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Corticosteroid-Induced Central Serous Chorioretinopathy

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Abstract: Five patients were identified by medical records and fluorescein angiography as having developed central serous chorioretinopathy (CSC) during corticosteroid treatment. These five and 28 previously reported corticosteroid-induced CSC occurrences were studied to clarify the differences between idiopathic CSC and corticosteroid-induced CSC. Nine previously reported occurrences of corticosteroid-induced multiple posterior pigment epitheliopathy (MPPE) were also reviewed. Corticosteroid-induced CSC patients were older and less male-dominant; in MPPE, female patients predominated and most had bilateral involvement. The onset of CSC was within 70 days of corticosteroid administration in the short latency group, and more than 6 months after administration in the prolonged latency group. Daily doses of prednisolone usually exceeded 20 mg in the short latency group and was less than 20 mg in the prolonged latency group. Immunosuppressive agents such as cyclophosphamide were related to a lower daily dose at onset. **Jpn J Ophthalmol 1997;41:180–185** © 1997 Japanese Ophthalmological Society

Key Words: Central serous chorioretinopathy, corticosteroid, multifocal posterior pigment, epitheliopathy, prednisolone, side effect.

Introduction

In 1980, Wakakura and Ishikawa[1] described five occurrences of systemic corticosteroid-induced central serous chorioretinopathy (CSC); this report also described six of nine CSC patients who had received systemic corticosteroids and developed further visual acuity loss and serous detachment during the treatment. One patient developed CSC during each of three separate courses of corticosteroid therapy for retrobulbar optic neuritis.[2] Nineteen Japanese patients with CSC[3–10] and nine with a severe form of CSC, multiple posterior pigment epithelio-pathy (MPPE),[11] have also been observed during systemic corticosteroid treatment (for various conditions).[12–19] Gass and Little[20] showed systemic corti-costeroid treatment to be the probable cause of the severe exacerbation of retinal detachment and persistent visual loss in some patients with idiopathic CSC.

In the present study, we examined five instances of CSC diagnosed during systemic corticosteroid treatment, in addition to 28 previously reported cases, in order to clarify the characteristics and pathogenesis of this disease which has, until this time, been described almost entirely in the Japanese literature.

Patients and Methods

Three hundred sixty-five occurrences of CSC identified by fluorescein angiography (FAG) at our hospital from April 1980 to March 1996 were reviewed. There were 12 that developed during corticosteroid therapy; in five of these, it was possible to obtain detailed clinical information. Clinical information was also examined in all occurrences of CSC or MPPE reported as a side effect of corticosteroid administration.

Case 1

A 34-year-old man complaining of blurred vision in the left eye for 2 days was first seen at our ophthalmology department on September 2, 1980. He had been treated for nephrotic syndrome originating from membrane nephropathy for 2 months with oral prednisolone (50 mg/day, which was reduced after 5 weeks). At the time of the visit to our clinic, the dos-

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age was 30 mg/day. Corrected visual acuity was 1.2 OD and 0.8 OS. Goldmann perimetry showed central scotoma in the left eye. Intraocular pressures (IOP) were slightly elevated: 23 mm Hg (right) and 20 mm Hg (left). There were no cataracts. Fundus ophthalmoscopy revealed CSC with a 3 disc diameter detachment at the left macular area; FAG identified two leakage sites. On September 18, the leakage was treated with laser photocoagulation. At that time, bilateral IOP was 17 mm Hg. Prednisolone (PSL) was reduced to 20 mg/day on September 20. Reduction of the serous detachment was seen on September 24. PSL was continued unchanged; the serous detachment was no longer evident on November 30 and left visual acuity had improved to 1.0.

Case 2

A 46-year-old woman, first seen in our clinic on June 9, 1982 complaining of vision loss in the left eye for 2 weeks, had been receiving PSL 40 mg/day from May 5 to May 25, 1982 for protein-losing gastroenteropathy. Corrected visual acuities were 1.0 OD and 0.5 OS; fundus examination found CSC with a 3 disc diameter detachment in the left macular area. FAG revealed leakage, but laser therapy was not advised because the site was close to the foveola. The PSL dosage was immediately reduced, and by June 30 the detachment had almost completely disappeared on a daily PSL dosage of 9 mg. Left visual acuity was 0.8.

