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Ophthalmic zoster: mucous plaque keratitis

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SUMMARY Data taken from 1221 patients attending the Zoster Clinic of Moorfields Eye Hospital over the past 15 years were used to characterise the clinical appearance and behaviour of zoster mucous plaque keratitis (MPK). The typical greyish branching plaques are usually accompanied by a limbitis, stromal keratitis, or decrease in corneal sensation and are commonly associated with cataract, raised intraocular pressure, or corneal ulceration. MPK may begin at any time within two years of onset of the rash, but when it appears after three months there are more complications. Usually MPK settles within one month if appropriate treatment with topical steroids and acetylcysteine drops is given, but surgical intervention is sometimes required to control glaucoma or neuroparalytic keratitis or to remove cataracts. The results of surgery are surprisingly good.

Dendriform corneal epithelial disturbances have long been recognised in ophthalmic zoster but only relatively recently described in the literature.[1-3] The collective term 'pseudodendrite' precludes a satisfactory classification of these disturbances, gives no indication of the nature of the lesion, and is unhelpful in their management.

There are two distinct entities. The first, acute epithelial microdendrites, occurs a few days after the rash and resolves rapidly without complications. Viable virus is recoverable from the lesions.[1] The second, mucous plaque keratitis, by contrast has no clear temporal relationship to the rash and is a chronic disorder which is commonly associated with severe ocular sequelae such as glaucoma, cataract, and neuroparalytic ulcers.[4,5] Viable virus cannot be identified in the lesions.[4] The white-grey plaque which characterises the keratitis is adherent to the surface epithelium, has sharply demarcated margins, and may be linear or branched. There are usually several, which vary in size, shape, position, and number day by day, with no preferential corneal site. They stain sparingly with alcian blue, moderately with fluorescein, and brilliantly with rose Bengal (Fig. 1). They are deposited on a diffusely thickened and abnormal epithelium. Their onset varies from one week to two years after the rash. They are usually accompanied by a limbitis, stromal keratitis, diminished corneal sensation, or iritis and may be preceded by an episcleritis, disciform keratitis, or iritis. Debridement of the plaque leaves an intact but abnormal epithelium.

Mucous plaques also occur with filamentary keratitis, keratoconjunctivitis sicca, superior limbic keratitis, vernal keratitis, varicella keratitis,[6] and rarely with herpes simplex.[5] The aim of this study is to define the clinical behaviour of zoster mucous plaque keratitis, to emphasise the difference from herpes simplex keratitis, to plan logical management and to report that the complicating glaucoma, cataract, and neuroparalytic ulcers may be successfully treated surgically.

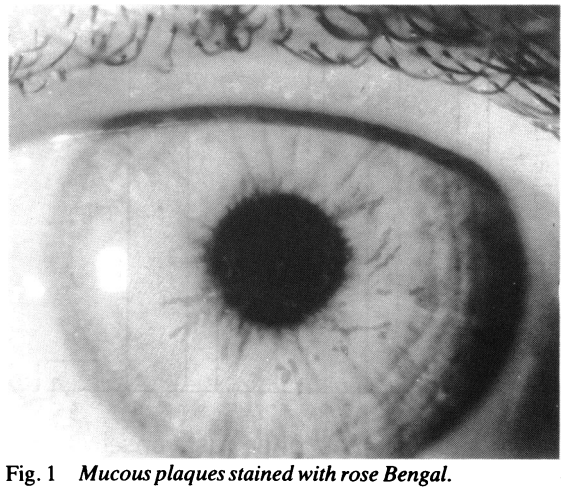

Fig. 1 *Mucous plaques stained with rose Bengal.*

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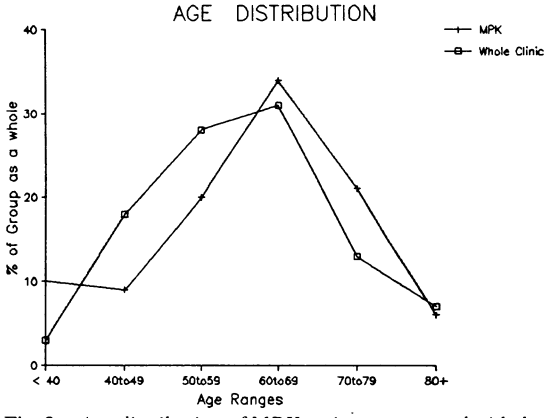

