see Purtscher’s retinopathy.

Visual impairment in diabetes is a growing public health concern. Apart from the well-defined diabetic retinopathy, disturbed optic nerve function, which is dependent on the myelin sheath, has recently been recognized as an early feature of visual impairment in diabetes. However, the underlying cellular mechanisms remain unclear. Using a streptozotocin-induced diabetic mouse model, Wu et al. observed a myelin deficiency along with a disturbed composition of oligodendroglial lineage cells in the diabetic optic nerve. They found that new myelin deposition, a continuous process that lasts throughout adulthood, was diminished during pathogenesis. Genetically dampening newly generated myelin by conditionally deleting olig2 in oligodendrocyte precursor cells within this short time window extensively delayed the signal transmission of the adult optic nerve. In addition, clemastine, an antimuscarinic compound that enhances myelination, significantly restored oligodendroglia and promoted the functional recovery of the optic nerve in diabetic mice. The results point to the role of new myelin deposition in optic neuropathy under diabetic insult and provide a promising therapeutic target for restoring visual function [1)]

1)
Wu H, Chen X, Yu B, Zhang J, Gu X, Liu W, Mei F, Ye J, Xiao L. Deficient deposition of new myelin impairs adult optic nerve function in a murine model of diabetes. Glia. 2023 Jan 20. doi: 10.1002/glia.24341. Epub ahead of print. PMID: 36661098.