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INFLAMMATORY DISORDERS

Clinical manifestation of Fuchs uveitis syndrome in childhood

Christoph Tappeiner · Jens Dreesbach · Martin Roesel · Carsten Heinz · Arnd Heiligenhaus

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Abstract
Purpose The aim of this study was to describe clinical signs and complications of Fuchs uveitis syndrome (FUS) with onset in childhood.
Methods Ophthalmologic findings and complications in patients with FUS becoming manifest before the age of 16 years were analyzed in a retrospective study at a tertiary referral uveitis center. Inclusion criteria were the presence of pathognomonic FUS findings at any time point and exclusion of any systemic immune-mediated or infectious disease.
Results A total of 23 patients (male=16, female=7) with juvenile FUS (unilateral n=20, bilateral n=3 patients) were included in the study. Mean ages at uveitis and FUS diagnosis were 12.0±4.2 and 22.7±10.7 years, respectively. In six patients, inflammation was noted at age≤7 years. The following inflammatory signs were observed in a total of 26 eyes: ≤ 1+ anterior chamber cell grade (n=26), vitreous cells (n=24), fine keratic precipitates (KPs; n=23), stellate KPs (n=11), mutton-fat KPs (n=23), diffuse (n=24) or inferior (n=8) distribution of KPs, Koeppe nodules (n=10), and iris heterochromia (n=14). A representative subgroup of patients (n=5) is shown who presented with non-specific clinical signs in the beginning and in whom typical FUS signs became manifest only at a later stage. Secondary complications such as cataract (n=19), ocular hypertension (n=3), or glaucomatous disc damage (n=2) were found after a mean uveitis duration of 11.6, 19.5, and 20.3 years, respectively.
Conclusion FUS may begin in early childhood, and the characteristic findings may not be present at onset of disease. The diagnosis is often delayed for years, occasionally with the consequence of overtreatment with anti-inflammatory drugs.

Keywords Fuchs uveitis syndrome · Children · Diagnosis · Clinical signs · Complications

C. Tappeiner · J. Dreesbach · M. Roesel · C. Heinz · A. Heiligenhaus (✉)
Department of Ophthalmology, St. Franziskus Hospital, Hohenzollernring 74, 48145 Münster, Germany
e-mail: arnd.heiligenhaus@uveitis-zentrum.de

C. Heinz · A. Heiligenhaus
University of Essen, Essen, Germany

C. Tappeiner
Department of Ophthalmology, Inselspital, University of Bern, Bern, Switzerland

Introduction

Fuchs uveitis syndrome (FUS) describes a chronic, granulomatous, low-grade, and often unilateral anterior segment inflammation of the eye. This syndrome was first described by Ernst Fuchs in 1902 [1]. Typical clinical features include mutton-fat or stellate keratic precipitates (KPs) on the entire corneal endothelium surface, a low anterior chamber cell grade, some cells and/or opacities in the anterior vitreous, the absence of posterior synechiae, and, commonly, iris atrophy [2]. Iris nodules may be observed on the pupillary margin (Koeppe) and also in the iris stroma (Busacca) [3, 4]. FUS patients comprise about 6.2 % [5] to 6.9 % [6] of the uveitis cases at tertiary referral centers and up to 12 % when considering only patients with chronic anterior uveitis [7]. In certain countries, for example, in Italy, these patients may account for up to 22.7 % of uveitis cases in general or up to 45 % of cases when considering only patients with anterior uveitis [8]. Only a small amount of clinical data about FUS in children have been published up to now [9, 10].

