1600, and 2400. Student's *t* test was used to compare growth hormone concentrations before and during treatment.

The table shows mean circulating concentrations of growth hormone before and on the third day of treatment. Concentrations ranged from 32.5 to 234 mIU/l before treatment but were suppressed by 30-79% by the third day of treatment ($p < 0.001$). The maximal reduction in growth hormone concentration was achieved two to four hours after administration of the drug and was 50-95% of the basal concentration. After administration of the drug the concentration fell within 30 minutes to 30-83% of the basal concentration in all patients and remained suppressed for up to eight hours.

The patient with an intractable headache experienced relief for periods of two to three hours during the three days of treatment, which reduced his growth hormone concentration by 46%. Placebo was substituted on the fourth day and his headache promptly returned. He was then prescribed long term treatment with selective mini somatostatin in doses up to 100 µg every three hours. After seven months' continuous treatment his symptomatic relief was maintained and his growth hormone concentration was 34% of the concentration before treatment. A further patient (case 4) was treated for five months with selective mini somatostatin 100 µg twice daily. His condition improved, and his mean growth hormone concentration remained suppressed by 80%. Neither patient showed glucose intolerance during treatment or reported any difficulty with self injection or side effects even with the higher dose.

Comment

A clear relation exists between active acromegaly and increased morbidity and mortality.³ We believe that every effort should be made to treat such patients adequately. Complete surgical resection is often impossible, and pharmacological agents (for example, bromocriptine) if effective do not usually stop the secretion of growth hormone, so that concentrations remain raised. Radiotherapy takes a long time to achieve its full effect.⁴ Treatment with selective mini somatostatin seems likely to be a useful, fast acting adjunct in patients whose acromegaly is not rendered inactive by other treatment, because the underlying cause of acromegaly has been postulated to be either excessive growth hormone releasing factor or deficiency in somatostatin.⁵ All patients in our study, irrespective of the dose of selective mini somatostatin given, showed significant suppression of growth hormone concentrations, and two patients followed up for five and seven months were satisfactorily maintained on the drug. Long term trials are required to evaluate this potentially important new treatment.

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Intermediate filaments in the retina and retinoblastoma

Intermediate filaments are fibrillary organelles that are present in almost all eukaryotic cells. There are five different classes of these filaments—namely, keratins in epithelial cells, vimentin in mesenchymal cells, desmin in muscle cells, glial fibrillary acidic protein in astrocytes, and neurofilaments in most types of neurones. When a cell undergoes malignant change it usually retains its intermediate filaments, which can then be used as tissue type specific markers to diagnose and classify malignant neoplasms.¹

The origin of retinoblastoma remains unclear. A neuronal or neuroectodermal origin is presently favoured on the basis of morphological, biochemical, and immunological observations. The type of retinal cell from which the tumour develops, however, is unknown. We studied the types of intermediate filament present in the retina and retinoblastoma in man to try to clarify the histogenesis of retinoblastoma.

Methods and results

We examined 55 specimens of retinoblastoma fixed in formalin and embedded in paraffin, which represented all common histopathological types of the tumour. We used rabbit antiserum against vimentin and monoclonal antibodies to glial fibrillary acidic protein and to the 200 kilodalton neurofilament triplet protein, which were prepared in our laboratory and well characterised. A sensitive immunoperoxidase method of staining was used that makes use of the strong interaction between avidin and reagents coupled to biotin.

Müller's cells in normal retina stained positively for vimentin and often for glial fibrillary acidic protein. These two types of filament were also constantly seen in astrocytes in the inner retinal layers. Processes of ganglion cells in the nerve fibre and inner plexiform layers were stained by the monoclonal antibodies to neurofilaments. Surprisingly, we did not see any reactivity against the intermediate filaments studied in the horizontal cells, which contain vimentin and the three neurofilament triplet proteins in other species.² To verify this finding frozen sections of an eyeball enucleated because of retinoblastoma were studied by indirect immunofluorescence microscopy. The results were identical even though a rabbit antiserum that detected all three neurofilament triplet proteins was used in this experiment.

We did not detect any of the types of intermediate filament studied in retinoblastoma specimens. Vimentin and glial fibrillary acidic protein were seen, however, on many stromal elements within these tumours. In particular, cell processes that stained for neurofilaments could always be traced to infiltrated parts of the retina. A negative reaction was seen in the frozen sections studied by indirect immunofluorescence microscopy.

Comment

The human retina has previously been studied only for the presence of glial fibrillary acidic protein, and results have been inconsistent. The retinal complement of intermediate filaments in seven other species has been studied in detail,² and our results, with minor variations, agreed with these findings. The main difference was that, whereas vimentin and the three neurofilament triplet proteins are found in the horizontal cells in other species,² we could not detect any of the three types of intermediate filament studied in the horizontal cells of man. This emphasises the great interspecies variation in the intermediate filaments contained in horizontal cells.

As in previous studies^{3,4} glial fibrillary acidic protein was not detected in retinoblastoma cells, and these cells were also devoid of the other types of intermediate filament studied. Thus the cells from which retinoblastomas develop may be photoreceptors, bipolar cells, amacrine cells, or horizontal cells, all of which can occur in forms that do not contain intermediate filaments.² Recently, retinoblastoma cells grown in tissue culture were reported to contain glial fibrillary acidic protein.⁵ This observation indicates a need for further research.

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