Fig. 2 *Age distribution of MPK patients compared with that of the whole clinic.*

Patients and methods

The data are derived from patients attending the Zoster Clinic at Moorfields Eye Hospital over the past 10 years, of whom the majority were primary referrals. Their follow-up has been regular and consistent over this period. Corneal sensitivity was measured with the aesthesiometer of Luneau and Coffignon in four peripheral sectors and centrally. The information was put on computer storage. 1030 patients (85%) had follow-up visits at least three-monthly over two years, and these form the study group. We compared the accompanying features of MPK with those of all ophthalmic zoster patients (with similar follow-up).

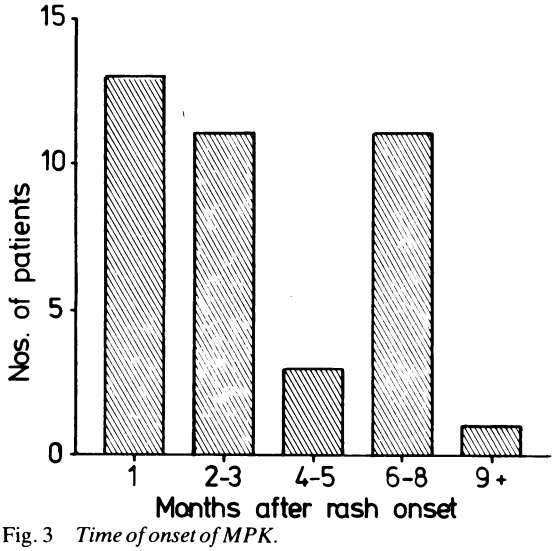

Fig. 3 *Time of onset of MPK.*

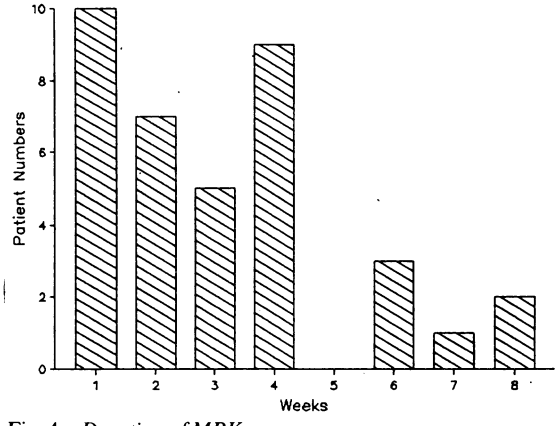

Fig. 4 *Duration of MPK.*

Results

Forty-seven (4%) cases of mucous plaque keratitis were found, of whom 39 had reliable follow-up data for two to 13 years (mean six years). Compared with the whole clinic population the patients with MPK were on average younger (Fig. 2). Fig. 3 shows the time of plaque onset in relation to the rash, the majority occurring within the first three or between six and seven months. Fig. 4 shows the duration of the plaque, which was usually less than one month.

Associated clinical features may be considered under the following headings: those preceding the onset of the plaque, those associated with it, and those subsequent to it.

There were no preceding features in 28%, but a complication distinguishing them from the rest of the clinic population was an increase in hypertensive iritis (Table 1; $p<0.05$ by χ^2 test).

