Journal of Taibah University Medical Sciences (2013) 8(1), 24–30

Taibah University

Journal of Taibah University Medical Sciences

www.sciencedirect.com

Clinical Study

Clinical characteristics of Vogt–Koyanagi–Harada syndrome in a tertiary medical centre in western region of Kingdom of Saudi Arabia

Ahmed M. Bawazeer, FRCSC *, Shaik H.M. Nizamuddin, FRCS (Glasg), Lina H. Raffa, MD and Nawaf K. Marzouki, MD

Ophthalmology Deptartment, King Abdulaziz University, Jeddah, Kingdom of Saudi Arabia

Received 14 October 2012; revised 9 November 2012; accepted 5 December 2012

KEYWORDS

Saudi patients;
VKH;
Vogt–Koyanagi–Harada syndrome;
Uveitis;
Panuveitis

Abstract *Objective:* To characterize the clinical features of Vogt–Koyanagi–Harada (VKH) syndrome in a tertiary medical centre in western region of Kingdom of Saudi Arabia.

Method: Retrospective review of the records of patients who presented to the uveitis clinic between December 2001 and 2011.

Results: We found 48 patients of VKH, and enrolled 32 patients who had completed at least 6 months of follow up. The mean duration of follow up was 17.34 ± 13.45 months (range 6–60 months); 65.6% were female and 34.4% were male; and the mean age at disease onset was 33 ± 10.8 years (range 15–58 years). The majority (56.2%) had incomplete disease while 28.1% had complete disease and 15.6% probable disease. Extra ocular symptoms were common. We categorized the patients by presentation patterns. Those in the acute stage presented with panuveitis and bullous retinal detachment, while most of those in the convalescent stage presented with sunset glow fundus and nummular choreo-retinal scars. Auditory manifestations were seen in patients with chronic and recurrent stage, and cutaneous manifestations were seen in 53% of all patients. Cataract (53%) and glaucoma (9.3%) were the main complications. Acute-stage patients were treated with systemic steroids and those with chronic and recurrent disease with immunosuppressives. Over all, the visual prognosis was good, 62.5% of patients maintaining 20/40 or better visual acuity.

Conclusion: VKH is a common cause of uveitis in Kingdom of Saudi Arabia, often with extra-ocular symptoms. Patients can maintain good visual acuity when treated aggressively with systemic steroids in acute stage and immunosuppressive agents in chronic and recurrent stages.

* Corresponding address: Chairman & Associate Professor, King Abdulaziz University (KAU), Faculty of Medicine, P.O. Box 5095, Jeddah 21422, Kingdom of Saudi Arabia. Tel.: +966 505353015; fax: +966 22574294.
E-mail: drbawazeer@yahoo.com (A.M. Bawazeer)

Peer review under responsibility of Taibah University.

ELSEVIER Production and hosting by Elsevier

1658-3612
http://dx.doi.org/10.1016/j.jtumed.2013.01.001

Introduction

Vogt–Koyanagi–Harada (VKH) syndrome is a multisystem disease of presumed autoimmune etiology. It is characterized by chronic, bilateral, diffuse, granulomatous panuveitis with extra ocular manifestations affecting central nervous system (CNS) (neuro-otological symptoms like meningism, tinnitus and decreased hearing) and integumentary system (alopecia, poliosis and vitiligo).[1–4] VKH syndrome is reported to be commoner in darkly pigmented races, including Asian, Middle Eastern, Hispanic, and Native American populations.[3,5–9] In Kingdom of Saudi Arabia, VKH is common, accounting for 2.5–19.4% of all uveitis cases in various studies.[10–13]

VKH syndrome manifests in stages.[3,14–16] Initially, patients may present with neuro-otological symptoms, followed by ocular and integumentary involvement. After the acute stage, the treated patients may go into convalescent stage whereas untreated patients may develop chronic recurrent disease with vision-threatening complications. Integumentary manifestations appear weeks or months later. The syndrome presents with isolated ocular involvement (probable VKH) or with ocular, CNS and integumentary involvement (complete VKH) or ocular with either CNS or integumentary involvement (incomplete VKH), as determined by an International committee on nomenclature in 2001.[17]

