present. Haemoglobin electrophoresis showed a single band of Hb S. March 3: Bilirubin 4.5 mg./100 ml.; blood urea 43 mg./100 ml. March 5: Hb 3.8 g./100 ml.; P.C.V. 14%; M.C.H.C. 21%; reticulocytes 16.2%; W.B.C. 18,300/c.mm. (normoblasts 23 per 100 W.B.C.). March 9: Hb 4.1 g./100 ml.; reticulocytes 50%. March 12: Hb 4.6 g./100 ml.; P.C.V. 17%; M.C.H.C. 26.5%; reticulocytes 40%; W.B.C. 25,900 c.mm. (normoblasts 11 per 100 W.B.C.). X-ray films of the skull and hands showed no abnormality.

His condition improved gradually while he was in the oxygen tent and having digitalis. On March 14 he was given a transfusion of 160 ml. of packed cells and the haemoglobin immediately after was 14 g./100 ml. The next day the cardiac murmur was barely audible.

Investigation of the parents' blood gave the following results. Mother: Hb 15.1 g./100 ml.; Hb F 2.6%; sickling weakly positive; haemoglobin electrophoresis showed haemoglobin A/S. Father: Hb 14.9 g./100 ml.; Hb F 0.81%; sickling positive; haemoglobin electrophoresis showed haemoglobin A/S.

These results support the diagnosis of sickle-cell anaemia for the boy.

COMMENT

This report is of importance, since this condition has only rarely been recorded as occurring before the age of 6 months. In view of the many families from West Africa and the West Indies now in this country, similar cases may occur and might be missed because sickle-cell anaemia would not be considered.

I am grateful to Dr. Dennis Cottom for allowing me to report this case, and would like to thank Dr. G. Wetherley-Mein for his advice.

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Unusual Complication of Glandular Fever

The following unusual occurrence in a case of glandular fever may be found of interest.

CASE REPORT

A student nurse aged 19 was admitted to hospital on November 4, 1960, complaining of a sore throat for 10 days, which had become much worse recently. On examination both tonsils were large and there was considerable peritonsillar and palatal swelling, but with no exudate. The pharynx was red. Both tonsillar glands were enlarged and the tip of the spleen was palpable. A provisional diagnosis of glandular fever was made.

Investigations on day of admission: W.B.C. 7,100/c.mm. (neutrophils 26% lymphocytes 37%, monocytes 3%, atypical mononuclears 34%). E.S.R. 17 mm. in one hour (Westergren). Paul-Bunnell test—unabsorbed serum positive to a titre of 1:5,120; guinea-pig absorbed serum positive to a titre of 1:5,120; ox-cell absorbed serum positive to a titre of 1:160. Throat swab culture: normal flora only.

In the course of the next week the throat became extremely painful and the tonsils very enlarged, meeting in the midline. Further throat swabs on November 9 showed normal flora, no Vincent's organisms, and in particular no beta-haemolytic streptococci. Antibiotics were not given, treatment consisting only of analgesics and sedatives.

On November 10 the patient suddenly complained of an unpleasant taste in the mouth followed by a gush of foul-smelling blood-stained pus. Examination showed that the left tonsillar swelling had disappeared, the right remaining very swollen but with no exudate visible. In view of this it was decided to incise the right peritonsillar swelling. This

produced a large quantity of blood-stained pus, the right tonsil then returning to its normal position. Culture of this material grew only normal throat commensals.

The clinical recovery was dramatic, and the temperature, which had been rising up to 103° F. (39.4° C.), immediately fell to normal (see Chart). She subsequently made an uneventful recovery and was discharged 10 days later.

Chart showing the sudden fall in temperature which occurred immediately after evacuation of the peritonsillar swellings.

COMMENT

In the anginose form of glandular fever it is common for the throat to become secondarily infected, usually with haemolytic streptococci or Vincent's organisms, and peritonsillar abscesses may occur as a result. However, in this case there was no evidence of secondary infection.

Spontaneous rupture without secondary infection is known, for example, in the spleen (Shinton and Hawkins, 1956).

Other cases of suppurating cervical adenitis and retropharyngeal abscesses have been reported (Moir, 1930). Most of these have been thought to be due to secondary infection, but the absence of this has not been well authenticated. Post-mortem examination in some reported fatal cases of glandular fever has shown haemorrhage and necrosis in sections of the cervical glands and tonsils (Custer and Smith, 1948; Shinton and Hawkins, 1956).

We feel that secondary infection is excluded in our case, and suggest that the fever and malaise were probably due to necrotic material under extreme tension, which when relieved resulted in a dramatic recovery.

We believe that the spontaneous rupture of a sterile collection of necrotic material from the peritonsillar region is an unusual occurrence in glandular fever. It is not mentioned by Tidy (1948) or by Bernstein (1940) in his exhaustive review of the literature up to 1940 or in a leading article (*Brit. med. J.*, 1960) dealing with this subject. It is quite possible that many cases have considerable necrosis which absorbs spontaneously, as appearances of almost similar severity are fairly common. However, we are not suggesting that incision of these swellings is indicated in all cases of glandular fever.

We thank Miss T. Turner, the matron of St. Thomas's Hospital, and Dr. H. K. Goadby, honorary physician to the nursing staff, for allowing us to publish this case.

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