MPK is associated with a combination of episcleritis (usually perilimbal), iritis, superficial corneal stromal infiltration, and a decrease in corneal sensation, though none of these taken individually is more

Table 1 *Incidence of associated clinical features occurring before and simultaneously with MPK. The incidence of these features in the clinic as a whole is shown in the last column*

Category	*Previous involvement*	*Coincident involvement*	*Overall clinic incidence*
Iritis	22 (56%)	20 (51%)	50%
Raised IOP	16 (41%)	9 (23%)	14%
Episcleritis	12 (31%)	22 (56%)	59%
Diminished corneal sensation	14 (36%)	14 (36%)	33%
Keratitis			
Microdendrite	9 (28%)	0	20%
Nummular	13 (33%)	15 (38%)	49%
Disciform	7 (18%)	4 (10%)	12%
Oedema	7 (18%)	?	5%

Table 2 *Incidence of clinical features following MPK*

Category	*Early plaque <3 months*	*Late plaque 3 months or more*	*Overall clinic incidence*
Corneal stromal haze	16 (67%)	15 (100%)	?
Ulcers	2 (8%)	4 (27%)	4%
Diminished sensation	6 (25%)	9 (60%)	33%
Disciform keratitis	6 (25%)	5 (33%)	12%
Lipid keratopathy	4 (17%)	2 (13%)	5%
Lens opacities	10 (42%)	12 (80%)	12%
Refractory glaucoma	1 (4%)	5 (33%)	3%
Recurrence	6 (25%)	5 (33%)	4%

common in mucous plaque keratitis than in the clinic population as a whole (Table 1).

The sequelae can be split into two groups: one with plaque onset within the first three months and the other after this time. In Table 2 they are compared with the incidence of eye complications in the clinic population as a whole.

Table 2 indicates a generally more severe outcome in plaques of later onset except with respect to lipid keratopathy, disciform reactions, and uveitis. There is a greatly increased tendency to recur, a very frequent occurrence of diffuse anterior stromal haze, diminished corneal sensation, neuroparalytic ulceration, glaucoma, and cataract ($p<0.05$ by χ^2 test). Only one case had a coincident herpes simplex infection. Two patients rapidly developed large interpalpebral ring-shaped subepithelial plaques with underlying stromal thinning (Fig. 5). These complications often led to visual loss (Table 3). Most of the morbidity was due to cataract and the remainder to various degrees of corneal scarring.

MANAGEMENT

The active keratitis was treated with topical steroid, the dosage being matched to the degree of inflammation (we graded episcleritis, keratitis, and iritis on a scale of 0–6). We started with dexamethasone 0·1% eyedrops four-hourly and, as the condition ameliorated, reduced the frequency over two months by degrees to twice daily. If control was maintained, the drop was changed to betamethasone, but any later recurrence necessitated returning to dexamethasone immediately. Over the next six months we tried to substitute prednisolone 0·3% eyedrops three times a day. Most cases subsided after this period but required a maintenance dose for long term use because there was a pronounced tendency for relapse even after two years, especially on reducing treatment or even on stopping prednislone drops 0·3% once daily. Acetylcysteine 10% eyedrops dissolved the plaques, and artificial tears helped maintain the precorneal tear film. Timolol eyedrops were successful in most cases in controlling raised intraocular pressure due to iritis and steroids. Topical iodoxuridine, adenine arabinoside, and trifluorothymidine made the epithelial problem worse, and acyclovir had no effect on this keratitis.

