AN "EXPLOSIVE" OUTBREAK OF HAEMOLYTIC STREPTOCOCCAL TONSILLITIS ON AN R.A.F. STATION

BY

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The *Bulletin of War Medicine* of July, 1943, Vol. III, No. 11, published an abstract of an article by Bloomfield and Rantz. This described an outbreak of streptococcal sore throat in an American Army camp. The following description of an epidemic which occurred on an R.A.F. station may prove of value. It will be seen to be similar in many respects, but it presents some interesting points of comparison.

Incidence of Cases

The outbreak occurred in Nov., 1942, and of a total camp personnel of 14 officers and 351 airmen, 89 airmen were affected. The "explosive" nature of the epidemic is indicated by the daily incidence of fresh cases, which was as follows:

1st day	..	..	..	4	5th day	..	..	..	5
2nd "	..	..	..	42	6th "	..	..	..	2
3rd "	..	..	..	25	7th "	..	..	..	2
4th "	..	..	..	8	8th "	..	..	..	1

Subsequent to this, no fresh cases were reported.

Clinical Features

Clinically, the patients were acutely ill, showing marked local and general reactions. The tonsils were red and oedematous, with patches of exudate. In many cases the cervical glands were enlarged and tender. Those patients without tonsils had a diffuse pharyngitis. Temperatures ranged up to 104°.

Response to the sulphonamide group of drugs was not as dramatic as might have been hoped, and in many cases there was a recrudescence of pyrexia around the tenth day. Blood sedimentation rates were almost invariably markedly raised—in some instances as high as 85 mm. (Westergren) in 1 hour—and even after the lapse of 4 to 5 weeks an increase was observed in many cases. Convalescence tended to be prolonged, 3 to 4 weeks elapsing before discharge from hospital. Two cases of otitis media, one of acute carditis, and two of arthritis were among the complications. Throat swabs from a number of patients all gave cultures of a haemolytic streptococcus which belonged to Group A Lancefield and was serologically Type 9.

Investigation

In the course of investigation it was noted that the cases were confined entirely to those who dined in one particular mess. Of the officers, N.A.A.F.I. personnel, or civilians (none of whom fed in this mess) none was affected. There was no relation between the case incidence and the distribution of personnel in the sleeping billets. These features suggested a food-borne infection rather than the more usual "droplet" spread from case to case, although it is probable that the 10 cases occurring from the fifth to the eighth day were infected in this fashion. By studying the movements of affected personnel immediately before the outbreak, the period when the infection was presumed to have occurred was narrowed down to between 2 and 3 days prior to the first case being seen. Bacteriological examination of food-handlers was carried out. It was discovered that a cook who had been in charge of the preparation of milk on one of the days under suspicion was heavily infected with haemolytic streptococci. These were of the same serological strain as those isolated from patients, although the cook was himself symptom-free. The milk in question was tinned, and was prepared 2 to 3 hours before use by mixing one part of tinned milk with two parts of boiling water. This would give the mixture an initial temperature of about 65 to 70° C. When it was allowed to stand this would rapidly drop, providing an excellent medium for growth of organisms at optimum temperature. During the period in question the cook was suffering from a severe head cold and was sneezing continually. The conclusive proof of isolating the organisms from the milk under suspicion was impossible, as a sample was not available. Circumstantial evidence, however, strongly supports the assumption that this was the medium by which the infection was spread.

Carrier Rate

With regard to the cookhouse personnel, the following statistics were obtained when throat swabs were taken 12 days after the outbreak: 20% had developed acute tonsillitis; 45% were carriers of haemolytic streptococci (30% Group A Type 9, 15% not Group A) and symptom-free; 35% had negative throat swabs and were symptom-free. If it is permissible to consider these persons as a representative cross-section of the community (the approximate similarity between the percentages who actually developed the condition would seem to support this assumption—20% of the cookhouse personnel and 25% of the station personnel), it can be concluded that 12 days after the outbreak of the epidemic 45% of the community were carriers of haemolytic streptococci. In 30% of cases the organism belonged to Group A Type 9.

After the lapse of a further 12 days, swabbing of the throats of a number of personnel chosen at random showed that the haemolytic streptococcus carrier rate had fallen to 25%. Unfortunately grouping and typing of this further series of throat swabs was not carried out, but it is probable, however, that this decline of 20% occurred in the numbers of those who harboured haemolytic streptococcus Group A Type 9.

Presuming that the decline in carrier rate during the first 12 days subsequent to the outbreak was approximately the same as that during the second 12 days (in all probability it was even greater), it would appear that on the first day of the epidemic the proportion of personnel who harboured this particular strain of organism in their throats but were symptom-free (i.e., were immune) would have been in the region of 50%.