A delay from the manifestation of the first symptoms, detecting the presence of uveitis, and correctly diagnosing FUS represents a common problem [11]. Patients with FUS are

Table 1 Epidemiological data. Patients (23 patients; 26 eyes) with Fuchs uveitis syndrome becoming manifest before the age of 16 years

Sex	
Male (N/%)	16/69.6 %
Female (N/%)	7/30.4 %
Eyes with uveitis	
Unilateral (N/%)	20/87 %
Bilateral (N/%)	3/13 %
Age (years) at uveitis diagnosis	12.0 ±4.2 (4.0 – 16.0)
Age (years) at FUS diagnosis	22.7 ±10.7 (9.0 – 37.5)
Age (years) at first visit to reporting uveitis center	23.8 ±10.9 (8.8 – 37.5)
Mean follow-up (years) since uveitis diagnosis	14.0 ±11.3 (0.03 – 36.5)

frequently asymptomatic for years before diagnosis. Iris heterochromia may be absent or subtle, especially in patients with a brown iris [12–14]. An infectious etiology has been discussed recently, e.g., possible associations with toxoplasmosis [15, 16], rubella virus [17–21], or herpes viruses [20, 22]. However, FUS is still diagnosed by recognizing the patterns of typical clinical signs and is not primarily based on any laboratory testing.

The aim of this study was to characterize the manifestation of FUS in children, including early clinical signs, and occurrence of complications during further follow-up.

Methods

Medical records of all children with FUS (onset of disease before 16 years of age) seen between 1997 and 2011 at the Department of Ophthalmology at the St. Franziskus Hospital Muenster (Germany) were retrospectively reviewed. No institutional review board approval is required for such chart review studies in Germany. Uveitis in childhood was defined as an onset of disease before 16 years of age, according to childhood definition in pediatric nomenclature and to allow comparability to uveitis associated with juvenile idiopathic arthritis.

FUS diagnosis was based on the following criteria [2, 3, 11, 12, 14, 23]: chronic anterior uveitis with≤1+ cell grade in the anterior chamber and insidious onset of flare; absence of significant acute exacerbations [24]; fine and stellate KPs diffusely spread on the entire corneal endothelium; diffuse iris atrophy with or without obvious heterochromia; and lack of posterior synechiae. Rheumatologic examinations included a review of systems, laboratory tests (including HLA-B27, complete blood count, urine analysis, angiotensin-converting enzyme, and anti-nuclear antibodies [ANA]), and chest X-ray examination if an underlying systemic disease was suspected. Any associated immune-mediated systemic (e.g., sarcoidosis, juvenile idiopathic arthritis) or infectious diseases (e.g., lues, borreliosis, or tuberculosis) were excluded by laboratory testing and medical history.

Medical history and ophthalmologic findings such as best-corrected visual acuity (BCVA), slit-lamp examination, applanation tonometry (Goldmann), gonioscopy, and fundoscopy were recorded according to a standardized protocol, which was previously introduced for documenting patients with JIA-associated uveitis [25]. Features of KPs regarding form (mutton-fat, fine, or stellate [2]), pigmentation (without/with), and distribution (disseminated on entire corneal endothelium or inferior half of corneal endothelium) were documented. Data including current age, gender, age at first diagnosis of uveitis, duration of uveitis prior to first presentation to our tertiary uveitis referral center, age at diagnosis of FUS, ocular complications (cataract, ocular hypertension, and glaucomatous disc damage), surgical eye procedures, and duration of follow-up were also collected. Sustained intraocular pressure (IOP)≥21 mmHg without pathological disc cupping was defined as ocular hypertension, whereas the presence of

Table 2 Type of keratic precipitates (KPs) by time point of first documentation. Patients (23 patients; 26 eyes) with Fuchs uveitis syndrome becoming manifest before the age of 16 years

	N (eyes)/%	Age (years) at initial documentation mean±SD (range)	Years after uveitis diagnosis mean±SD (range)
Type of KPs			
Fine	23/88.5 %	21.9±11.3 (6.9 – 37.5)	9.0±10.5 (0 – 36.5)
Stellate	11/42.3 %	18.7±8.9 (7.4 – 37.7)	7.5±8.9 (0 – 27.4)
mutton-fat	23/88.5 %	22.6±10.5 (9.0 – 37.5)	10.6±11.0 (0 – 36.5)
Localization of KPs			
Inferior	8/30.8 %	12.4±4.5 (6.8 – 19.9)	1.8±2.9 (0 – 9.0)
Diffuse	24/92.3 %	22.8±10.6 (9.0 – 37.5)	10.6±10.8 (0 – 36.5)
Pigmentation of KPs			
No	23/88.5 %	23.5±12.1 (6.8 – 37.5)	10.9±12.0 (0 – 36.5)
Yes	8/30.8 %	27.3±12.3 (14.9 – 37.5)	13.7±11.6 (0.8 – 36.5)

