**DACRYOCYSTITIS IN ACUTE LEUKAEMIA***

**BY**

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Leukaemic infiltration of the lacrimal sac is rare (Duke-Elder, 1952) and dacryocystitis due to leukaemic involvement of the sac has seldom been reported (Stokes, 1938; Busina, 1950). The number of cases recorded is increased when the reports of Sulzer and Duclos (1906), Pascheff (1927), and Weve (1928) are considered, but these authors described lymphomata of the sac without the typical blood changes of leukaemia. Infiltration of the lacrimal glands in leukaemia is much more commonly seen (Trevor-Roper, 1958). The patient here described had lacrimal obstruction in acute leukaemia together with terminal enlargement of the lacrimal glands.

**Case Report**

An 80-year-old female was admitted to the Victoria Infirmary, Glasgow, in February, 1961, to have treatment for acute lymphatic leukaemia with secondary anaemia. The blood picture on admission was: haemoglobin 5 g./100 ml.; white cells 24,700 per cu. mm. (17 per cent. neutrophils, 70 per cent. lymphocytes, 13 per cent. lymphoblasts).

The patient complained of constant and profuse watering from the left eye, and after the leukaemia was brought under control by blood transfusions and steroid therapy she was transferred to the ophthalmic wards. Although she had not made any complaint until just before admission, her history was one of a general deterioration in health for 6 months, and during the same time there had been constant severe left-sided epiphora with occasional mild right-sided epiphora. During this initial period of treatment, repeated bacteriological culture had shown the lacrimal passages on both sides to be sterile.

**Examination.**—Before the patient was transferred to the ophthalmic wards the blood picture was: haemoglobin 10 g./100 ml.; white cells 21,000 (20 per cent. neutrophils, 78 per cent. lymphocytes). There were some enlarged cervical lymph nodes and slight enlargement of the spleen and liver, although signs of leukaemia were less evident than on admission. There was an obvious firm swelling of rubbery consistency over the left lacrimal sac which was not patent. There was a smaller and softer swelling over the right lacrimal sac and on this side the tear passages were patent. There was no other evidence of leukaemic deposits in the eyes or orbits and the nose and nasopharynx were normal. The lacrimal glands were not enlarged and Schirmer’s test indicated some lacrimal secretion on both sides. There was an almost mature cataract in the left eye which obscured the fundus. There was no retinopathy in the right fundus.

Bacteriological culture from the left lacrimal passages now produced a heavy growth of penicillin-resistant *Staphylococcus aureus pyogenes* (coagulase-positive). Chloramphenicol was instilled into the left lacrimal passages at frequent intervals until there was no evidence of infection, but despite this the symptoms persisted and there was no reduction in the size of the swelling. This was in contrast to the splenomegaly, hepatomegaly, and lymphadenopathy, all of which decreased as the blood picture and feeling of well-being continued to improve.

**Operation.**—The left lacrimal sac was removed intact under local anaesthesia together with portions of the surrounding tissue. Dacryocystectomy was performed in preference to a drainage operation because the nature of the sac was consistent with leukaemic

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infiltration and also because of the patient's age. The sac was obviously enlarged but was clearly differentiated, and when it was opened the walls were seen to be greatly thickened and the lumen almost occluded.

Microscopic examination showed the wall of the sac to consist almost entirely of mature lymphocytes arranged in dense sheets with some connective tissue between the sheets. The epithelium was normal (columnar and non-ciliated). Inflammatory cells were searched for in serial sections using special stains, but the only cells other than lymphocytes which could be found were a few plasma cells at the lower end of the sac beneath the epithelium. There were no other inflammatory cells and no evidence of the fibrosis of chronic inflammation. The microscopic picture was entirely consistent with a leukaemic infiltration which had virtually replaced the wall of the sac (Figure). The small shreds of tissue surrounding the sac which had been removed for examination showed normal connective tissue and striped muscle fibres with no inflammatory changes and no lymphatic infiltration.

**Figure.**—Lymphocytes in lymphatic infiltration of lacrimal sac. Haematoxylin and eosin. × 400.

**Result.**—After operation there was only occasional slight epiphora on the left side and this disappeared in a few weeks. By this time the blood picture showed that the leukaemia had been converted from acute to chronic, but before long symptoms of acute peptic ulcer developed and steroid therapy had to be stopped.

Some time after this the patient's general condition began to deteriorate, other systemic signs of chronic lymphatic leukaemia appeared, and the patient eventually died. The last blood report showed: haemoglobin 7·8 g./100 ml.; white cells 29,500 (2 per cent. neutrophils, 14 per cent. lymphocytes, 82 per cent. smear cells).

In the weeks before death the lacrimal glands became enlarged and were thrown into prominence by sinking of the eyes into the orbit as cachexia increased. Oedema appeared at the right macula and the retinal veins became engorged but no other sign of retinopathy had been seen in the course of the illness.
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Post-mortem Examination.—The lacrimal glands were alike and showed the appearances of leukaemic infiltration in chronic lymphatic leukaemia. There were areas of normal gland tissue interspersed with large areas of lymphocytic infiltration. The infiltration was completely and uniformly lymphocytic, no plasma cells, polymorphs, or other inflammatory cells being seen. There was no fibrosis in relation to the lymphocyte aggregates which one would expect in chronic inflammation. The lymphocytes formed thick layers which appeared to infiltrate at first between the acini which remained normal in appearance until destroyed by pressure.

The cause of death was confirmed to be chronic lymphatic leukaemia, but there was only moderate enlargement of the lymphoid tissue throughout the body and slight infiltration of the viscera by lymphocytes, and nowhere was the lymphocytic infiltration so gross as in the lacrimal glands.

Discussion

Many cases of leukaemia, acute and chronic, are seen in this hospital, but this is the first in which leukaemic dacryocystitis has been noted. The left lacrimal sac appears to have been specifically affected insofar as there was no evidence of similar infiltration elsewhere in the body and only moderate lymphocytic infiltration of the viscera and lymphatic tissue was found post mortem. There was, moreover, no sign of leukaemic deposit in any neighbouring structure and the possibility of the lacrimal sac being involved in direct spread from the conjunctiva or orbit, as has often been described (Trevor-Roper, 1958), was excluded by histological examination of the surrounding tissues. The possibility that the right sac was involved could not be explored since, although there was slight swelling over it, operation was not justified and permission to examine the face post mortem was not obtained.

While the lymphadenopathy and visceral enlargement subsided with appropriate therapy, at least in the early stages of treatment, it is interesting to note that there was no decrease in the size of the lacrimal sacs and that the symptom of epiphora was not relieved until the sac was excised. It is also noteworthy that, while the lacrimal glands are the parts of the lacrimal apparatus most commonly involved in leukaemia, they were not affected until a late stage in this case by which time one lacrimal sac was already occluded.

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REFERENCES

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