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Prothrombin time in retinitis pigmentosa

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Abstract

The prothrombin time was recorded for 87 primary retinitis pigmentosa (RP) patients belonging to three different clinical categories. All categories showed prothrombin time higher than normal. There was no correlation between the age of onset and the prothrombin time, nor between duration of disease and the prothrombin time. The high prothrombin time in patients with RP suggests that further study of prothrombin time and related factors may help in better understanding of the pathogenesis of RP.

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Full Text

During the studies on retinitis pigmentosa (RP) a consistent observation had been that the blood of these patients always took a longer time to clot. Hence the prothrombin time, the time required for the clotting to take place in oxalated plasma to which optimum amounts of thromboplastin and Ca2+ have been added,[1] was evaluated for 87 patients of RP.

Materials and Methods

After a thorough clinical evaluation,[2] 87 primary RP patients belonging to three different clinical categories, namely, early onset - onset of the disease at or before the age of 15, late onset - onset between 16 to 40 years, and senile onset - onset of the disease after the age of 40,[3] were choosen for this study. Age of onset was ascertained by recording the history of the patients based on an elaborate questionnaire.[2]

Prothrombin time was determined according to Varley's method.[1] Along with every patient, the prothrombin time for an age-matched control was invariably recorded to facilitate comparison with healthy individuals.

Results

It was found that 86 out of the 87 RP patients had prothrombin time >20 sec (Table). The reported normal prothrombin time value is 18-22 sec.[1] The prothrombin time was significantly higher in the early and late onset RP patients than in the age-matched controls ($p<0.001$, t-test). No conclusion could be drawn for the sensile onset category because of the small number of observations. There was no correlation between the age of onset and prothrombin time ($r = -0.007$ for early onset, $r = -0.18$ for late onset). Similarly, there was no correlation between the duration of RP and prothrombin time ($r = -0.19$ for early onset, $r = -0.17$ for late onset).

Discussion

RP has been described as a group of inherited disorders of the retina that are characterized by progressive dysfunction involving the photoreceptors and often subsequently other cell layers associated with progressive cell loss and eventually atrophy of several retinal layers.[4]

Ultrastructural study of the RP retinae have shown degenerative lesions in addition to widespread photoreceptor loss, severe membranous distortion and vesiculation along with formation of autophagocytic vacuoles.[5] Also, the events which take place in RP retina have been shown to be akin to apoptosis, programmed cell death, wherein endonuclease induced fragmentation of the genomic DNA takes place followed by nuclear disintegration and cellular fragmentation into a cluster of membrane-bound apoptotic bodies.[5]

This study indicates that RP patients irrespective of the sex, age of onset or the progress of the disease (duration) show higher prothrombin time. The prothrombin time is inversely proportional to the concentrations of prothrombin and of blood clotting factors V, VI, and X. Liver disorders, Ca^{2+}, and vitamin K also affect the clotting time.[1]

Increased prothrombin time suggests altered level(s) of one or more of these factors. Prothrombin and hence thrombin could be one of them. Thrombin is known to be mitogenic; it stimulates DNA synthesis in cultured retinal pigment epithelial (RPE) cells. It may act as an endocrine mediator of RPE cell proliferative activity and it may participate in normal or exaggerated ocular wound healing.[6] It is also known that X-linked RP cosegregates with haemophilia A.[7] However, these patients have not been reported to have increased tendency for bleeding diathesis. Hence this increase in prothrombin time may not be clinically significant. Nevertheless, the possibility of some relation between prothrombin time and the spontaneous degeneration of photoreceptors in the RP retinae cannot be ruled out. Hence, a thorough study not only of thrombin but also of other clotting factors which are known to affect the prothrombin time becomes necessary in order to get an insight into the mechanism of the pathogenesis of RP.

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