

ABSTRAK

Pendahuluan

Glaukoma merupakan penyebab kebutaan kedua terbanyak di dunia setelah katarak, ditandai dengan degenerasi sel ganglion retina (*Retinal Ganglion Cell*) melalui mekanisme apoptosis. Upaya terapi saat ini berfokus pada penurunan tekanan intraokular, namun belum sepenuhnya mencegah progresivitas kerusakan saraf optik. Penelitian terkini menyoroti peran faktor neurotropik seperti *Brain-Derived Neurotrophic Factor* (BDNF) dan protein apoptosis seperti *caspase-3* dalam patogenesis glaukoma. Epigallocatechin-3 Gallate (EGCG), flavonoid utama teh hijau, memiliki sifat antioksidan, anti-apoptosis, dan neuroprotektif.

Tujuan

Membuktikan pengaruh suplementasi EGCG oral terhadap ekspresi BDNF dan *caspase-3* pada tikus Wistar model glaukoma yang diinduksi dengan injeksi N-Methyl-D-Aspartate (NMDA) intravitreal.

Metode

Penelitian merupakan uji eksperimental *post-test only randomized controlled group* dengan 14 ekor tikus Wistar model glaukoma (jantan, usia 6–8 minggu, berat 150–300 g), dibagi menjadi dua kelompok: perlakuan (EGCG oral 50 mg/kgBB/hari) dan kontrol (plasebo aquadest), diamati selama 14 hari. Penilaian ekspresi BDNF dan *caspase-3* pada lapisan RGC dilakukan dengan pemeriksaan *real-time Polymerase Chain Reaction*

Hasil

Ekspresi BDNF kelompok perlakuan ($14,36 \pm 2,15$) lebih tinggi daripada kelompok kontrol ($8,32 \pm 3,06$) dengan perbedaan signifikan ($p=0.001$). Ekspresi *Caspase-3* kelompok perlakuan ($8,12 \pm 1,96$) lebih rendah daripada kelompok kontrol ($18,23 \pm 4,99$) dengan perbedaan signifikan ($p=0.002$). Analisis korelasi menunjukkan hubungan negatif antara ekspresi BDNF dan *caspase-3* yang tidak bermakna pada kelompok perlakuan ($p=0.056$).

Kesimpulan

Suplementasi EGCG oral berpotensi memberikan efek neuroprotektif dan antiapoptosis pada glaukoma dengan meningkatkan ekspresi BDNF sekaligus menurunkan ekspresi *caspase-3*.

Kata kunci

BDNF, *Caspase-3*, EGCG, glaukoma, sel ganglion retina, RGC

ABSTRACT

Background

Glaucoma is the second leading cause of blindness worldwide after cataracts, characterized by retinal ganglion cell degeneration through apoptosis. Current therapeutic approaches mainly focus on lowering intraocular pressure but have not fully prevented the progressive damage of the optic nerve. Recent studies highlight the role of neurotrophic factors such as Brain-Derived Neurotrophic Factor (BDNF) and apoptotic proteins such as caspase-3 in the pathogenesis of glaucoma. Epigallocatechin-3 Gallate (EGCG), the main flavonoid in green tea, is known for its antioxidant, anti-apoptotic, and neuroprotective properties.

Objective

To determine the effect of oral EGCG supplementation on the expression of BDNF and caspase-3 in Wistar rats with a glaucoma model induced by intravitreal injection of N-Methyl-D-Aspartate (NMDA).

Metode

This study was a true experimental post-test only randomized controlled group design using 14 male Wistar rats with glaucoma model (aged 6–8 weeks, weighing 150–300 g). The animals were divided into two groups: treatment (oral EGCG 50 mg/kgBW/day) and control (aquadest placebo), observed for 14 days. The expression of BDNF and caspase-3 in the RGC layer was assessed using real-time Polymerase Chain Reaction.

Hasil

BDNF expression in the treatment group was significantly higher ($14,36 \pm 2,15$) than in the control group ($8,32 \pm 3,06$) ($p=0.001$). Caspase-3 expression in the treatment group ($8,12 \pm 1,96$) was significantly lower than in the control group ($18,23 \pm 4,99$) ($p=0.002$). Correlation analysis showed a negative relationship between BDNF and caspase-3 expression in the treatment group, which was not statistically significant ($p=0.056$).

Conclusion

Oral EGCG supplementation has the potential to exert neuroprotective and anti-apoptotic effects in glaucoma by increasing BDNF expression while decreasing caspase-3 expression.

Keywords

BDNF, *Caspase-3*, EGCG, glaucoma, retinal ganglion cell, RGC