Table 3 Type of keratic precitipates (KPs) by time point of first documentation. Patients (six patients; eight eyes) with Fuchs uveitis syndrome seen at the tertiary uveitis center before the age of 16 years

	N (eyes)/%	Age (years) at initial documentation mean±SD (range)	Years after uveitis diagnosis mean±SD (range)
Type of KPs			
fine	8/100 %	11.6±3.4 (6.9 – 15.4)	0.6±0.4 (0 – 1.3)
stellate	5/62.5 %	11.1±3.0 (7.4 – 14. 9)	0.7±0.6 (0 – 1.8)
mutton-fat	8/100 %	12.3±2.4 (9.0 – 15.2)	1.3±1.3 (0 – 3.06)
Localization of KPs			
inferior	5/62.5 %	10.1±3.6 (6.8 – 15.2)	0.8±0.6 (0 – 1.8)
diffuse	7/87.5 %	12.3±2.2 (9.0 – 15.2)	1.2±1.2 (0 – 3.1)
Pigmentation of KPs			
No	7/87.5 %	11.5±3.3 (6.8 – 15.2)	0.9±1.1 (0 – 2.9)
Yes	2/25 %	15.0±0.2 (14.9 – 15.2)	0.9±0.1 (0.8 – 1)

glaucomatous disc damage was defined as glaucoma [26]. Data were analyzed concerning the first time typical inflammatory signs and complications of FUS were documented.

Descriptive statistical analysis was performed using GraphPad Prism version 6.0e (GraphPad Software Inc., La Jolla, CA, USA). Values are provided as mean±standard deviation (SD) and range (minimum – maximum).

Results

In a retrospective chart analysis, 23 patients (female n=7, male n=16) with FUS and onset before 16 years of age were identified (in six patients FUS was noted at≤7 years of age). This represented 7.8 % of all FUS patients and 3.2 % of patients with uveitis onset before 16 years of age who were in the database of the tertiary uveitis center during this time period. The epidemiological data are summarized in Table 1. In three of the patients, both eyes were affected. The mean age at uveitis diagnosis was 12.0±4.2 years. Patients were referred to us following a mean duration of uveitis of 11.9±11.3 years. Topical steroids were applied in 14 out of 23 patients prior to FUS diagnosis.

Different types of KPs were documented (Table 2), and also a change in KP type during the course of disease was obvious for certain patients. At initial documentation, fine KPs were found in 20 eyes (76.9 %), whereas stellate KPs were present in nine eyes (34.6 %) and mutton-fat KPs in 19 eyes (73.1 %). During follow-up, new fine KPs were found in three eyes, new stellate KPs in two eyes, and new mutton-fat KPs in four eyes. Diffuse KPs on the entire corneal endothelium were observed in 19 eyes at initial documentation and in a total of 24 eyes (92.3 %) after a mean uveitis duration of 10.6 years (range 0 – 36.5). Inferior KPs were documented in eight eyes (30.5 %) after a mean uveitis duration of 1.8 years (range 0 – 9.0; five eyes with inferior KPs documented in the first year after uveitis diagnosis, three eyes with inferior KPs documented within 2.5 years). In eight eyes (30.8 %) pigmented KPs were observed after a mean of 13.7 years (range 0.8 - 36.5) following uveitis onset. Analyzing the subgroup of patients who had been seen at our tertiary uveitis center already before 16 years of age and not later on (six patients; eight eyes; Table 3), fine KPs were documented 0.6±0.4 years after uveitis diagnosis, whereas stellate or mutton-fat KPs were documented slightly later (0.7±0.6 and 1.3±1.3 years after uveitis diagnosis, respectively). Furthermore, inferior KPs were documented earlier than a diffuse

Table 4 Clinical signs with age at initial documentation and observation interval after uveitis diagnosis. Patients (23 patients; 26 eyes) with Fuchs uveitis syndrome becoming manifest before the age of 16 years. *n.a* not applicable