A diagnosis of VKH syndrome is suggested by pathognomonic clinical findings.[3,14] Bilateral panuveitis along with exudative retinal detachment (RD) in the acute stage and sunset glow fundus in chronic stage are highly specific for VKH.[14] Ancillary tests like fundus fluorescein angiography (for multifocal leaks), ultrasonography (for choroidal thickening) and lumbar puncture (for cerebrospinal fluid pleocytosis) may be helpful.[3,4,14,15,18–20]

The standard initial therapy for VKH is prompt, aggressive use of systemic corticosteroids, to which the disease is especially responsive, particularly in the early stages.[19–21,23] Immuno-modulatory agents may be needed for non-responsive patients or when corticosteroid side-effects are not tolerated. Visual prognosis is generally good with prompt diagnosis and aggressive treatment.[22,23]

The aim of the study was to evaluate the clinical characteristics of VKH in a tertiary medical centre in western region of Kingdom of Saudi Arabia.

Materials and Methods

A retrospective review of records of all patients with uveitis who attended Magrabi Eye and Ear Hospitals and Centers in Jeddah, Kingdom of Saudi Arabia, between December 2001 and 2011 was carried out to collect the patients with the diagnosis of VKH. They were reviewed for demographic data such as age and gender, and for clinical features such as ocular and extra-ocular manifestations. Patients who completed a minimum of 6 months follow-up were included for the study. All cases were classified or reclassified according to the revised diagnostic criteria.[17] All patients who presented with acute uveitis had attended the clinic within 3 months of onset of symptoms, whereas those with chronic and recurrent disease had presented with symptoms of at least of 6 months duration. All patients underwent a complete ophthalmic work up including best corrected visual acuity, slit lamp examination, applanation tonometry and dilated indirect ophthalmoscopy, at the initial and final visits. Fundus fluorescein angiography and ultrasonography were performed in patients for whom the diagnosis was unclear. Indocyanine green angiography was not available at our centre. Lumbar puncture was not conducted in our patients as most refused to undergo the procedure. Ocular complications were noted. Corticosteroid treatment, immunosuppressive therapy and the length of follow-up were documented.

Results

Our retrospective review revealed 48 patients with VKH syndrome among 587 cases of uveitis between December 2001 and December 2011 and hence, 8.1% prevalence. Thirty two patients (64 eyes) had completed 6 months of follow-up and were included in the analysis. The mean duration of follow up was 17.34 ± 13.45 months (range 6–60 months). The demographic characteristics and extra-ocular manifestations were shown in Table 1. The majority (71.8%) were Saudi nationals and most (65.6%) of patients were females. The mean age at disease onset was 33 ± 10.8 years (range 15–58 years). All patients had bilateral ocular involvement.

The extra-ocular manifestations included neuro-otological symptoms (meningism, tinnitus and deafness) in 19 (59%) patients and cutaneous symptoms (alopecia, poliosis and vitiligo) in 17 (53%); nine patients had both integumentary and neuro-otological involvements. Five (15.6%) patients had no extra ocular manifestations.

According to the revised diagnostic criteria for VKH, 9 patients (28.1%) had complete disease, 18 (56.2%) had incomplete disease and 5 (15.6%) manifested probable disease.

The patients were classified into 3 groups according to the stage at which they were seen initially.

Nine patients presented to us in acute stage (Table 2), one with complete disease, three with probable disease and five with incomplete disease. All patients had panuveitis and most (77%) had exudative RD. One patient had bilateral cystoid macular edema, three developed cataracts and two developed macular scars. Only one patient presented with vitiligo. None had auditory symptoms. All these patients received topical and systemic corticosteroids and one received pulse steroid treatment. The mean follow up was 10.3 months, when 50% of patients had better than 20/40 vision.

Ten patients presented in convalescent stage (Table 3), one with complete disease, one with probable disease and eight with incomplete disease. Most showed resolving inflammation with nummular choreo-retinal scars and sun set glow fundus. Two patients had macular scars and two had cataracts; vitiligo was seen in three patients, alopecia in three and poliosis in two. None had auditory symptoms. All patients were on tapering doses of corticosteroids. Most had been followed up for more than 1 year (12.6 months) when 70% of eyes had better than 20/40 vision.

