Retinal haemorrhages after prolonged use of salicylates

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The possibility of retinal haemorrhages originating from the prolonged use of sodium salicylate was considered by Walsh and Hoyt (1969) in a patient who had taken 40 grams several times a week for 10 years. He had been using a solution of sulphathiazole for a long time to paint his throat and it was suggested that this might have caused some periarteritic lesion accounting for the retinal haemorrhage.

Salicylates are analgesic antipyretic drugs and may be given in high doses of up to 7 to 8 grams daily for long periods in cases of rheumatic fever, rheumatoid arthritis, and chronic types of rheumatism.

Salicylate therapy may cause haemorrhages, hypoprothrombinaemia, and hypo-coagulability of the blood; Ashworth and McMemie (1944) reported two cases in which intravenous salicylates were probably responsible for death, widespread haemorrhages particularly involving the brain being found at autopsy.

We have observed two cases of retinal haemorrhage due to the prolonged use of salicylates, and these are described below because of their rarity.

Case reports

There were a few petechial haemorrhages on the skin. The chest, abdomen, and blood pressure were normal. There were no abnormal neurological findings and no septic foci. The urine was free of albumen and sugar and the faeces free of parasitic ova. The total and differential blood counts, blood sugar curve, and blood cholesterol were normal. The erythrocyte sedimentation rate was moderately high. The prothrombin level was low. Bleeding and clotting times were slightly increased. There was no headache or vomiting. Besides salicylates, vitamins and corticosteroids had been given in moderate amounts. There was no history of trauma, strain, or asphyxia.

Case 1, a 60-year-old woman, complained of deterioration of visual acuity in both eyes for 15 days. She had been taking 6 g. sodium salicylate daily by mouth for the last 2 months for the treatment of chronic rheumatic arthritis of the knee joints.

Externally both eyes appeared normal. The visual acuity in each eye with correction was 4/60. The ocular tension was normal. Both fundi showed fine flame-shaped retinal haemorrhages, but the retinal vessels and optic disc were normal for the patient’s age. An erroneous diagnosis of arteriosclerotic retinopathy was made.

When the salicylates were stopped the visual acuity gradually improved to 6/9 with correction. Both fields of vision were normal. After 2 months the retinal haemorrhages had resolved.

Case 2, a 10-year-old girl, who had been given 4 g. sodium salicylate by mouth daily for 40 days for rheumatic fever, complained of diminution of visual acuity.

Externally the eyes were normal, but the visual acuity with correction was only 2/60. The ocular tension was normal.

The right fundus showed multiple flame-shaped retinal haemorrhages, and the left fundus retinal haemorrhages of various sizes, some of which were changing towards a yellowish colour. On the temporal side of the optic disc a small pre-retinal haemorrhage was seen with incipient retinitis proliferans. The retinal vessels and optic discs were normal.

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When the salicylates were stopped the retinal haemorrhages gradually absorbed and the visual acuity improved to 6/12 in the right eye and 6/60 in the left. The fields of vision were normal in both eyes and when the patient was last seen the visual acuity was still improving.

**Discussion**

The prolonged use of salicylates gives rise to variable degrees of hypoprothrombinaemia and hypocoagulability of the blood. In the two cases described the retinal capillaries were susceptible, giving rise to retinal haemorrhages. There was no retinal vascular narrowing as is seen in cases of quinine poisoning.

De Schweinitz (1896) cited the case of a 16-year-old girl who fell asleep after consuming 8 g. sodium salicylate in ten hourly doses and awoke completely blind in both eyes. After 10 hours, however, she could count fingers and within 24 hours the visual acuity was normal. When dogs were given large amounts of salicylates parenterally their vision diminished but there was no narrowing of the retinal arteries as in quinine poisoning. As Grant (1962) pointed out, loss of vision is not always associated with papillary involvement and retinal vascular narrowing does not always develop after there has been loss of vision.

The individual susceptibility to salicylates varies within wide limits. Hypersensitivity may be associated with urticarial skin rashes, angioneurotic oedema, or asthma. The eyelids, face, and tongue swell, and there is a danger of laryngeal oedema with resultant asphyxia. Aspirin may rarely also produce keratitis and conjunctivitis as well as hypotony. In large amounts it may cause gastrointestinal bleeding (Pierson, Holt, Watson, and Keating, 1961).

A large single dose may cause intoxication, and severe poisoning may be produced by 10 to 30 g. sodium salicylate in adults, but individual susceptibility varies greatly and much larger amounts may be taken without symptoms ensuing. In mild cases of salicylate poisoning, there is ringing in the ears, and in more severe cases excessive sweating, dimness of vision, and disorientation with excitement. Greer, Ward, and Corbin (1965) reported toxic brain swelling and papilloedema in one of five patients who took large amounts of aspirin (up to 20 tablets daily) for a considerable time. Their experience was that salicylates accounted for 26 per cent. of all poisonings, 7.7 per cent. of all deaths by poisoning, and 18 per cent. of all fatal poisonings in children under 5 years of age. Deafness and transitory third nerve paralysis have also been reported. Renal impairment increases the possibility of salicylate poisoning.

**Summary**

1. Two rare cases of retinal haemorrhage due to the prolonged use of a large dose of sodium salicylate are described.

2. This large dose may give rise to hypoprothrombinaemia and hypocoagulability of the blood, and retinal haemorrhages may occur if the retinal capillaries are susceptible.

**References**


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