	N (eyes)/%	Age (years) at initial documentation mean±SD (range)	Years after uveitis diagnosis mean±SD (range)
AC cell-grade≤1+	26/100 %	12.0 ±4.2 (4.0 – 16.0)	n.a
Koeppe nodules	10/38.5 %	21.7±8.8 (10.5 – 40.4)	9.0±8.4 (0 – 24.4)
Iris			
diffuse atrophy	24/92.3 %	23.9±11.2 (9.0 – 37.5)	11.8±11.4 (0 – 36.5)
heterochromia	14/53.8 %	25.9±11.2 (9.8 – 37.5)	14.0±11.4 (0.8 – 36.5)
Vitreous cells	24/92.3 %	21.9±8.9 (8.8 – 39.6)	9.9±9.7 (0 – 33.1)
Vitreous haze≥1+	16/61.5 %	21.9±8.0 (8.8 – 39.6)	9.8±9.4 (0.4 – 33.1)
Posterior synechiae	0/0 %	/	/

Table 5 Initial documentation of secondary complications. Patients (23 patients; 26 eyes) with Fuchs uveitis syndrome becoming manifest before the age of 16 years

	N (eyes)/%	Age (years) at initial documentation mean±SD (range)	Years after uveitis diagnosis mean±SD (range)
Cataract	19/73.1 %	23.8±9.5 (5.3 – 39.6)	11.6±10.0 (0 – 33.1)
Ocular hypertension	3/11.5 %	34.5±0.7 (34.0 – 35.5)	19.5±2.8 (17.0 – 23.4)
Glaucoma	2/7.7 %	34.8±0.9 (34.0 – 35.7)	20.3±3.3 (17.0 – 23.6)
Macular edema	0/0 %	/	/

distribution of KPs (0.8±0.6 and 1.2±1.2 years after uveitis diagnosis, respectively).

The maximal AC cell grade during follow-up was 1+ in all patients. Koeppe nodules, iris atrophy, and heterochromia were observed after a mean of 9.0 (range 0 – 24.4; n=10 eyes), 11.8 (range 0 – 36.5; n=24 eyes), and 14.0 years (range 0.8 – 36.5; n=14 eyes) of uveitis duration, respectively (Table 4). Cells were found in the anterior vitreous cavity in 24 eyes (92.3 %) after a mean of 9.9 years (range 0 – 33.1). Vitreous haze≥1+ [24] was observed in 16 out of 26 eyes (61.5 %) after a mean of 9.8 years following uveitis onset (range 0.4 – 33.1), Table 4.

In none of the FUS eyes were posterior synechiae observed at any time point. Secondary complications, such as cataract (19 eyes, 73.1 %), ocular hypertension (three eyes, 11.5 %), or glaucoma (two eyes, 7.7 %), were diagnosed after a mean of 11.6 years (range 0 – 33.1), 19.48 years (range 17.0 – 23.4), and 20.3 years (range 17.0 – 23.6), respectively (Table 5). Macular edema was not found at any time point in these patients. Cataract surgery (14 eyes, 53.8 %), vitrectomy (7 eyes, 26.9 %), and glaucoma surgery (2 eyes, 7.7 %) were performed after a mean uveitis duration of 11.7, 13.7, and 29.6 years, respectively. KPs did not differ between patients with or without surgery (data not shown).

Figure 1 shows a selection of five patients who presented with non-specific clinical signs in the beginning and in whom typical FUS signs became manifest only at a later stage. In all of them, KPs initially were located inferiorly, while diffuse distribution of KPs – typical for FUS – appeared only during the later course of disease. Initially, fine KPs were documented, whereas stellate or mutton-fat KPs were seen either simultaneously (n=1 patient) or only during the further course of disease (n=4 patients). The pattern did not differ between patients who received (n=14) or did not receive (n=9) topical corticosteroids. In all of these patients, vitreous cells and/or haze were observed only at a later stage.