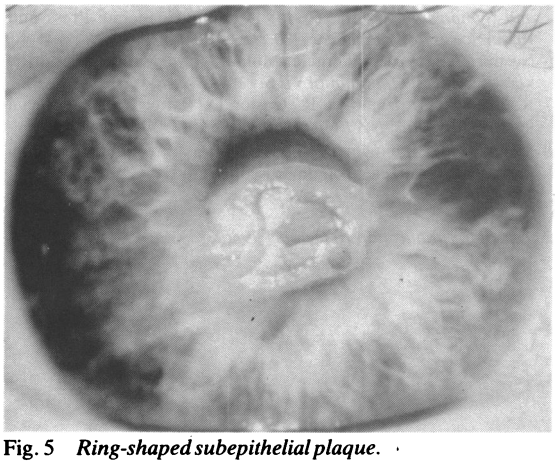

Fig. 5 *Ring-shaped subepithelial plaque.*

Table 3 *Numbers of patients with decreased Snellen visual acuity (lines)*

Lines lost	*No of patients*
0	11
1	5
2	7
3	3
4	3
5	10

Two patients developed severe cataract and glaucoma which required surgical treatment. Neuroparalytic ulcers developed from one month to three years after MPK onset and were always treated with a lateral third tarsorrhaphy. Table 4 depicts the number of cases requiring surgery. In the three cases requiring glaucoma surgery it was successful in controlling intraocular pressure without antiglaucoma therapy, and the acuities of those patients who also had intraocular lens implants were 6/9. All cases required a booster dose of topical steroid over three months postoperatively because of relapsing iritis. The tarsorrhaphies were followed by healing of corneal epithelial ulcers within three days.

Table 4 *Cases requiring surgery*

Operation	*No*
Combined trabeculectomy+extraction+implant	2
Trabeculectomy	1
Laser trabeculoplasty	1
Lateral third tarsorrhaphy	5

Discussion

These results indicate that mucous plaque keratitis associated with herpes zoster ophthalmicus has a distinct clinical appearance and evolution. MPK is not difficult to diagnose if an adequate history is obtained. Although the appearance is superficially similar to that of herpes simplex, detailed examination of the morphology and of the staining characteristics will distinguish them. In our experience it is very rare to have coincident zoster and simplex. If there is doubt, a two-day intensive course of topical steroids will usually settle the inflammatory component of mucous plaque keratitis and obviously aggravate simplex dendrites. MPK is a self-limiting condition, but, while the inflammation may soon settle, the epithelium may take much longer to recover, especially if there is loss of corneal sensation or a degree of exposure.

At least two factors are likely to lead to MPK: altered corneal epithelium and disturbance of tear film mucus.[7] Normal epithelium has mucous receptors primarily involved in the maintenance of the tear film. Alteration of these could reasonably lead to an accumulation of mucus, especially if the mucus derived from goblet cells is less soluble than usual. The entire corneal epithelium appears abnormal,[3] as probably is the conjunctival epithelium. This may be due to infection, exposure, inflammatory mediators, and denervation with loss of 'trophic factors', all of which may lead to alterations in cell surface properties. The changing shape, size, and distribution would support the concept of a generalised abnormality that is quite different from the local lesions of acute herpes simplex keratitis, but more akin to those seen in keratoconjunctivitis sicca (although morphologically distinct). The subsequent diffuse stromal haze and decrease in corneal sensation would lend support to this hypothesis.

There are two groups: one with early onset (within the first three months of the rash) and the other with late onset. The latter group of patients have the more severe problems, such as cataract, raised intraocular pressure, and corneal ulceration. The high risk of recurrence may necessitate repeated observations and prolonged topical steroids. Posterior subcapsular lens opacities can arise from both chronic iritis and long-term topical steroid, but steroid is not recognised as giving rise to nuclear sclerosis. Raised intraocular pressure may be due to the necessarily intense and prolonged topical steroid treatment or a trabeculitis accompanying the iritis. An acute hypertensive uveitis will usually settle within a few days on thorough treatment with topical steroids, but if there is steroid-induced glaucoma the prednisolone is replaced by fluoromethalone and timolol eyedrops. With severe refractory glaucoma drainage surgery may be necessary. Continuing denervation of the cornea and cojunctiva leads to neuroparalytic keratitis and ulceration, which is compounded by steroids in the abscence of a tarsorrhaphy. The circinate plaque deposits are distinct from those described in corneal infections.[8]

The results of surgery were good in this series. A booster dose of topical steroid is required postoperatively after all intraocular surgery for at least three months. Neuroparalytic ulcers were completely healed within a few days of tarsorrhaphy. Usually a temporal third was sufficient but occasionally a middle third was essential, and it was important to maintain the topical medication. Despite traditional reservations about intraocular surgery in patients with complicated ophthalmic zoster and the relatively small number of cases, we were pleasantly surprised by our encouraging results.

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