Thirteen patients presented to us in **chronic stage with recurrent inflammation** (Table 4), seven with complete disease, one with probable disease and five with incomplete disease. Eight patients had anterior uveitis, five had panuveitis. Six had exudative RD and two had focal exudative RD (FED). Three eyes

Table 1: Patient characteristics and extra ocular manifestations.

No.	Age	Nationality	Sex	CNS	Ear	Skin	Stage at presentation	Type
1	36	Saudi	M			V	Convalescent	Incomplete
2	58	Saudi	F				Acute	Probable
3	15	Saudi	F		DH	P	Chr, rec	Complete
4	21	Saudi	F	H&NP			Acute	Incomplete
5	33	Syrian	F				Convalescent	Probable
6	46	Saudi	F	H&NP	T	A	Chr, rec	Complete
7	36	Saudi	F	H&NP	T	V	Chr, rec	Complete
8	46	Saudi	M			A	Convalescent	Incomplete
9	25	Ethiopian	M	H& NP			Acute	Incomplete
10	15	Saudi	M	H&NP		A	Convalescent	Complete
11	19	Saudi	M	H& NP			Acute	Incomplete
12	43	Saudi	M	NP	DH	A	Chr, rec	Complete
13	17	Saudi	M	H&NP			Convalescent	Incomplete
14	40	Ethiopian	F				Acute	Probable
15	21	Saudi	M	H			Acute	Incomplete
16	25	Saudi	F			V	Chr, rec	Incomplete
17	19	Sudanese	F			P	Convalescent	Incomplete
18	30	Saudi	M				Acute	Probable
19	33	Syrian	F			P	Convalescent	Incomplete
20	34	Saudi	F				Chr, rec	Probable
21	15	Yamani	F	H& NP	DH	A	Chr, rec	Complete
22	15	Saudi	F	H&NP			Convalescent	Incomplete
23	17	Saudi	F			A	Chr, rec	Incomplete
24	26	Saudi	F	H			Chr, rec	Incomplete
25	32	Palestenian	F		T	A	Chr, rec	Complete
26	25	Saudi	F	H&NP		A	Chr, rec	Complete
27	24	Saudi	F	H		V	Acute	Complete
28	23	Yamani	F			V,A	Convalescent	Incomplete
29	36	Saudi	F			V	Convalescent	Incomplete
30	19	Saudi	M	H&NP	T		Chr, rec	Incomplete
31	20	Indonesian	F	H&NP			Chr, rec	Incomplete
32	25	Saudi	M	H			Acute	Incomplete

M, male; F, female; H, headache; NP, neck pain; T, tinnitus; DH, decreased hearing; V, vitiligo; A, alopecia; P, poliosis.

of three patients had choroidal neovascular membrane (CNVM) at presentation. Two of these patients with active CNVM received intravitreal injection of Bevacizumab (Avastine, Genentec), while the other patient had a scarred inactive membrane. Three patients had bilateral disc edema and three developed bilateral cataract. One patient had poliosis, six had alopecia and two vitiligo; three patients presented with decreased hearing and four with tinnitus. Imuran (Azathioprine) was the most commonly (11/13, 84.6%) used agent; followed by cyclosporine in two patients and methotrexate in one. One patient received imuran and cyclosporine. All patients were on immunosuppressive treatment in addition to systemic steroids. Patients with anterior segment inflammation were also treated with topical corticosteroids. Follow up of these patients ranged from 6 months to 5 years with a mean follow up was 25.3 months when 69.2% of eyes had better than 20/40 visual acuity.

At final follow up, cataract was found in 53% of eyes, glaucoma in 9.3%, macular scar 10.9% and CNVM in 4.6% (Table 5). At presentation, 20/64 eyes (31%) had a best-corrected visual acuity (BCVA) of $\geqslant$20/40, whereas 19/64 eyes (29.6%) had poor BCVA ($\leqslant$20/200). At the last visit, a BCVA of $\geqslant$20/40 was found in 40/64 eyes (62.5%) and $\leqslant$20/200 in only 13/64 eyes (20.3%).

