Ocular pathology review: A comprehensive text and review.

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Introduction

Epidermal growth factor, transforming growth factor-β, keratinocyte growth factor, hepatocyte growth factor, fibroblast growth factor and platelet-derived growth factor have been detected in the anterior segment of the eye. On binding to cellular receptors, these factors activate signalling cascades, which regulate functions including mitosis, differentiation, motility and apoptosis [1]. Production of growth factors by corneal cells and their presence in the tear fluid and aqueous humour is essential for the maintenance and renewal of normal tissue in the anterior eye and the prevention of undesirable immune or angiogenic reactions. Growth factors also play a vital role in corneal wound healing, mediating the proliferation of epithelial and stromal tissue and affecting the remodelling of the Extra Cellular Matrix (ECM).

Ocular findings in subjects with Congenital Heart Disease (CHD). The commonest anatomic cardiac anomalies were ventricular or atrial septal defects, tetralogy of Fallot, pulmonary stenosis, and transposition of the great arteries [2]. The heart lesions were divided physiologically into volume overload, cyanotic, and obstructive. In all, 105 syndromic subjects included velocardiofacial syndrome, Down's syndrome, Charge association, DiGeorge syndrome, Williams's syndrome, Edwards's syndrome, Noonan syndrome, Vacterl association, and Patau syndrome (trisomy 13). The paediatric team recognized 51 patients as syndromic. Two independent geneticists recognized additional 54 patients as syndromic. Positive eye findings were present in 55% and included retinal vascular tortuosity, optic disc hypoplasia, trichomegaly, congenital ptosis, strabismus, retinal haemorrhages, prominent eyes, and congenital cataract. There was a strong correlation between the retinal vascular tortuosity and both a low haematocrit (P=0.000) and a low arterial oxygen saturation (P=0.002) [3].

Immunoblot analysis of protein derived from white blood cells of a living brother, also affected with choroideremia, confirmed the absence of Rab escort protein-1, the normal CHM gene product. Direct sequencing of the coding region and adjacent splice sites of the CHM gene was undertaken on genomic DNA from the living brother and revealed a transition mutation, C to T, in exon 6 (R253X) which resulted in a stop codon and was predicted to truncate the protein product. Histopathological examination of the eye of the deceased brother showed relative independent degeneration of choriocapillaris, retinal pigment epithelium, and retina, similar to observations in the mouse model of choroideremia. In addition, mild T-lymphocytic infiltration was found within the choroid. The ophthalmic features and the pathology of choroideremia are discussed in light of new findings in the current case [4].

The 13-15 trisomy syndrome (also called D-trisomy and Patau syndrome1) comprises an entity in which an extra chromosome is associated with the 13-15 or D group of paired chromosomes. Its clinical features consist of multiple congenital abnormalities that commonly include ocular defects. The most frequent systemic abnormalities are harelip, cleft palate, umbilical hernia, polydactyly, cardiac defects, and malformations of the central nervous system. Microphthalmia, iris colobomas, cataracts, and retinal dysplasia have been especially noted in recent reviews of the subject [5].

Conclusion

Complement systems and regulatory proteins are present in the human and rodent eye, which contribute to various ocular diseases such as keratitis, uveitis, and age-related macular degeneration. Growth factors, such as epidermal growth factor, transforming growth factor-β, keratinocyte growth factor, hepatocyte growth factor, fibroblast growth factor, and platelet-derived growth factor, are detected in the anterior segment of the eye. These factors regulate functions such as mitosis, differentiation, motility, and apoptosis. Growth factors are essential for maintaining normal tissue in the anterior eye and preventing immune or angiogenic reactions.

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