Original Article

Purtscher-like retinopathy associated with antibiotic anaphylaxis

HUANG Zhixuan, ZENG Zhibing, XU Zhuping

Departmen of Ophthalmology, West China Hospital of Sichuan University, Chengdu 610041, China

Abstract: We report a case of an 18-year-old woman with systemic antibiotic anaphylaxis who presented anasarca and bilateral visual loss two weeks after the intravenous use of pazufloxacin. Ancillary fundus tests revealed bilateral cotton-wool spots, Purtscher flecken, edema, and retinal arteriolar occlusion around the optic disc. After pulse corticosteroid, administration of anti- anaphylactic agent, and general support therapy for one month, the patient showed a favorable change, with the symptoms lessened or free systemically, except the continuous aggravation of fundus ischemic change. After two intravitreal injections of Ranibizumab, there were still a large area of capillary non-perfusion and neovacularizations in the binocular retina, resulting in vitreous hemorrhage. The patient's visual acuity had still light perception after right-eye vitrectomy and presented no improvement during the postoperative follow-up of two years. Literature review revealed that there were many causes associated with Purtscher-like retinopathy, and the consequent visual impairment varied significantly. Prompt management of the underlying condition is crucial in giving the patient the best chance to restore vision.

Keywords: Purtscher's retinopathy; Purtscher-like retinopathy; antibiotic; anaphylaxis

INTRODUCTION

Purtscher's or Purtscher-like retinopathy is an occlusive retinal microangiopathy most commonly developed after craniocerebral trauma, thoracic crushing injury, acute pancreatitis, renal failure, various kinds of autoimmune diseases, or due to the administration of a multitude of chemotherapeutic agents or [1-2]. We describe here a case of Purtscher-like retinopathy associated with systemic antibiotic anaphylaxis, and review the literatures on this rare condition.

CASE REPORT

An 18-year-old female inpatient of rheumatology department presented subacute painless decreased vision in both eyes. She had a history of intravenous use of pazufloxacin 2 weeks before admission due to high fever, chill and sore-throat. Itching rashes appeared on her chest and back 3 days after the drug administration and consequently the drug was withdrawn and changed to cefazolin. One week later she was admitted because of deterioration of her whole body condition and was diagnosed as anaphylaxis probably due to pazufloxacin. During the first week after admission, she underwent epileptic seizure twice. Imaging examination showed pulmonary interstitial changes, plural effusion, pericardial effusion, and pelvic carving effusion. Visual acuity was counting fingers in each eye. No inflammation in anterior chamber and vitreous body presented. Ocular fundus examination showed bilateral multiple cotton-wool spots and Purtscher flecken around the optic disc, and a few intra-retinal hemorrhages (Fig.1).

Fluorescein angiography showed retinal vein tortuous expansion, retinal arterial and capillary occlusion around the optic disc and macular non-perfusion in the early stage (Fig.2 A&B) and vessel wall staining, leakage of veins, venules and some arterioles in the late phase (Fig.2 C&D). OCT showed inter-retinal edema and structure disorganization of macular due to exudate and leakage (Fig.3). Bilateral Purtscher-like retinopathy was diagnosed, probably caused by antibiotic pazufloxacin anaphylaxis. After pulse corticosteroid, administration of anti-anaphylactic agent, and general support therapy for one month, the patient recovered systemically. Despite twice intravitreal injection of steroid and Ranibizumab, there were still large area of capillary non- perfusion and neovacularizations developed in the bilateral retina, resulting in vitreous hemorrhage. The patient's visual acuity was still light perception after right-eye vitrectomy and presented no improvement during the postoperative follow-up of two years.

Discussion

Purtscher's retinopathy generally occurs as a result of craniocerebral trauma, thoracic crushing injury, or long bone fracture. It was first described in 1910 by Otmar Purtscher a middle-aged man who fell off a tree and suffered cranial trauma [2, 11]. When there is a

Received: 2018-02-05 **Accepted:** 2018-03-13
Supported by National Major Scientific Equipment Program of China (2012YQ12008005).
Corresponding author: XU Zhuping, Tel: 028-85422542, E-mail: xuzp@hotmail.com.

