CASE REPORTS

- **Relapsing Fever Probably Caused by Borrelia Duttonii**
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Relapsing Fever Probably Caused by Borrelia Duttonii

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UP TO THE TIME of the case herein reported, the health officer for the southeast district of the Los Angeles County Health Department had had no reports of relapsing fever in the district since a physician in La Verne reported a case more than five years ago, although it is suspected that many persons had the disease in the interim and it was not recognized. The number of cases reported to the California State Department of Public Health in the five years 1945-49 averaged ten a year.[1]

REPORT OF A CASE

A white male college student 22 years of age had had intermittent chills and fever for 23 days when first observed September 24, 1951. He had been well until September 3, when, while at Big Bear Lake after twelve days on a camping trip, chills and fever developed suddenly and lasted a day and a night. At the same time the patient had headache, anorexia, vague abdominal pain, slight stiffness of the joints and a feeling of tightness in the chest. During the next week he felt well except for lassitude. Eight days after the onset, a second bout, which lasted three days, occurred. The temperature was 101 degrees F. and there was profuse sweating. Then again the patient was free of symptoms except for lassitude and loss of ten pounds in weight. Chills and fever recurred September 17 and lasted two days. A fourth attack, during which the patient vomited once, occurred September 24 and medical advice was sought.

Upon physical examination the patient appeared to be acutely ill. The tongue was coated. A few fine rales were heard in the hilar region of the chest. The abdomen was tympanitic, the spleen enlarged but not tender. In roentgen films the heart, lungs and mediastinum appeared to be normal.

The hemoglobin content of the blood was 15 gm. per 100 cc. and erythrocytes numbered 4,960,000 per cu. mm. The color index was 1.04. Leukocytes numbered 9,450—20 per cent lymphocytes, 8 per cent monocytes, 54 per cent segmented neutrophils, 17 per cent non-segmented neutrophils, and 1 per cent eosinophils. In bacteriological examination of the blood, no plasmodia were observed but many Borrelia spirochetes, probably Borrelia duttonii were present (Figure 1). Results of agglutination tests were negative for typhoid, paratyphoid and brucellosis. No organisms grew on a culture of the blood. The urine was not examined.

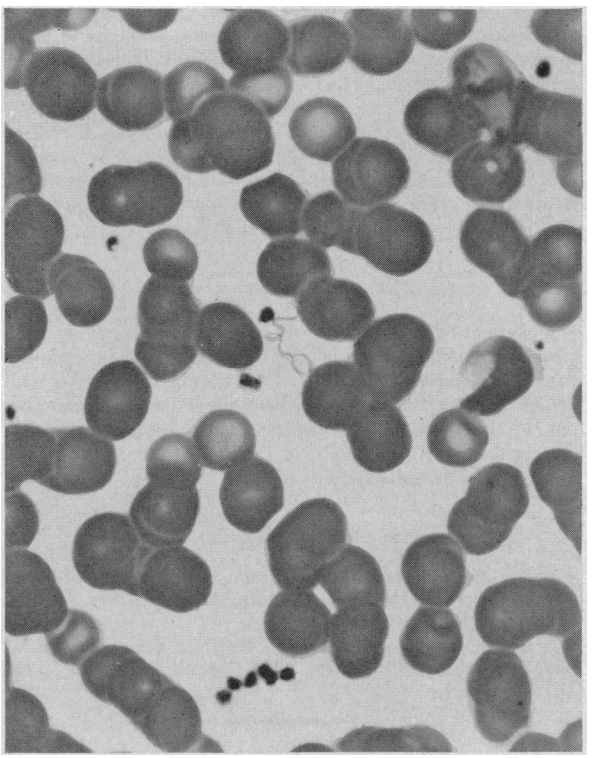

Figure 1.

Upon advice communicated from the clinic of Dr. Earle Moore at the Johns Hopkins University School of Medicine and Dr. T. B. Turner of the School of Hygiene and Public Health of the same university, the patient was given penicillin, 1,000,000 units daily for the first week, 600,000 units daily for the second week, and 300,000 units a day for the next five days. The fever abated soon after administration of penicillin was started, and the patient had no further attacks.

Textbooks mention relapsing fever caused by Borrelia duttonii, but few reports of cases could be found in the literature. Wynn and Beck[2] in a study of data on 283 cases that had been reported to the California State Department of Public Health between 1921 and 1944, made the following observations: In a majority of cases infection occurred along the Sierra Nevada range at or above 5,000 feet, where O. hermsi was the vector. In other cases the disease was con-