Discussion

Our data show that FUS may become manifest as soon as in early childhood. As the initial clinical signs – especially in the first months of uveitis onset – may not be pathognomonic for FUS, a correct diagnosis may be difficult and is often delayed. Especially in children, a slight decrease in visual acuity or vitreous floaters may presumably go unnoticed for a longer period of time. The differential diagnosis includes all other anterior uveitis entities with insidious onset of flare, especially juvenile idiopathic arthritis-associated uveitis (JIAU). Important factors for diagnosing JIAU are antinuclear antibodies (ANA), early onset≤4 years, and early/initial onset of complications [27]. On the other hand, clinical signs such as vitreous cells are common in FUS, but rare in JIAU with a mild degree of inflammation.

This study was performed at a tertiary uveitis center with appropriate documentation and internationally acknowledged uveitis nomenclature [24]. Nevertheless, we detected a

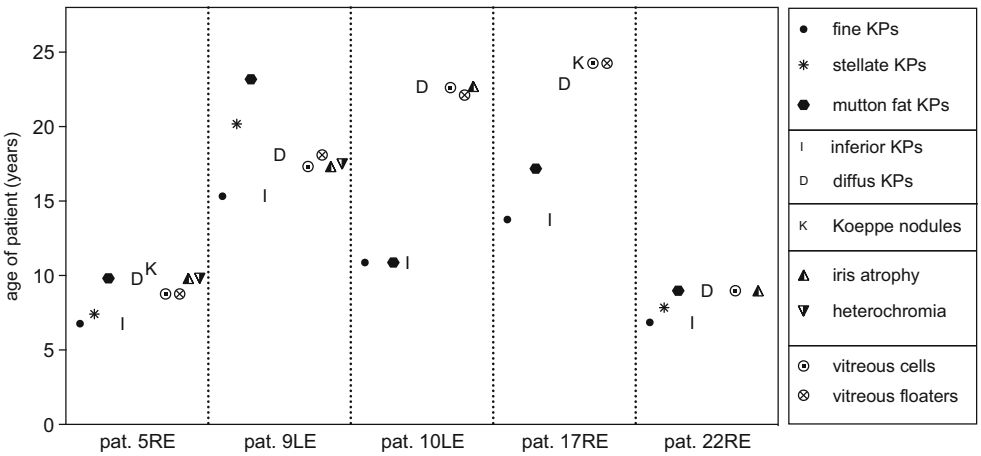

Fig. 1 Representative case series of 5 patients with non-specific uveitis signs in the beginning who only developed typical signs of Fuchs uveitis syndrome during the further course of disease

subgroup of patients with non-specific uveitis signs in the beginning and in whom typical signs for FUS only became manifest during the further course of disease. Interestingly, we observed a change in the type of KPs: an inferior distribution of fine and/or stellate KPs was found in early stage, whereas no characteristic or pathognomonic clinical signs for FUS were present. At a later stage, KP type changed to a diffuse distribution of KPs, with mainly a fine and mutton-fat appearance, which is typical for FUS.

For differential diagnosis, it has to be considered that stellate KPs may not only be seen in FUS, but have also been described in uveitis due to sarcoidosis [28], toxoplasmosis [29], and viral uveitis, for example, due to HSV, VZV, CMV, or rubella [30]. Our data suggest that stellate KPs are not always present at a very early stage and may develop during the further course of disease. Typical signs such as iris atrophy or heterochromia were observed at a late stage. When these signs are seen as early as at the first diagnosis of uveitis, they may indicate a long-standing, subclinical course of disease. On the other hand, subtle iris changes may go unrecognized.

Complications such as cataract, ocular hypertension, and glaucoma have been found mainly in the third decade of life (range from 5 – 40 years), with cataract as the predominant complication. This highlights the necessity for an early diagnosis of FUS in order to avoid long-term treatment with corticosteroids, which may not be helpful in FUS.

In previous publications, bilateral manifestation was found in 5-10 % of patients with FUS. This is in line with the results of our present study. The characteristic findings of FUS may be subtle, and FUS may be difficult to distinguish from other uveitis entities, e.g., JIA-associated disease. Here, ANA positivity and the early development of synechiae and glaucoma may help in differential diagnosis [27].

In conclusion, FUS may begin in early childhood, but diagnosis is often delayed for years, as the characteristic findings may not be present at disease onset. This may occasionally have the consequence of overtreatment with anti-inflammatory drugs.

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