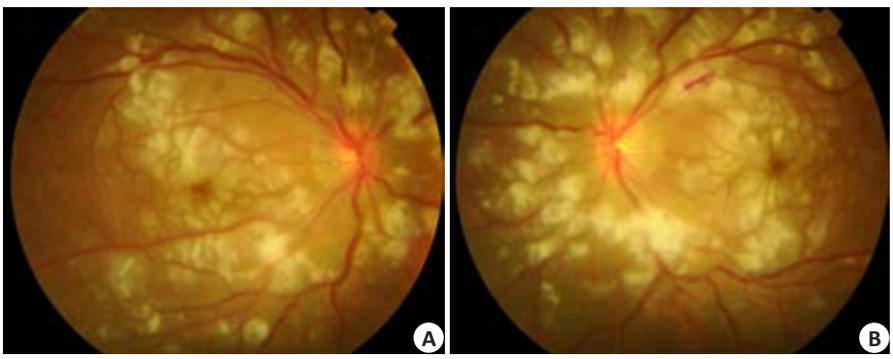

Fig.1 Ocular fundus examination. **A**: Right eye; **B**: Left eye. Photos showed bilateral symmetric multiple cotton-wool spots and Purtscher flecken surround the optic disc, and a few intra-retinal hemorrhages.

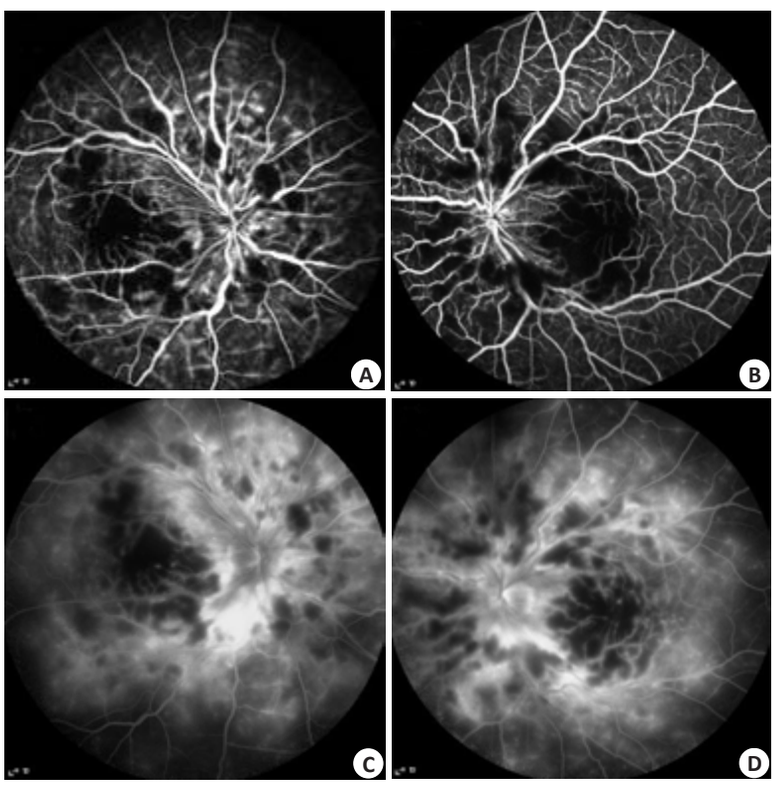

Fig.2 Fudus fluorescein angiography. **A**, **B**: retinal vein tortuous expansion, retinal arteriolar and capillary occlusion around the optic disc and macular non-perfusion in early stage of the right eye and left eye; **C**, **D**: Vessel wall staining and leakage of veins, venules and some arterioles in the late phase.