In the intervening period between the two series of throat swabs the community had been divided into two sections, which were isolated from one another. The personnel of one section carried out gargling with pot. chlor. and phenol twice daily, while among those of the other section no preventive measures were taken. It was noted that, in spite of this, the carrier rates of the two sections remained exactly the same. This would seem to substantiate the doubts which have been cast on the usefulness of gargling in an outbreak of this kind.

Conclusion

It is concluded that this was an epidemic in which the infection was primarily food-borne, with case-to-case spread playing a very minor part. It is thought that the reason for the comparatively small incidence of case-to-case infection may have been the fact that those susceptible had been picked out by the primary infecting agent, the few who escaped this hazard rapidly succumbing to "droplet infection." This is borne out by the remarkably large number of immunes—probably at least 50% of the community.

Comparison with a Similar Outbreak

On comparing this epidemic with that described by Bloomfield and Rantz the following will be noted: (a) That in both cases a food-borne infection was suspected. (b) That in both instances case-to-case infection was minimal: while in the latter this was presumed to be due to a low virulence of the infecting organism, in the former the reason was thought to be that the majority of those who escaped were immune to the particular strain concerned. (c) That the causal organism in the American epidemic was of a different Griffith subtype, being Group A Lancefield subtype 15. (d) That the main clinical features were similar in both, with the exception that Bloomfield and Rantz describe the appearance of a characteristic scarlatiniform rash with subsequent desquamation in one-fourth of the cases, while in no instance was a rash observed in the present epidemic. This suggests that haemolytic streptococci belonging to subtype 9 play no part in the causation of scarlet fever, in contradistinction with those belonging to subtype 15.

Summary

An explosive outbreak of haemolytic streptococcal tonsillitis is described.

The evidence suggested that it was a food-borne infection, probably by contamination of milk by an infected cook, who was a carrier.

A large proportion of the community were immune to this particular strain of organism.

Routine gargling had no effect in reducing the carrier rate during the epidemic.

A comparison is made with a similar type of outbreak which occurred in an American Army camp.

I wish to tender thanks to Dr. N. Crowley for the help she gave me in the bacteriological investigation of throat swabs, etc.

TREATMENT OF RHEUMATOID ARTHRITIS WITH BISMUTH

BY

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The use of gold against active rheumatoid arthritis has fully justified itself in spite of the faulty premisses which led to its introduction. Analysis of my last 200 cases indicates that it results in the disappearance of symptoms and signs of activity in 70%, but relapse within six months occurs in the majority of those subjected to only one course of injections—usually 0.8 g. Four courses in two years appear to eradicate the disease in 55%, but the possibility of recurrence, even 15 years later, forbids the use of the word "cure." The great objection to gold treatment is the high incidence of toxic reactions. In the early days it was as much as 45%, but with decreased dosage, and the use of the less toxic calcium aurothiomalate, the figure has fallen to 10 in the last 60 arthritics so treated—i.e., approximately 16%. Immediate interruption of treatment at the first sign of any one of the numerous reactions which may arise will almost invariably be followed by a slow but complete disappearance of the toxic effect. I have seen one case of agranulocytosis recover after five months, and one of gold nephrosis with oedema recover after three months. In each case instructions as to dosage, choice of preparation, and watch for toxic signs had not been observed. The commonest and most persistent reaction is seen in the skin, in which various irritating rashes are produced. It is this which has led to such fear of gold.

It seems probable that gold acts by virtue of its being a heavy metal, and not from any specific effect on the infecting agent responsible for the arthritis. In order to see whether it were possible to obtain the same effect with a less toxic substance, I started, late in 1942, administering bismuth. This substance was chosen because pharmacologically it acts similarly to heavy metals, it is easily procurable in injectable form, and its toxicity is low.

Method

Twelve consecutive patients with active rheumatoid arthritis were treated with weekly intramuscular injections of injectio bismuthi (9) or injectio bismuthi salicylatis (3). One mil of the former and 2 mils of the latter contain 0.2 g. of bismuth and bismuth salicylate respectively. These quantities were used for 10 injections, giving a course-dose of 2 g.

Clinical Material.—Ten females, two males; aged from 24 to 63 years. With the exception of one who had suffered for 19 years, they gave histories of 9 years (2), 8 (2), 4 (4), 2 (1), 1 year (1), 1/2 year (1). Eleven patients had had one or more courses of gold in the past; in three only had it failed to do any good. The others had relapsed, usually within 6 months of finishing the course.

