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Atovaquone Alone or with Fluconazole as Oral Therapy for Indian Kala-Azar

In an experimental model of visceral leishmaniasis (kala-azar) caused by *Leishmania donovani,* treatment with oral atovaquone induced microbiostatic activity [1]. Because atovaquone is well tolerated and active for treatment of other infections [2], we treated 15 Indian patients in Muzarffarpur (Bihar State) [3] to determine the effects of the agent in the treatment of human kala-azar. In this pilot study of previously untreated patients, 12 were male and 10 were <18 years old (age range, 5–45 years). Baseline data (mean ± SEM) included duration of illness (1.9 ± 0.3 months), body weight (29.6 ± 3.2 kg), spleen size (5.4 ± 0.8 cm), WBC count (5.3 ± 0.4 × 10^6/L), hemoglobin level (9.3 ± 0.6 g/dL), platelet count (154 ± 0.2 × 10^6/L), and microscopic splenic-aspirate parasite density score (3.1 ± 0.2) using a logarithmic grading scale of 0 (no amastigotes in 1,000 high-power fields) to 6+ [3, 4]. Atovaquone (Glaxo Wellcome, Research Triangle Park, NC) was given for 15–30 days, in suspension form, once daily at 30 mg/kg, along with one piece of buttered bread (~25 g of fat).

One patient was withdrawn from the study during week 1 because of vomiting. On day 15, two of 14 patients were afebrile, and repeated splenic aspirate results (blinded) for six patients indicated either partial parasitologic responses (decrease of ⩾2 grades in aspirate parasite density score; $n = 5$) or apparent parasitologic cure (density score of 0; $n = 1$). As per the protocol, the eight nonresponders were removed from the study; the six responders were treated for 15 additional days. After 30 days, two of the six responders were designated as apparent cures (afebrile, clinically improved conditions, decreased spleen size, and aspirate parasite density score of 0) [3, 4]; the conditions of four patients were improved, but aspirates still showed parasites (scores of 1+ to 2+). Both patients who were apparent cures were not treated further and relapsed within 8 weeks.

Because fluconazole also has measurable effects in kala-azar [3], we treated 13 additional patients for 15–30 days with atovaquone (30 mg/[kg·d]) plus oral fluconazole (once daily, 12 mg/kg) to determine if combining two partially active oral agents could induce an enhanced effect. Ten subjects were male, seven were <18 years old (age range, 7–58 years), and baseline data (mean ± SEM) included duration of illness (3.9 ± 0.3 months), body weight (35.6 ± 3.3 kg), spleen size (8.6 ± 1.2 cm), WBC count (4.0 ± 0.5 × 10^6/L), hemoglobin level (7.9 ± 0.5 g/dL), platelet count (153 ± 0.2 × 10^6/L), and microscopic splenic-aspirate parasite density score (3.6 ± 0.4). Three patients showed clinical progression of infection ($n = 2$) or increased hepatic transaminase values ($n = 1$) during weeks 1–2 and were removed from the study before day 15. Nine of the remaining 10 patients were afebrile by day 7, and on day 15, three were apparent cures and six were partial clinical and/or parasitologic responders; one patient showed no response and was withdrawn. For the nine patients treated for 15 additional days, day-30 evaluation showed apparent cures in two, clearcut but incomplete responses in four (aspirate parasite density scores of 1+, scanty parasites), and no further improvement in three patients. Both apparent cures were healthy and had parasite-free bone-marrow aspirate smears after 6 months and were designated as definitive cures [4]. In both parts of this trial, all nonresponders and relapses received and responded to conventional parenteral pentavalent antimony therapy.

That atovaquone's effects were limited in scope and durability may reflect its apparent leishmanistatic rather leishmanicidal activity [1]. Because of the desirability of an oral regimen for treatment of kala-azar, we combined atavoquone with fluconazole, which also exerts clear but limited activity [3]. Although more active than atovaquone alone, the combination did not appear substantially more effective than fluconazole alone [3]. Nevertheless, there does appear to be a subset of patients with Indian kala-azar among whom oral atovaquone alone initially exerts measurable (albeit limited) antileishmanial activity. Because atovaquone plus antimony is active experimentally and is synergistic [1], it is possible that atovaquone may be more useful clinically if combined with conventional injectable antimony as an approach to using an oral agent to enhance antimony's declining efficacy [4]. In addition, because kala-azar has predictably been added to the list of AIDS-related opportunistic infections [5] and because patients with AIDS often receive fluconazole or atovaquone for other reasons, there may be a potential role for these drugs in suppressing reactivation of latent visceral infection and/or recurrences once primary therapy is completed [5].

Shyam Sundar, Prasanna Kumar, Manoj Makharia, Ashish Goyal, Michael Rogers, David Gibbs, and Henry Murray

Kala-Azar Medical Research Center, Banaras Hindu University, Varanasi, India; and Glaxo Wellcome, Research Triangle Park, North Carolina; Pfizer, Inc. and Cornell University Medical College, New York, New York, USA

Reprints or correspondence: Dr. Henry W. Murray, Cornell University Medical College, 1300 York Avenue, Box 130, New York, New York 10021.

