Correspondence

Iritis in Reiter’s disease: an example of the Auer principle

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Sir, Auer (1920) noted the effects of local inflammation in serum sickness. Xylol was used to produce local inflammation in the ear of a rabbit with serum sickness, and the animal was then given a repeat injection of serum. The treated ear became inflamed and gangrenous, while the other ear remained unaffected. Similar experiments were carried out in a rabbit’s eye (Gamble et al., 1970), where, after an intravenous dose of bovine serum albumin (BSA), 1 eye was given an intravitreous injection of endotoxin. Local inflammation was produced, and when it had resolved a further parenteral injection of BSA produced an acute iritis only in the eye injected with endotoxin. One explanation put forward for these reactions was that under appropriate conditions circulating immune complexes localized into that area associated with inflammation.

Acute iritis is a recognized complication of Reiter’s disease, occurring in 8% of patients (Csonka, 1972), but its cause is unknown. We would like to report a case of Reiter’s disease whose development of iritis may be an example of Auer’s principle.

CASE REPORT

A 44-year-old male, HLA B27 positive, was diagnosed as having Reiter’s disease in 1958 when he developed urethritis, conjunctivitis, and arthritis, the latter involving the knees and ankles. He was troubled with recurrent iritis in the right eye in 1958, 1963, and 1967, for which he was treated with a partial iridectomy in 1967. In 1971 he developed low back pain and at that time was noted to have radiological changes of bilateral sacroiliitis and ankylosis of the thoracolumbar spine. In 1972 he developed further arthritis in the knees and ankles, which settled with non-steroidal anti-inflammatory drugs. In May 1977 he received a blow over the right eye and developed bruising of the eyelids but no obvious local damage to the eye. Five days later he developed an acute iritis, which resolved after 2 weeks’ treatment with topical corticosteroids. At that time his peripheral joints were inactive, he had minimal back pain, and his therapy was indomethacin 100 mg a day. Laboratory investigations showed an erythrocyte sedimentation rate (ESR) of 10 mm in 1 hour, C-reactive protein 1-44 mg/100 ml (normal <0-8 mg/100 ml), and circulating immune complexes as detected by the method of Hay et al. (1976) were greater than 3 standard deviations above the normal range. Six months later he had only bilateral Achilles tendinitis, and there had been no further attacks of iritis. The ESR was 41 mm in 1 hour, C-reactive protein 3-1 mg/100 ml, and circulating immune complexes remained elevated.

COMMENT

This was the first attack of iritis experienced by this patient in 10 years, so that its occurrence following the blow over the eye is unlikely to be coincidental. Gamble et al. (1970) concluded that damage to the uveal vasculature appeared to be necessary to facilitate deposition of circulating immune complexes and the development of iritis. Concussion to the iris, no matter how small, produces vascular changes in the uveal vessels. These consist initially of an ischaemic spasm, followed by a prolonged reactive vasodilatation and hyperaemia of the uveal tract (Duke-Elder, 1972).

Compared with normal controls this patient had elevated levels of circulating immune complexes, as measured by the solid phase Clq radioimmunoassay, before and after his episode of acute iritis. This patient may be an example of the Auer principle, in that vascular changes occurring after the blow to the eye enabled the deposition of circulating immune complexes in the iris, causing acute iritis. The cause of iritis in Reiter’s disease is unknown, but his case leads to speculation that it could be secondary to deposition of circulating immune complexes.

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References


Notes

Search