non-traumatic etiology, the correct designation is Purtscher-like retinopathy. Purtscher-like retinopathy has been reported to occur in several pathologic conditions and after different medical disorders and interventions, including acute pancreatitis, collagen diseases, renal failure, childbirth, retrobulbar anesthesia, periorbital injections, chemotherapy for cancer, cerebro- or cardiovascular surgery, and acute myocardial infarction [3-10]. Patients with Purtscher-like retinopathy usually present with a wide range of visual acuities, from a visual acuity (VA) of 6/6 with minimal visual disturbance, to severe visual loss with a vision of light perception, which have similar appearance in the ocular fundus. The most common signs of this retinopathy are cotton-wool spots, retinal hemorrhages, Purtscher flecken, pseudo-cherry red spot, and macular edema [2, 9, 11]. Relative afferent pupillary defects have been reported in cases with unilateral disease [12] as well as bilateral disease [13]. It is widely assumed that the Purtscher flecken is the result of occlusion of the pre-capillary arterioles [13]. The characteristic finding is intra-retinal whitening but with a clear zone (within 50 mm) on either side of the retinal arterioles, venules, and precapillary arterioles. This is in contrast to cotton-wool spots, which have ill-defined edges and are located superficially over vessels [2]. Fluorescein angiography may show leakage of dye from retinal arterioles, capillaries, and venules in patients with relatively mild

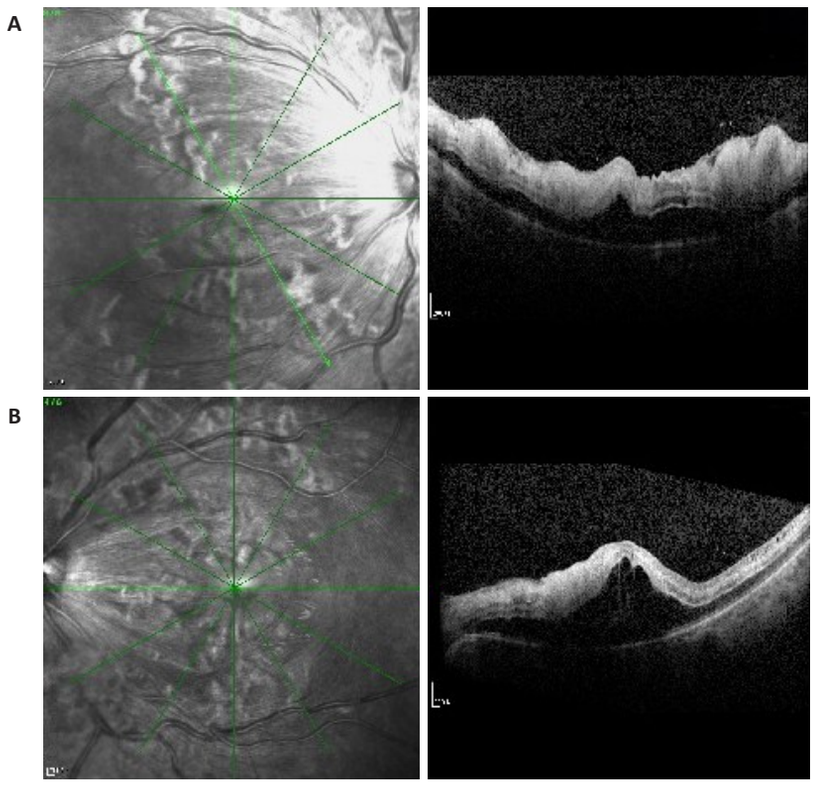

Fig.3 Optical coherence tomography(OCT) bilaterally. **A**: right eye; **B**: left eye. Tomography showed inter-retinal edema and structural disorganization of macular due to exudate and leakage.

Purtscher's retinopathy. Arteriolar obstruction is noted in more severe cases [3-11]. The typical appearance of the macula on optical coherence tomography (OCT) at initial diagnosis is that of varying degrees of retinal thickening and edema with some cases even presenting subretinal serous fluid [14-16]. Several months on, most cases have resolution of the thickening, but some still show retinal atrophy and destruction of the foveal architecture. There are many theories about pathogenesis but Purtscher's microangiopathy is typically thought to be caused by precapillary occlusion, fat emboli, leukoaggregation, or other mechanisms[4, 17-18].