Discussion

In this study, VKH constituted 8.1% of all cases of uveitis presenting to our clinics. We found previously that it is the second most common cause of identifiable uveitic entity after Behcet's disease.[11] Hamade et al. reported a similar prevalence (8%), [12] while other studies in Kingdom of Saudi Arabia reported a prevalence of 2.5% and 19.4%.[10,13]

In our study, Saudi nationals and Arabs predominated with two patients from Ethiopia. Although it has been reported that VKH is common in darkly pigmented people, it is rare in sub-Saharan Africa.[5,16] Most studies suggest a female predominance among patients with VKH disease,[5–14] as in our study. Most studies report a mean age at presentation of 35–45 years of age.[1,5–9] while those in our series presented at a mean age of 33 years.

At presentation, 28.1% had complete disease, 56.2% had incomplete disease and 15.6% probable disease, similar to the findings of a Japanese study (11.8% were classified as complete, 71% incomplete, and 8.9% as probable disease).[24]

Extra-ocular symptoms were common in our patients, 50% presenting with headache and 40% with neck pain. Rao et al.[14] found that 49% of their patients had headache and 36% neck stiffness. Auditory manifestations were noted in 21% of our

Table 2: The patients presented in acute stage.

Pt No.	Age	Eye	VA 1	Clinical findings at presentation	VA 2	Follow up	Complications
2	58	OD	20/30	PU	20/30	1 year	Macular scar
		OS	CF	PU, exudative RD, sub macular fibrosis	CF		
4	21	OD	20/50	PU, CME, FED	20/100	6 months	
		OS	20/60	PU, CME, FED	20/100		
9	25	OD	CF	PU	20/400	6 months	Cataract
		OS	HM	PU, exdative RD, sub macular inflammation	HM		Macular scar
11	19	OD	20/25	PU, exudative RD	20/200	1 year	Cataract
		OS	20/25	PU, exudative RD	20/200		Cataract
14	40	OD	20/25	PU, FED	20/25	6 months	Cataract
		OS	20/80	PU, exudative RD	20/80		Cataract
15	21	OD	20/100	PU, exudative RD	20/20	1 year	
		OS	20/60	PU	20/20		
18	30	OD	CF	PU, exudative RD	20/400	6 months	Cataract
		OS	CF	PU, exudative RD	CF		Macular scar
27	24	OD	20/200	PU, exudative RD	20/30	2 years	Glaucoma
		OS	20/200	PU, exudative RD	20/30		Glaucoma
32	25	OD	20/200	PU, exudative RD	20/30	9 months	Cataract
		OS	20/100	PU, exudative RD	20/30		Cataract

VA, visual acuity; OD, right eye; OS, left eye; CF, counting fingers; HM, hand motions; RD, retinal detachment; PU, panuveitis; CME, cystoid macular edema; FED, focal exudative retinal detachment.
Patient No. 9 received I.V. methyl prednisolone.

Table 3: The patients presented in convalescent stage.

Pt No	Age	Eye	VA 1	Clinical findings at presentation	VA 2	Follow up	Complication
1	36	OD	20/30	NCR, FED	20/30	1 year	Glaucoma, cataract
		OS	20/40		20/40		Glaucoma, cataract
5	33	OD	20/20	NCR, sunset glow fundus	20/20	1 year	
		OS	20/20	NCR, sunset glow fundus	20/20		
8	46	OD	20/50	NCR, FED	CF	1 year	Macular scar
		OS	20/60	NCR, FED	20/30		Cataract
10	15	OD	20/20	NCR, FED, disc edema	20/20	1 year	
		OS	20/20	NCR, FED, disc edema	20/20		
13	17	OD	20/20	Macular scar	20/20	1 year	Macular scar
		OS	CF		CF		
17	19	OD	20/50	Cataract, exudative RD	CF	1 year	Macular scar
		OS	20/60		20/20		
19	33	OD	20/20	Sunset glow fundus	20/20	6 month	
		OS	20/20	Sunset glow fundus	20/20		
22	13	OD	20/100	Iritis	20/80	1 year	Cataract
		OS	HM	Macular scar	20/400		Macular scar
28	24	OD	20/40	NCR, sunset glow fundus	20/30	2 years	Cataract
		OS	20/60	NCR, sunset glow fundus	20/30		Cataract
29	36	OD	20/80	NCR, sunset glow fundus	20/80	1 year	Cataract
			20/30	NCR, sunset glow fundus	20/30		Cataract

VA, visual acuity; OD, right eye; OS, left eye; CF, counting fingers; HM, hand motions; RD, retinal detachment; NCR scars, nummular choreo retinal scars.
All these patients received tapering doses of systemic steroids for 6 months.

