

whole-mount cornea on day 14 post-inoculation. Anti-LYVE-1 antibody, anti-CD11b antibody and anti-F4/80 antibody were used for immunostaining. Corneal infection was graded by a previously reported method and central corneal thickness as a measure of corneal edema was evaluated by anterior segment optical coherence tomography (CASIA SS-1000; Tomey, Nagoya, Japan).

【Results】 Lymphangiogenesis was significantly reduced by macrophage depletion on day 14 post-inoculation. There is no significant difference the control group and the macrophage depleted group on day 7 postinoculation. However, clinical infection score and corneal edema significantly increased in the macrophage depleted group on day 14 post-inoculation.

【Conclusions】 These results suggest that the process of lymphangiogenesis in bacterial infection of the cornea presumably suppresses keratitis in the late stage of infection.

P1-05.

Amyloid and tau positron emission tomography in diabetes-related dementia

(社会人大学院博士課程3年高齢総合医学)

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Our proposed clinical entity, referred to as diabetes-related dementia (DrD), describes a dementia state predominantly associated with type 2 diabetes mellitus (DM)-related metabolic abnormalities. We studied 11C-PiB and 11C-PBB3 positron emission tomography (PET) in 29 subjects with DrD and 5 subjects with Alzheimer disease (AD) associated with DM to assess amyloid and tau deposits in the brain. Different from

AD, only 11 out of 29 subjects (38%) with DrD showed positive PiB, whereas 17 out of 19 (89%) showed positive PBB3. Depending on positivity of PiB and PBB3, we classified subjects with DrD into a negative PiB and positive PBB3 pattern (53%), indicating tauopathy, a positive PiB and positive PBB3 pattern (32%), indicating AD pathology, or a negative PiB and negative PBB3 pattern (16%), indicating non-specific neuronal damage. DrD showed variable amyloid and tau accumulation patterns in the brain. DrD may be associated predominantly with tau pathology, in addition to AD pathology and non-specific neuronal damage due to DM-related metabolic abnormalities.

P1-06.

Analysis of aggregated proteins in HSPB8 myopathy using zebrafish models

(病態生理学)

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Heat shock protein B8 (HSPB8), a member of the small heat shock protein family, is known to have chaperone activity and be involved in protein quality control. Previous studies reported that mutations in *HSPB8* cause several neuromuscular diseases. Recently, two novel candidate mutations of *HSPB8* were identified in families with protein-aggregated myopathy. However, the pathogenic mechanisms of HSPB8 myopathy remains to be elucidated. In this study, we firstly establish zebrafish models of HSPB8 myopathy to confirm the pathogenicity of these novel *HSPB8* mutations. We also tried to identify abnormal aggregated proteins for the purpose of clarifying the pathological mechanisms of HSPB8 myopathy. We carried out microinjection of wild-type or mutant human *HSPB8* mRNA in zebrafish embryos at 1-2 cell stage. Then we analyzed phenotype of these fish at 5 days post-fertilization. Overexpression of mutant *HSPB8* mRNA resulted in morphological abnormalities at higher rate compared to expressing wild-type *HSPB8* mRNA-injected and uninjected fish. Furthermore, it revealed that these abnormal fish had severe muscle degeneration and protein aggregation. Our data suggest that the