive strength is at a minimum in that area in normal corneas (*Smolek and Beekhuis, 1997*).

The main clue to an early diagnosis is the corneal topography, which is most useful, highly sensitive & specific for diagnosis of KC (*Agrawal et al., 2005*).

Nowadays oculus pentacam provide an intelligent system for detection & assure the presence or absence of the disease by collection of a variety of data including slit scanning, pachymetry, front & back elevation maps using the invaluable benefits of the Holladay report (*Holladay, 2008*).

The treatment of KC must be tailored according to the condition of the case. Several factors may influence the management of each case, including degree of KC, presence or absence of corneal opacities, corneal thickness, tolerability of contact lenses & Integrity of corneal endothelium. Rigid contact lense represent the treatment of choice in most cases of early KC (*Rabinowitz, 1998*).

Intrastromal corneal ring-segments have been used to reshape keratoconic corneas to improve V/A & to delay or prevent the need for keratoplasty (*Ertan and Colin, 2007*).

So far there hasn't been any successful way to stop the progression of KC. A new non surgical, non-invasive treatment, based on collagen cross linking with ultraviolet-A (UVA, 370 nm) & riboflavin (Vitamine B<sub>2</sub>) a photosensitizing agent is now available (*Coskunseven et al., 2009*).

The technique of corneal collagen cross-linking has been used to at least block progression of KC in the progressive phase (*Wollensak et al., 2003*). Corsslinking arrest the furthur progression of corneal collagen thinning & increase the biomechanical stability of the cornea (*Wollensak, 2006*).

The combination of riboflavin & UV-A radation induces a photopolymerization that increase the rigidity of corneal collagen & its resistance to keratectasia through the formation of new interfibrillar covalent bonds of corneal collagen (*Spoerl et al., 2007*).

Corneal collagen cross-linking treatment is not a cure for (KC) rather, it aims to slow or even halt the progression of the condition. Patient may need to continues to wear spectacles or contact lenses following the crosslinking treatment but it's hoped that it could limit

further deterioration in V/A& reduce the need for keratoplasty (*Vinciguera et al., 2009*).

Corneal cross-linking treatment can be combined with Intacs to flatten the KC cone even more than with Intacs alone. In these cases, corneal cross linking treatment stabilize KC from getting worse as well as help the Intacs reverse the KC steeping that had already occurred (*Wollensak, 2006*).