Table 4: The patients presented with chronic recurrent inflammation.

Pt No.	Age	Eye	VA 1	Clinical findings	VA 2	Treatment	Follow up	Complication
3	15	OD	20/50	Iritis 1+	20/50	Cyclosporine	1 year	Cataract
		OS	20/400	Scarred CNVM	20/400			Scarred CNVM
6	46	OD	20/50	Iritis + 4, CME, FED, NCR	20/40	Imuran	6 monyh	Cataract
		OS	20/50	Iritis + 4, CME, FED, NCR	20/50			Cataract
7	36	OD	20/40	Disc edema, FED	20/25	Imuran	6 month	Cataract
		OS	20/30	Disc edema, FED	20/30			Cataract
12	43	OD	CF	Iritis + 4, exudative RD	20/60	Methotrexate	1 year	Cataract
		OS	CF	Iritis + 4, exudative RD	20/30			Cataract
16	25	OD	20/50	Iritis + 4, vitriis, NCR	20/20	Imuran	2 years	
		OS	20/60		20/20			
20	34	OD	CF	Iritis 1+, cataract	HM	Imuran	6	Glaucoma, optic atrophy
		OS	CF	Iritis 1+, cataract	CF			Glaucoma, optic atrophy
21	13	OD	20/100	Irititis 1+, FED, NCR	20/25	Imuran	4 years	Cataract
		OS	20/200		20/25			Cataract
23	17	OD	20/40	Iritis + 4, vitriis, exudative RD	20/20	Imuran	3 years	
		OS	20/40		20/20			
24	26	OD	20/60	Iritis 1+, DF nodules, NCR	20/20	Imuran	2 years	
		OS	20/100		20/20			
25	32	OD	20/30	Iritis + 3, vitritis2+	20/50	Imuran, cyclosporine	3 years	Cataract
		OS	CF	Iritis + 4, vitritis1+, disc edema, CNVM	20/30	Avastine		CNVM
26	26	OD	20/100	Exudative RD	20/30	Immuran	5 years	Cataract
		OS	20/400	CNVM	20/400	Avastine		CNVM
30	19	OD	HM	Exudative RD	20/40	Imuran	18 month	Cataract OU
		OS	CF		20/30			
31	20	OD	20/400	Exudative RD	20/30	Imuran	42 month	Cataract OU
		OS	20/400		20/30			

VA, visual acuity; OD, right eye; OS, left eye; CF, counting fingers; HM, hand motions; RD, retinal detachment; OU both eyes; CME, cystoid macular edema; NCR scars, nummular choreo retinal scars; DF nodules, Dalen Fuch's nodule; FED, focal exudative retinal detachment; CNVM, choroidal neovascular membrane.

Table 5: Improvement of visual acuity according to the stage of disease at presentation.

Stage(No. of eyes)	Pre/post treatment	⩾20/40	20/40–20/200	⩽20/200
Acute (18 eyes)	Pre-treatment	4 (22%)	9 (50%)	5 (27%)
	Post-treatment	8 (44%)	5 (27%)	5 (27%)
Convalescent (20 eyes)	Pre-treatment	11 (55%)	7 (35%)	2 (1%)
	Post-treatment	14 (70%)	2 (1%)	4 (205)
Chronic/recurrent (26 eyes)	Pre-treatment	5 (19.2%)	9 (34.6%)	12 (46.1%)
	Post-treatment	18 (69.2%)	4 (15.3%)	4 (15.3%)
Total (64 eyes)	Pre-treatment	20 (31.2%)	25 (39%)	19 (29.6%)
	Post-treatment	40 (62.5%)	11 (17.1%)	13 (20.3%)

cases; Kitamura et al. found them in 27.2%.[24] In our study 12.5% had tinnitus and 9.3% had decreased hearing while Rao et al. found rates of 36% with tinnitus and 32% decreased hearing. Late integumentary signs developed in 53% of our patients which is higher than the Japanese study (17.2%).[24] This may be because the majority (40%) of our patients presented in chronic recurrent stage when the integumentary signs usually develop. In our study, 9.3% presented with poliosis, 15.6% with vitiligo and 25% alopecia. Rao et al. reported 28% patients with poliosis, 20% with vitiligo and 18% alopecia. None of our patients had sugiura's sign, which has been observed mainly in Japanese patients. Rao et al. noted 5% patients (all from Japan) with sugiura's sign in their multi-ethnic study.[14]