Case 3

A 34-year-old man had been taking mizorbine (50 mg/day; increased to 100 mg/day), PSL (20 mg/day), and cyclophosphamide (25 mg/day) for treatment of nephrotic syndrome since May 25, 1984. At his initial visit to our clinic on January 17, 1985, there was a central scotoma in the left eye. Corrected visual acuities were 1.0 OD and 0.6 OS. Fundus examination found CSC with a 2 disc diameter detachment in the left macular area. FAG found leakage near the foveola. On March 25, the cyclophosphamide was discontinued. The central serous detachment persisted for several months but the left visual acuity improved to 1.0 by June. On November 1, slit-lamp biomicroscopy revealed that the serous detachment had cleared but some metamorphopsia remained.

Case 4

A 46-year-old man was referred to our department for assessment of IOP and a fundus examination prior to methylprednisolone [pulse therapy]. The ocular examination was normal and methylprednisolone [pulse therapy] of 1000 mg/day for 3 days was begun on September 28. This was followed by a tapered administration of oral PSL beginning at 30 mg/day on October 1; reduced to 25 mg/day on November 24; to 20 mg/day on January 19, 1988, and to 15 mg/day on March 15. Blurred right vision was noted on May 20, 1988 and the patient visited our clinic on June 27. At that time visual acuities were 0.7 OD and 1.2 OS; serous retinal detachment with a depigmented area was seen in the right macular area. FAG indicated leakage from the supero-temporal portion of the macula and a lesion of the retinal pigment epithelial layer was identified around the leakage site (Figure 1). The detachment enlarged and was treated by laser photocoagulation on July 27. On September 5, visual acuity in the right eye had improved to 1.2 and there was no retinal detachment.

Case 5

Malignant lymphoma (stage IIA) was confirmed in a 42-year-old man by cervical node biopsy on November 31, 1994. The nodule was irradiated (30 Gy) in January 1995 followed by an intravenous chemotherapy regimen of cyclophosphamide 800 mg/day, doxorubicin 60 mg/day, and vincristine 2 mg/day for 10 days each month from March to June. Daily oral PSL (80 mg) was given during the chemotherapy. Central scotoma and metamorphopsia were seen in the right eye on April 19, 1996, the ninth day after the second chemotherapy administration. Ophthal-

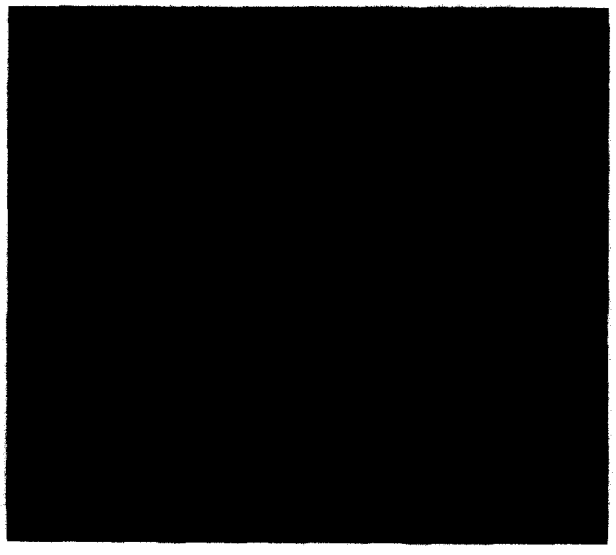

Figure 1. Fluorescein angiogram: right eye, case 4. Note leakage and granular background lesions at the supero-temporal portion of macula.