Signs of Activity.—In all cases there were pain and swelling of many joints, a recent extension of the disease to joints previously spared, and a raised E.S.R.: in one hour the figure limits (Westergren method) were 22 mm. to 100 mm.

Results

The effect of treatment was classified as:

Group 1.—*Very good* if at the end of the course pain and swelling (not due to old deformity) had disappeared, and if the E.S.R. was below 7 mm. for one hour. Only four patients qualified for this category, the initial and final E.S.R. figures being 22/2, 23/5, 70/5, 23/5.

Group 2.—*Good* was the classification of those patients whose clinical signs had improved as in the former group but in whom the final E.S.R. for the first hour was between 7 and 15 mm. There were two of these with E.S.R. figures of 52/11 and 50/11.

Group 3.—*Moderate improvement:* those patients whose pain decreased and whose ability to perform certain movements—e.g., making a fist and walking—improved beyond doubt. Two fell into this category, with a noteworthy inertia of sedimentation rate. The initial and final figures were 40/22, 35/49.

Group 4.—*Failure*—i.e., those arthritics whose symptoms and signs showed no improvement. Of these there were four. The corresponding E.S.R. figures were 55/34, 38/35, 31/26, 100/110.

None of the patients has been under observation for less than 9 months or more than 1½ years from the beginning of bismuth treatment.

For practical purposes Groups 1 and 2 may be taken together as showing good results and Groups 3 and 4 as being failures. This gives improvement in half of a small series. There was no relation between the duration of the disease and the age of the patient with regard to the effect, or absence of effect, of bismuth on the activity of the arthritis. The two males were failures. Three patients who had had gold previously and obtained no relief provided three bismuth failures.

Relapses.—In Group 1 there has been no relapse. In Group 2 both have relapsed within 2 months of the end of treatment. In Group 3 both members relapsed within 7 weeks.

Toxicity.—There were no toxic reactions.

Difference of Preparation.—Bismuth salicylate provided two failures and one good result. It is probably immaterial whether bismuth or the salicylate is employed. The bulk of the former is less.

Control.—Six rheumatoid arthritics were given weekly intramuscular injections of 1 mil of sterile water. At the end of six weeks this was stopped because no objective improvement was seen, and in only one case did the patient declare herself to suffer less pain.

Conclusion

(1) Bismuth by intramuscular injection appears to be capable of exerting a beneficial influence on the course of rheumatoid arthritis. (2) It compares unfavourably with gold in this respect, the probability of lasting benefit being less than half that expected from gold. (3) Relapse appears to occur more quickly than that following gold. (4) Gold-resistant arthritics are unlikely to benefit from bismuth.

Bismuth is indicated only for rheumatoid arthritics who show intolerance, whether physical or mental, to gold.

Medical Memoranda

Trypanosomiasis in a European treated with Pentamidine

As cases of early trypanosomiasis may occasionally be seen at the present time among Europeans returning from tropical Africa, an account of the treatment of such a case with 4,4-diamino-diphenoxypentane (pentamidine, or M & B 800) is topical. An extensive trial of this drug on natives in Uganda was reported by Lawson (1942), and a fatality, possibly attributable to it, by McComas and Martin (1944).

CASE HISTORY

The patient, a Polish officer aged 32, had been stationed in a tsetse-fly area in Sierra Leone from April, 1942, to May, 1943. He thinks that he was bitten several times, but remembers particularly an occasion in Jan., 1943, when he had what he took to be a tsetse fly inside his trousers. Apart from malaria he was well until May, 1943, when he had a brief attack of pain and swelling of the glands in the neck, axillae, and groins. In June, 1943, he returned to the United Kingdom, and noticed occasional headaches, fever, and swelling of the face in the mornings. In the middle of September he reported sick with what he thought was malaria; scanty trypanosomes were found in thick blood films, and he was admitted to a military centre for tropical diseases.

Examination revealed the following: Irregular pyrexia to 100.5°; pulse rate almost constantly 100 to 110 a minute; mental state apparently normal; puffy swelling of lower eyelids and adjacent face; slight enlargement of occipital, axillary, and inguinal glands; no rash; spleen not palpable. A blood count showed: red cells, 3.7 millions per c.mm.; haemoglobin, 78%; white cells, 12,500 per c.mm.—neutrophils 70%, eosinophils 5%, monocytes 4%, lymphocytes 21%. Very scanty rings of *Plasmodium falciparum* were found in thick blood films, and, on two occasions, scanty trypanosomes in blood films and in juice from a cervical gland.

Treatment was begun in Sept., 1943, with 100 mg. of pentamidine dissolved in 5 c.cm. of sterile distilled water, given intravenously