As patients present in the acute, the convalescent or the chronic-recurrent stage, the approach and management of

each group differ. All of our patients in the acute stage presented with signs and symptoms of acute panuveitis and 77% with exudative RD. Most clinicians consider that treatment for at least 6 months is required to prevent recurrences and complications of chronic inflammation.[3,15,20,21,23] Rao et al. found that 50% of their patients presented with bullous RD. In both our series and that of Rao et al., one patient presented with vitiligo in the acute stage. We found no patient with auditory symptoms in this stage whereas; Rao et al. reported that 15% of their patients had hearing loss in the acute stage.[14]

Of the patients who presented in the convalescent stage, seven had cutaneous symptoms (vitiligo in three, alopecia in three and poliosis in two. None had auditory symptoms.

Of the patients who presented in chronic stage, 69% had cutaneous symptoms, 53% had auditory symptoms. Rao et al. reported vitiligo in 22% of their patients in chronic stage, alopecia in 19% and poliosis in 31%, while 18% presented with decreased hearing.[14]

Rao et al. reported that 75% of their patients in chronic stage had sunset fundus and nummular choreo-retinal scars while we found a prevalence of 80%.

Consistent with the results presented elsewhere,[3,6–9,14] the most frequent complication was cataract (53%). Other complications included glaucoma (9.3%), macular scarring (10/9%) and CNVM (4.6%). Rao et al. reported that 37% of their patients developed cataract, 10% glaucoma and 2% CNVM.

The incidence of neuro-otological symptoms has been reported to be related to ethnicity.[16] Deafness and meningism are more prevalent in Japanese while tinnitus appears to occur uniformly except among Hispanics who are least affected by otological symptoms.[9] In our patients all the otological symptoms were seen in chronic and recurrent stage and none in patients in acute and convalescent stages.

In our series, the overall visual prognosis was good with 40/64 eyes (62.5%) maintaining a best corrected visual acuity of 20/40 or better, however, 13/64 eyes (20.3%) had poor visual outcome.

Our study suffers from the drawbacks inherent to a retrospective study; however, we tried to describe the clinical features of our small number of patients with VKH in a rational way. A multicentre study involving the majority of uveitis centers in Kingdom of Saudi Arabia could address the prevalence of extra ocular symptoms in patients with VKH syndrome.

Conclusions

Vogt–Koyanagi–Harada syndrome is a common cause of uveitis in Kingdom of Saudi Arabia, frequently associated with extra-ocular manifestations. Otological symptoms appear late. Cataract and glaucoma are the major complications. Patients can maintain good visual acuity if they are treated aggressively with systemic steroids in acute stage and with immunosuppressive agents in chronic recurrent stages.

Authors' contributions

Drs. Ahmed M. Bawazeer, Shaik H.M. Nizamuddin, Lina H. Raffa and Nawaf K. Marzouki contributed in the design, preparation and correction of this manuscript in decreasing order of importance.

Acknowledgement

We thank Dr. May Mulham Al Baroudi, (Ophthalmology resident, King Abdulaziz University Hospital, Jeddah) for her help in editing the manuscript.