Jpn J Ophthalmol
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mological examination 2 days later found bilateral corrected visual acuities of 1.2; fundus examination found CSC with 1 disc diameter detachment in the right macular area. FAG revealed dye leakage (Figure 2). Kallidinogenase (150 IU/day) was administered without laser treatment. Chemotherapy and PSL were discontinued on June 6. The retinal detachment disappeared gradually and was no longer present on July 21.

Results

Patient data for corticosteroid-induced CSC are found in Table 1; for MPPE, in Table 2. Age differences between the idiopathic CSC patients (disease unrelated to corticosteroids) and the corticosteroid-induced CSC patients were apparent. There were 353 idiopathic cases reviewed (297 men, 56 women). The age at onset in idiopathic CSC ranged from 19 to 54 years (average, 36.8). The average age at onset for men was 36.4; for women, 38.9. In the corticosteroid-induced CSC, the age at onset ranged from 27 to 62 years (average 42.6). The average age at onset was 41.9 for men and 44.3 for women. Men predominate in both idiopathic (5.3:1) and corticosteroid-induced (2.3:1) CSC.

For MPPE, which is clinically indistinguishable from a severe form of CSC, ages ranged from 31 to 64 (average, 49.0). Unlike the idiopathic or corticosteroid-induced CSC, females predominated in MPPE 2.3:1.

Corticosteroid therapy was administered for a variety of systemic disease conditions such as nephropathy (15 patients, including 7 postrenal transplant); nephrotic syndrome (five patients) and ocular disease (14 including 6 with optic neuritis). This treatment is often required for elderly female patients with nephropathy or malignancy, and for patients with collagen disease.

Figure 2. Fluorescein angiogram: right eye, case 5. Dye leakage is essentially the same as in idiopathic central serous chorioretinopathy.

The total dosage of PSL at CSC onset ranged from 140 to 52 800 mg. There was no clear dose-dependency between CSC incidence and total dosage. Daily dosage at onset ranged from 10 to 100 mg. We believe this indicates a critical daily dosage of 15–20 mg. In patients with leakage treated by laser photocoagulation, there were no recurrences even with continued administration of PSL at daily doses exceeding 15 mg as is cases 7, 25, 32, 35, and 38.

In the CSC patients, there was a prolonged latency of more than 6 months in patients 3, 4, 18, 19, and 27. All other CSC patients, except 21 and 22, had shorter latency periods, with onset within 70 days, perhaps because of greater sensitivity to corticoste-roids. Patients 1 and 29, both in the short latency group, also had corticosteroid glaucoma. The three patients with intermediate latency periods (21, 22) were unusual with onsets between 71 and 179 days. Daily PSL dosage at onset in the short latency group was greater than 20 mg. In the prolonged latency group, including the postrenal transplant patients (19 and 27) the daily PSL at onset was less than 20 mg.

While age and sex distributions varied, clinical features of idiopathic and corticosteroid-induced CSC, including visual acuity at onset and at final examination, visual field, and fluorescein angiography, did not.

Discussion

Twelve of the 365 (3.3%) CSC occurrences reviewed in this survey developed in conjunction with corticosteroid therapy. While the usual age range for idiopathic CSC is 20–50 years, as also shown in the present results, and for MPPE, 30–50 years, with men predominating,[11,21–23] the corticosteroid-induced disease was found in an older, largely female, group.

Quillen et al[24] reviewed 61 women with CSC and found a later age at onset than in men; in 13 of these (25.5%), it occurred during corticosteroid administration. Our present study suggests that the daily dosage is a greater influence in onset of CSC than total dosage: the daily dose was greater than 15 mg, confirming our previous observations.[1,2,9] Exceptions included postrenal transplant patients (19, 24, 27) possibly related to an imbalance of the systemic