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Infective Endocarditis Caused by *Staphylococcus hominis* After Vasectomy

Vasectomy is a common surgical procedure. Complications are infrequent and usually benign. Dan et al. [1] reported a case of endocarditis that occurred after a vasectomy that had been complicated by epididymitis. We describe a remarkably similar case.

A 28-year-old man was admitted to the hospital. He had been healthy until 7 weeks before admission, when he underwent a vasectomy. Two days after surgery he was struck in the groin by a snowball and the suture line was disrupted. He was treated with ampicillin 3 weeks later for fever and epididymal tenderness. Two weeks before admission he received oral erythromycin for a 5-day history of fever and myalgia. The fever abated, but 5 days later his temperature was 104°F. Findings on a chest radiograph and urinalysis were normal, but the WBC count had increased to 20,000/mm^3. He was thus admitted to the hospital.

There was no history of cardiac disease. Examination on admission revealed the following findings: blood pressure, 130/68 mm Hg; pulse, 112/min; respirations, 32/min; and temperature, 101.8°F. No petechiae were noted. No murmur or rub was evident on auscultation. The genitalia were normal. The hemoglobin level was 13.4 g/dL, and the erythrocyte sedimentation rate was 70 mm/h. Results of urinalysis and a chest radiograph were normal. On the second hospital day, *Staphylococcus hominis* was recovered from multiple blood cultures. Treatment with vancomycin, 1 g iv q12h, was begun.

On the third hospital day a 2/6 systolic ejection murmur and a 2/6 decrescendo diastolic murmur were audible at the left lower sternal border, and the patient's dyspnea had increased. An echocardiogram showed a large, 1.5-cm × 1.0-cm vegetation on the posterior aspect of the aortic valve. Rifampin, 300 mg p.o. q12h, was added to the treatment regimen.

Pretibial, sacral, and pulmonary edema were evident on the fifth hospital day and stabilized with furosemide therapy. On the 10th hospital day, a Björk-Shiley prosthetic aortic valve was placed. Evaluation of the resected aortic valve revealed a bicuspid valve with a 1.5-cm × 1-cm friable vegetation. Cultures of operative specimens yielded *S. hominis*. Because a 10-day course of treatment with vancomycin and rifampin failed to halt the valvular destruction and to sterilize the heart, further investigations were performed; the *S. hominis* isolate was found to be resistant to vancomycin. Intravenous penicillin and rifampin were substituted for the vancomycin-rifampin treatment regimen.

The patient was discharged on the 43rd hospital day to receive outpatient parenteral penicillin and p.o. rifampin. He was followed for 12 months without evidence of recurrent disease.

Complications of vasectomy are infrequent and generally mild. Infectious complications include wound infections, abscesses, epididymitis, and orchitis. In a review of selected studies from 1969 to 1974, Wortman [2] noted that a total of 369 infections had resulted from 2,496 vasectomies (14.8%). Randall [3] found a much higher infection rate, 32.9% of 94 cases, including nine infections due to coagulase-negative staphylococci. In another study [4], 15 of 122 vasectomies were followed by infections. In these case-series, infections were localized to the wound, testis, or epididymis. Our review of the English-language literature since 1966 produced only the case reported by Dan et al. [1]. The case we describe, therefore, is the second report of endocarditis complicating vasectomy.

Coagulase-negative staphylococci are recovered from the semen of 40% of healthy patients and from the prostatic fluid of 37% of patients with clinical prostatitis [5, 6]. Cultures of semen and urine specimens obtained before vasectomy have yielded the same bacteria as cultures of specimens from postoperative infections, and positive semen cultures are good predictors of infection [7, 8]. Although *S. hominis* and *Staphylococcus warneri* comprise <14% of clinical isolates of coagulase-negative staphylococci from body fluids, they are the predominant *Staphylococcus* species in the semen, prostatic secretions, and urethra [9, 10]. Thus *S. warneri* and *S. hominis* are potentially pathogenic organisms and may enter the body through breaks in the host-defense barriers of the genitourinary tract.

Nevertheless, staphylococcal infections complicating vasectomy are rare, and prophylactic antibiotics are not warranted. The cases described, however, should encourage circumspect monitoring of a patient who has undergone vasectomy and who subsequently has symptoms of systemic infection.

R. Blake Kessler, Robert C. Kimbrough III, and Stephen R. Jones

Department of Medicine, Legacy Emanuel and Good Samaritan Hospitals, Oregon Health Sciences University, Portland, Oregon

Reprints or correspondence: Dr. Stephen R. Jones, Department of Medicine, Legacy Good Samaritan Hospital, 1015 N.W. 22nd Avenue, Portland, Oregon 97210.

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