tracted at elevations between 250 and 4,500 feet, and in those cases O. parkerii was the vector. Rodent reservoirs (for example, chipmunks and tamarack squirrels) were proved in four cases—all over the 5,000-foot level. There was a seasonal pattern of occurrence of the disease. The peak months were June, July, August and September, a period following the emergence of rodents from hibernation, the opening of summer cabins, and influx of vacationists. However, there were some "out of season" cases in residents of endemic areas and it was conjectured that a disease colloquially called "squirrel fever" might be relapsing fever. The disease may be confused with brucellosis, malaria, pyelitis, influenza, typhoid, typhus, and sunstroke. It is characterized by sudden onset with fever, frontal headache, pain in the back, the limbs, and the joints, and pronounced prostration. Nausea and vomiting may occur. In a few instances in the California series, a rash was noted. The attacks usually last two to four days and the symptoms usually recur within three to twelve days. During the afebrile period, the patient feels entirely well. If no treatment is given, there may be as many as twelve relapses. In the majority of cases in the California series the patient had two to four attacks, a few as many as eight but each of decreasing severity. The infecting organism may be identified by either of two methods: microscopic examination of a smear of the blood, or animal inoculation. Smears should be prepared as thick or thin films of blood obtained at the peak of the attack before treatment is given. Wet preparations may be examined under the dark field if equipment is available.

SUMMARY

A patient who had several bouts of chills, fever with anorexia, headache and abdominal pain after a camping trip in a mountainous region of California was treated with penicillin after Borrelia spirochetes were noted in examination of the blood. The symptoms abated promptly and did not recur.

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REFERENCES

1. Personal communication from Dr. Herbert Meehan, health officer for the southeast district of the Los Angeles County Department of Health.

2. Wynn, H. L. and Beck, M. Dorthy: Present status of relapsing fever in California, Calif. Health, 2:12, Tas 7., 89-94, 1944.

Anuria Owing To Urethral Obstruction by Sulfadiazine Crystals

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ANURIA WITH OBSTRUCTION of the upper urinary tract has been reported on many occasions since the sulfonamide derivatives first came into general use.[1, 3-6] Anuria due to urethral obstruction secondary to sulfonamide therapy, however, while undoubtedly observed by other physicians, has not been as widely publicized. At the time the case herein reported was observed, the literature was reviewed and no report of anuria caused by crystalline obstruction of the urethra by a sulfonamide product was found. Later (in January, 1952) Dukes[2] cited four reports of cases in which lower urinary tract obstruction developed following treatment with sulfadiazine.

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REPORT OF A CASE

The patient, a boy aged 4 years and 8 months, was referred by another physician on February 7, 1951, because he had been completely unable to void that day after awakening in the morning. (Later it was determined that he had not voided for eighteen hours.) The referring physician, treating the patient for infection of the upper respiratory tract, had prescribed sulfadiazine and a total of 4 gm. of the drug had been taken at the time he was observed by the author. Upon physical examination, dullness upon percussion of the suprapubic region of the abdomen, suggestive of elevation of the dome of the bladder, was noted. There was pronounced stenosis of the external urethral meatus, and a whitish substance suggestive of crystals of sulfonamide derivative exuded from the orifice. First with a probe and later with a small sound, the external urethral meatus was dilated slightly and the packed crystals were broken to some extent. Thereafter, a small red rubber catheter was passed into the bladder and approximately 200 cc. of clear urine drained freely. In the course of catheterization, a considerable quantity of whitish crystalline material was expressed from the urethra. A specimen of the urine obtained from the bladder and subjected to routine analysis was noted to be within normal limits except that the urinary sediment contained many crystals of a structure typical of sulfonamide crystals. No organisms grew on a culture of the urine in 48 hours. After the catheterization, meatotomy was performed and the patient was sent home with instructions to force fluids and to return in five days for dilatation of the external urethral meatus. When the patient returned, the mother reported that he had voided freely after the initial therapy in the office, and had had no difficulty since that time.

DISCUSSION

The case reported illustrates one more complication to anticipate when prescribing sulfonamide therapy. Dukes said that one would expect this complication to occur more commonly in children than in adults because the diameter of the adult urethra is of sufficient size to permit the passage of the precipitated crystals, thus precluding obstruction. The patient in the case here reported had stenosis of the external urethral meatus, but whether this was a significant factor in causing urethral obstruction is unknown. However, it seems logical that any organic obstruction would tend to cause precipitation of the crystalline material and blockage of the urethral passageway. Dukes made no mention of stenosis of the external urethral meatus in the four patients that he reported upon.

The diagnosis was established easily in the present case because the patient was known to have been given sulfadiazine. Also, when the catheter was passed into the bladder, a satisfactory quantity of urine was obtained. This was at least enough to indicate that the patient had very little, if any, upper urinary tract obstruction. There did not appear to be any evidence of toxic nephrosis in the patient in the present case or in the patients reported upon by Dukes.

The treatment, namely stopping the drug, catheterization, meatotomy and forcing fluids by mouth, relieved the patient at once. Other measures, suggested by Dukes are alkalization of the urine and hot sitz baths. Dukes also mentioned that, if these measures fail, removal of the granular material by urethroscopic means under general anesthetic may become necessary.

The prognosis for complete recovery from this complication seems excellent. In all cases reported, the patients recovered promptly and without any known sequelae. Apparently the occurrence of this complication need not preclude further sulfonamide therapy. The patient in the present case