Table 1. Corticosteroid-Induced CSC

Case	Age/Sex	Diagnosis	Affected Eye	History of CSC	Total Dosage at Onset (mg)	Corticosteroid Dose (mg/day)[a]				Corticosteroid	Treatment for CSC	References
						Initial	At Onset	Latency (Day)	At Recovery			
1	34/M	nephrotic syndrome	OS	no	1616	50	30	42	20	PSL	PC	[d]
2	46/F	protein-losing gastroenteropathy	OS	no	940	40	30	21	9	PSL	quick tapering	[d]
3	34/M	nephrotic syndrome	OS	no	4640	20	20	232	20	PSL	observation	[d]
4	46/M	membrane nephropathy	OD	no	7875	1250	15	241	15	MPSL, PSL	PC	[d]
5	42/M	malignant lymphoma	OD	no	3200	80	80	40	0	PSL	PC	[d]
6	48/F	erythema nodosum	OD	yes	150	30	30	5	?	PSL	quick tapering	1
7	37/M	rheumatic fever & carditis	OS	no	360	40	40	9	?	PSL	PC	1
8	45/F	breast cancer	OD	yes	160	20	20	8	?	PSL	quick tapering	1
9	38/M	optic neuritis	OD	yes	1360	100	80	14	?	PSL	quick tapering	1
10	39/M	optic neuritis	OD	no	760	20	80	10	?	PSL	PC	1
11	34/F	retinochoroiditis	OD	no	210	30	30	7	0	PSL	termination	9
12	38/M	optic neuritis	OD	no	140	40	30	4	0	PSL	PC	9
13	40/M	Vogt-Koyanagi-Harada syndrome	OS	no	1005	120	20	18	20	BETA	PC	9
14	53/M	idiopathic thrombocytopenic purpura	OU	yes	180	60	60	3	0	PSL	quick tapering	7
15	37/F	chronic glomerulonephritis	OD	no	1590	40	35	51	0	PSL	termination	7
16	59/F	sarcoidosis	OU	yes (OS), PED (OD)	5270	40	50	6	?	PSL	observation	7
17	55/M	SLE	OS	no	2100	40	60	44	?	PSL	PC	7
18	44/F	rheumatic arthritis	OS	PED	?	?	10	3650	10	PSL	PC	7
19	28/M	postrenal transplant	OD	no	21500	120	12.5	2190	?	PSL	PC	7
20	51/M	Fisher syndrome	OU	yes	3650	1000	50	20	0	PSL	PC	10
21	48/M	drug-induced enteropathy	OD	no	3990	40	30	97	10/20[b]	PSL	PC/observation[b]	4
22	33/F	nephrotic syndrome	OD	no	4650	60	30	100	20	PSL	PC	4
23	62/M	bilateral papillitis	OS	PED	320	60	40	6	10	PSL	quick tapering	8
24	45/F	postrenal transplant, SLE	OD	no	?	?	12.5	?	?	MPSL	observation	5
25	41/M	postrenal transplant	OU	no	?	1000	20	60	?	PSL	PC	5
26	44/M	postrenal transplant	OS	no	?	?	22	60	17.5	MPSL	quick tapering	5
27	52/F	postrenal transplant	OS	no	?	10	12.5	180	?	MPSL	observation	5
28	43/M	malignant melanoma	OU	yes (OS)	500	50	50	9	0	PSL	observation	6
29	35/M	Vogt-Koyanagi-Harada syndrome	OU	no	240	60	60	4	?	DEXA	quick tapering	3
30	39/M	optic neuritis	OD	yes	520	100	80	6	<20	BETA	quick tapering	2
31	44/M	optic neuritis	OS	yes	?	100	40	12	<20	BETA	quick tapering	2
32	27/M	retinal vasculitis	OU	no	52800	100	80	67	80	PSL	PC	20
33	45/M	left bullous detachment[c]	OD	no	6480	5	100	64	0	PSL	PC	20

PSL: prednisolone, BETA: betamethasone, DEXA: dexamethasone, MPSL: methyl-prednisolone, PED: pigment epithelial detachment, SLE: systemic lupus erythematosus.
[a]Dose of prednisolone.
[b]Two leakage sites were identified.
[c]Also treated by retrobulbar injection of MPSL.
[d]Present study.

