G82 Purtscher Retinopathy Detected by Postmortem Monocular Indirect Ophthalmoscopy

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The goals of this presentation are to: (1) describe the retinal features of the Purtscher retinopathy, (2) list conditions and disorders associated with Purtscher retinopathy, and (3) describe the histological features of Purtscher-flecken.

After attending this presentation, attendees will gain insight into the value of postmortem monocular indirect ophthalmoscopy (PMIO) in detecting Purtscher retinopathy and subsequent histopathological description of the observed retinal lesions. This presentation will impact the forensic community by providing an introduction to non-hemorrhagic retinopathies detectable by PMIO and consequent histopathological characterization.

In 1910, Dr. Otmar Purtscher described a patient with severe head trauma who had a hemorrhagic and vaso-occlusive retinopathy characterized by multiple variably sized cotton-wool spots (Purtscher-flecken) plus retinal hemorrhages around the optic nerve head. Two years later he designated the condition angiopathia retinae traumatica. Since then the term Purtscher retinopathy has been used to describe a clinical picture of angiopathia retinae traumatica even in the absence of known head trauma. Purtscher-like retinopathy has been observed in a variety of conditions including compressive chest injuries, long bone fractures, retrobulbar anesthesia, connective tissue and vasculitic diseases, orthopedic surgery, acute pancreatitis, strenuous childbirth, and battered child syndrome. The exact pathophysiologic mechanism causing Purtscher or Purtscher-like retinopathy remains controversial and published supportive histological descriptions are rare. Two cases are described of Purtscher retinopathy detected by postmortem monocular indirect ophthalmoscopy plus the histological features and immunohistochemical staining for β-amyloid precursor protein of observed Purtscher-flecken.

Case 1: A 36-year-old man sustained a traumatic brain injury with immediate loss of consciousness following an assault in a parking lot. Cranial computed tomography revealed subdural and subarachnoid hemorrhages. He remained comatose and died thirteen days after the injury. Neuropathological examination revealed traumatic axonal injury, a cerebral contre-coup contusion plus organizing subdural and subarachnoid hemorrhages. Prior to autopsy PMIO identified retinal hemorrhages and multiple posterior, peripapillary, polygonal foci of retinal whitening (Purtscher-flecken) distributed between retinal arterioles and veins. Histologically, these areas were collections of swollen, contracted axons (cytoid bodies) in the nerve fiber layer highlighted by immunohistochemical staining for β-amyloid precursor protein.

Case 2: A 27-year-old man had experienced nausea and vomiting for a number of days. While at his girlfriend's residence he collapsed following an episode of vomiting. Resuscitative efforts were unsuccessful and he was pronounced dead in the emergency department. Hepatosplenomegaly was present at autopsy and his hypercellular bone marrow contained > 20% myeloid blasts with Auer rods. The leukemic cells stained positively for CD68 and myeloperoxidase. Prior to autopsy PMIO revealed multiple bilateral retinal hemorrhages, many white-centered, plus posterior foci of small Purtscher-flecken. Histologically, these foci were cytoid bodies in the nerve fiber layer that stained positively for β-amyloid precursor protein.

No published reports of Purtscher retinopathy detected initially at autopsy are in the medical and scientific literature. Previous articles on the histopathology of Purtscher retinopathy have been two case reports from patients with acute pancreatitis who died 6 and 23 days after the onset of their illness. Both had focal areas of retinal edema and loss of architecture in the inner retinal layers with abrupt transition to normal retina. In the author's reported cases the detected Purtscher-flecken were cytoid bodies that stained positively for β-amyloid precursor protein. These inner retinal collections of swollen, contracted axons are relatively nonspecific and histopathologically similar to retinal cotton-wool spots and foci of axonal injury observed throughout the nervous system commonly associated with a variety of traumatic and non-traumatic conditions.

Purtscher Retinopathy, Traumatic Brain Injury, ß-Amyloid Precursor Protein

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