Because of the small frequency of this pathology, estimated as 0.24 per million [2], it's hard to perform prospective trial for the evidence-based treatment guideline. At present, there is no consensus on the treatment. The most frequent treatment prescribed is high-dose intravenous steroids, usually with a tapering dose of oral steroids subsequently. The rationale behind corticosteroid use is sound, as it is capable to inhibit complement activation and granulocyte aggregation, as well as to allow nerve fiber recovery by stabilizing damaged neuronal membranes and microvascular channels. However, there are controversies over the visual recovery via the administration of steroid [2, 17, 19]. Observation and treatment of the underlying etiology may be the most reasonable therapeutic option with no adverse drug effects. There are isolated reports using peripheral vasodilator, hyperbaric oxygen therapy, or low molecular weight heparin postulated intravenously to increase choroidal and retinal blood flow, dilate retinal arterioles, or increases oxygen supply to the choroids and inner retina [17, 19, 21-22]. For sever cases, the visual prognosis was usually poor. Large area of arteriole occlusion leads to hypoxia and neovascularization subsequently. Like the case we report here, prompt intra-vitreous injection of anti-VEGF agent or pan-retinal photocoagulation is necessary. Unfortunately, this patient could not obtain enough injections due to her poor systemic condition then.

CONCLUSION

For the first time, we present here a sever case of Purtscher-like retinopathy associated with antibiotic anaphylaxis probably due to pazufloxacin. The literatures associated with Purtscher-like retinopathy have pointed out diverse causes and the consequent visual impairment varies significantly [1, 23]. Prompt management of the underlying condition is crucial in giving the patient the best chance to restore vision[11].

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抗生素过敏反应所致Purtscher样视网膜病变1例报告及文献复习

黄志翾，曾志冰，徐筑萍
四川大学华西医院眼科，成都 四川 610041

摘要：本文报告1例由抗生素帕珠沙星过敏所致Purtscher样视网膜病变临床表现。患者18岁，因发热、寒颤和咽部疼痛予“帕珠沙星”抗感染治疗后出现皮疹、瘙痒、全身水肿和视力下降，停药后病情仍持续加重。影像学检查显示：肺间质改变、心包积液、腹腔积液和盆腔积液。双眼眼底为典型的Purtscher样表现：黄斑和视盘周围大量棉绒般、视网膜Purtscher斑，视网膜水肿。经过激素冲击治疗、抗过敏药物使用及全身支持治疗1月后，患者全身情况逐渐好转，但眼底缺血性改变继续加重，玻璃体腔注射抗新生血管药物康柏西普2次仍迅速发展至双眼视网膜新生血管形成和玻璃体出血，进行玻璃体切除术后视力仍为光感。术后随访2年视力无提高。Purtscher及Purtscher样视网膜病变病因多样，患者视力受损程度差异大，眼底表现为典型的双眼对称后极部散在视网膜棉絮斑、Purtscher斑、出血和水肿，眼底荧光血管造影表现为不同程度视网膜小动脉阻塞无灌注、后期荧光素血管管壁渗漏和着染，Purtscher斑分布的相应区域为弱荧光。通常结合患者眼底表现、全身疾病及其病史作出诊断。发病机制可能为各种原因的视网膜前小动脉阻塞。目前尚无治疗指南。激素治疗有争议，但若严重影响视力，应积极去除潜在全身病因，必要时激素冲击治疗。严重病例谨防病情发展至视网膜新生血管和出血，需密切观察及时进行抗新生血管的预防和治疗。

关键词：Purtscher-like视网膜病变；Perstscher 视网膜病变；过敏反应；抗生素

收稿日期：2018-02-05
基金项目：国家重大科学仪器设备开发专项(2012YQ12008005)
作者简介：黄志翾，住院医师，E-mail: kai8xuan@163.com
通信作者：徐筑萍，博士，副主任医师，E-mail: xuzp@hotmail.com