Table 2. *Corticosteroid-Induced MPPE*

Case	Age/Sex	Diagnosis	Affected Eye	Corticosteroid Dose (mg/day)[a]		Latency (day)	Total Dosage at Onset (mg)	Corticosteroid	Treatment for MPPE	References
				Initial Stage	At Onset					
34	51/M	chronic nephritis	(1) OD[a], (2) OU	15	(1) 15, (2) 30	(1) 160, (2) 730	(1) 2460, (2) 7600	PSL	PC	15
35	64/F	nephrotic syndrom	OS	30	30	120	3600	PSL	PC	17
36	31/F	postrenal transplantation	OU	675	20	60	5350	PSL, MPSL	PC	12
37	38/M	chorioretinopathy (*LE*)	OD	40	40	120	4800	PSL	PC	16
38	49/F	multiple myositis	OU	60	30	144	6000	PSL, BETA	PC	14
39	54/F	SLE	OS	60	30	52	?	PSL	observation	13
38	54/F	SLE	OU	5	10	>10 yrs	?	?	PC	19
39	58/F	nephrotic syndrome	OU	?	?	?	?	PSL?	PC	16
40	41/M	postrenal transplantation	(1) OS[a], (2) OU	?	?	(1) 60, (2) 205	?	MPSL	(1) observation, (2) PC	18

PSL: prednisolone, BETA: betamethasone, DEXA: dexamethasone, MPSL: methyl-prednisolone, PED: *pigment epithelial detachment*, SLE: *systemic lupus erythematosus*.
[a] Initially diagnosed as CSC.

metabolism and the effects of immunosuppressive agents. We noted that in patient 3, termination of cyclophosphamide resulted in improvement despite continuation of PSL at 20 mg/day.

In Tables 1 and 2 we have detailed a large number of corticosteroid-induced CSC, but only a few of MPPE. Not all authors consider corticosteroids to be causative factors[25,26] although Miura et al[27] described eight MPPE cases that were markedly exacerbated during corticosteroid therapy and others report MPPE (eight cases) complicated by postrenal transplant therapy[16,18,24,28] as well as one instance of MPPE in a patient receiving hemodialysis.[25] Yumiyama et al[26] observed enlarged serous retinal detachment in one patient following increased PSL.

Autoimmune and renal diseases have been directly linked to CSC, although the patients described also received corticosteroids,[27,29–31] Wakakura has pointed out the apparent adverse effects of corticosteroids on such patients.[32] Bouzas et al[33] reported CSC in 3 of 50 patients with endogenous hypercortisolism. Wakakura et al[34] described significantly elevated urinary adrenalin, noradrenalin, and vanillylmandelic acid in untreated CSC patients and in non-CSC patients receiving corticosteroids.

There are a number of experimental reports that explore the as yet not fully understood mechanisms of corticosteroid effects on the retina or choroid, as well as the development of idiopathic CSC. Yoshioka et al[35] established an experimental animal model of CSC in which intravenous adrenalin is repeatedly administered to the Macaca irus. Stern and Ernest[36] noted experimental serous retinal detachment in a rhesus monkey following choriocapillaris occlusion.

The basic pathogenesis of CSC appears to be a circulatory change in the choroid.[37–39] The effects of corticosteroids on choroidal circulation have not been studied, but corticosteroids appear to promote blood coagulation.[40] Disruption of the outer blood–retinal barrier is one cause of serous detachment. Kishimoto et al[41] found that corticosteroids inhibit the healing of laser photocoagulation lesions of the retinal pigment epithelial layer.

If CSC is induced by corticosteroids, why then might only one eye be involved? Seven of the corticosteroid-induced CSC patients (21%; Table 1) and six of the MPPE patients (75%; Table 2) had bilateral involvement, a greater frequency than is found in idiopathic CSC. It is possible that corticosteroid administration is a predisposing factor, rather than a direct cause, in the disruption of the outer blood–retinal barrier. As in patient 4, the pigment epithelial lesion identified around the leakage site may be an

expression of local predisposition although this patient has no history of CSC. The eleven cases (33%) with a history of CSC or pigment epithelial detachment support this possibility.

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