

Age-related macular degeneration and radiotherapy

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Opinion Article

In India, the leading cause of legal blindness is age-related macular degeneration (AMD). Just 32% of the time is the laser essential. These types of retrofoveal neovessels appear to benefit from radiotherapy at doses on the order of 15Gy, which appears to delay development in approximately 40% of cases. A randomized trial comparing radiotherapeutics and placebos is required to validate these promising findings.

Because of its antiangiogenic, antifibrotic, and anti-inflammatory effects, radiation therapy has been extensively studied as a treatment for choroidal neovascularization (CNV) caused by age-related macular degeneration (AMD). Radiation for AMD has traditionally been administered in two ways: (1) external beam radiotherapy (megavoltage x-rays, proton beam) and (2) brachytherapy with radioactive isotopes (strontium-90, palladium-103).

External beam radiation has yielded conflicting results in clinical trials in neovascular AMD, leaving retina specialists undecided on whether it is a viable treatment choice. External beam radiation is limited in its ability to target only affected cells without causing collateral damage to healthy tissue because it is not localised. Low-dose beta radiation has been shown to suppress angiogenesis.

Radiation's effects on neovascularization are normally delayed, often for months. The choroidal neovascular membrane will continue to expand and cause retinal damage during this period, resulting in a loss of visual acuity until the radiation effect kicks in. Radiation for wet AMD is unsuccessful as a monotherapy in the majority of patients due to its time-of-onset restriction.

What further scientific advancement can be expected now that the genetic basis for a large proportion of AMD cases has been elucidated, and what importance can these new results have for the diagnosis and treatment of AMD? In the near future, a better understanding of the genetic basis of AMD could emerge quickly. It would not come as a surprise, for example, if genetic polymorphism is discovered.

If genetic polymorphisms in additional complement elements, complement regulators, and possibly immune system effectors and inflammatory mediators are involved in AMD, it will not be shocking.

It would be possible to develop genetic screening tests based on this new genetic knowledge to determine those people who are most at risk of developing AMD later in life. Clinicians may be able to track vulnerable patients from an early age, as well as establish and evaluate potential prevention therapies in the early stages of the disease.

Conclusion

Currently, neither therapy is approved by the US Food and Drug Administration for use in humans in the United States. Results of large clinical trials, as well as outcomes following approval, will be important to determine what long-term effects may exist with these novel radiation delivery systems for patients with AMD.

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