References

1. Brockhurst RJ, Jakobiec FA. Uveal tract. In: Alberts DM, Jakobiec FA, editors. **Principles and practice of ophthalmology**, vol. 1. Philadelphia, PA: WB Saunders; 1994.
2. Nussenblatt RB, Whitcup SM, Palestine AG. *Uveitis: fundamentals and clinical practice*. 3rd ed. Philadelphia, PA: Mosby; 2004, p. 58–68.
3. American Academy of Ophthalmology. Vogt–Koyanagi–Harada syndrome. In: Intraocular inflammation and Uveitis. Basic and clinical course 2010, Section 9. p. 209–214.
4. Foster CS, Vitale AT. *Diagnosis and treatment of uveitis*. Philadelphia, PA: WB Saunders; 2002.
5. Chang JH, Wakefield D. Uveitis: a global perspective. **Ocul Immunol Inflamm 2002**; 10(4): 263–279.
6. Yang P, Zhang Z, Zhou H, et al. Clinical patterns and characteristics of uveitis in a tertiary center for uveitis in China. **Curr Eye Res 2005**; 30: 943–948.
7. Singh R, Gupta V, Gupta A. Pattern of uveitis in a referral eye clinic in north India. **Indian J Ophthalmol 2004**; 52: 121–125.
8. Wakabayashi T, Morimura Y, Miyamoto Y, Okada AA. Changing patterns of intraocular inflammatory disease in Japan. **Ocul Immunol Inflamm 2003**; 11: 277–286.
9. Sukavatcharin Somsiri, Tsai Julie H, Rao NA. Vogt–Koyanagi–Harada disease in Hispanic patients. **Int Ophthalmol 2007**; 27: 143–148.
10. Islam SM, Tabbara KF. Causes of uveitis at the Eye Centre in Saudi Arabia: a retrospective review. **Ophthalmic Epidemiol 2002**; 9(4): 239–249.
11. Bawazeer AM, Nizamuddin SHM. Referral pattern of uveitis in a tertiary centre in western region of Saudi Arabia. In: Proceedings of American Academy Ophthalmology Conference, San Francisco, CA, October 24–27, 2009.
12. Hamade IH, Elkum N, Tabbara KF. Causes of uveitis at a referral center in Saudi Arabia. **Ocul Immunol Inflamm 2009**; 17(1): 11–16.
13. Al-Mezaine HS, Kangave D, Abu El-Asrar AM. Patterns of uveitis in patients admitted to a University Hospital in Riyadh, Saudi Arabia. **Ocul Immunol Inflamm 2010**; 18(6): 424–431.
14. Rao NA, Gupta A, Dustin L, et al. Frequency of distinguishing clinical features in Vogt–Koyanagi–Harada disease. **Ophthalmology 2010**; 117(3): 591–599.
15. Smith RE, Nozik RA. *Uveitis, a clinical approach to diagnosis and management*. Baltimore, MD: Williams & Wilkins; 1989.
16. Allegri P, Rissotto R, Herbort CP, Murialdo U. CNS diseases and uveitis. **J Ophthalmic Vis Res 2011**; 6(4): 284–308.
17. Read RW, Holland GN, Rao NA, et al. Revised diagnostic criteria for Vogt–Koyanagi–Harada disease: report of an international committee on nomenclature. **Am J Ophthalmol 2001**; 131: 647–652.
18. Fardeau C, Tran TH, Gharbi B, et al. Retinal fluorescein and indocyanine green angiography and optical coherence tomography in successive stages of Vogt–Koyanagi–Harada disease. **Int Ophthalmol 2007**; 27(2–3): 163–172.
19. Miyanaga M, Kawaguchi T, Shimizu K, et al. Influence of early cerebrospinal fluid-guided diagnosis and early high-dose corticosteroid therapy on ocular outcomes of Vogt–Koyanagi–Harada disease. **Int Ophthalmol 2007**; 27(2–3): 183–188.

20. Touitou V, Escande C, Bodaghi B, et al. Diagnostic and therapeutic management of Vogt–Koyanagi–Harada syndrome. **J Fr Ophtalmol 2005**; 28(1): 9–16.
21. Bykhovskaya I, Thorne JE, Kempen JH, et al. Vogt–Koyanagi–Harada syndrome: clinical outcomes. **Am J Ophthalmol 2005**; 140: 674–678.
22. Errera MH, Fardeau C, Cohen D, Navarro A, Gaudric A, Bodaghi B, Westcott M, LeHoang P. Effect of the duration of immunomodulatory therapy on the clinical features of recurrent episodes in Vogt–Koyanagi–Harada disease. **Acta Ophthalmol 2011**; 89(4): e357–e366.
23. Bordaberry MF. Vogt–Koyanagi–Harada disease: diagnosis and treatments update. **Curr Opin Ophthalmol 2010**; 21(6): 430–435.
24. Kitamura M, Takami K, et al. Comparative study of two sets of criteria for the diagnosis of Vogt–Koyanagi–Harada's disease. **Am J Ophthalmol 2005**; 139(6): 1080–1085.