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EXTRACORPOREAL SHOCK-WAVE LITHOTRIPSY IN A PATIENT WITH MILD HEMOPHILIA

To the Editor: As Mulley states in his editorial (March 27 issue),[1] extracorporeal shock-wave lithotripsy is becoming the technique of choice for treatment of renoureteral lithiasis. Even though renal parenchymal damage occurs in all cases, renal subcapsular hematomas are the only major complications and can be treated conservatively.[2-4]

We recently treated a 68-year-old man for hypovolemic shock after extracorporeal shock-wave lithotripsy. He had a history of chronic obstructive pulmonary disease and laryngectomy for a benign vocal-cord tumor nine years before, with severe postoperative bleeding. He was seen for abdominal pain, lumbar hematoma, and weakness eight days after undergoing lithotripsy in another hospital because of lithiasis in the left renal pelvis. Preoperative study had been normal but did not include determination of the partial thromboplastin time; the procedure was carried out under epidural anesthesia and was unremarkable.[5] The patient was discharged 72 hours later in spite of hematuria and abdominal pain, which were considered to be "normal" after lithotripsy. On the eighth day he came to our hospital because of progressive deterioration.

He presented with hypotension and oliguria, which were treated by transfusion and infusion of fluids. Abdominal radiologic and echographic study showed a large extracapsular perirenal hematoma with extension into the retroperitoneum. Conservative treatment was carried out, with improvement. Progressive reabsorption of the hematoma was observed. The partial thromboplastin time was 10 to 15 seconds over that of control, and factor VIII was 25 percent, suggesting hemophilia. During the hospital stay, he had nosocomial pneumonia with respiratory failure that necessitated mechanical ventilation, and a urinary tract infection. The patient was discharged 46 days later with normal renal function.

Since the work of Chaussy et al.,[6] the indications for shock-wave lithotripsy have been expanded because of the low incidence of complications. In a recent series of 15 patients treated with lithotripsy, 4 (27 percent) had subcapsular hematomas that were detected by various techniques of renal imaging.[7] Our case of extracapsular hematoma occurred in a patient with mild hemophilia not detected preoperatively. We believe that extracorporeal shock-wave lithotripsy must still be considered a major intervention.

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IN VITRO AND IN VIVO RESULTS SUGGESTING THAT ANTI-SPOROZOITE ANTIBODIES DO NOT TOTALLY BLOCK *PLASMODIUM FALCIPARUM* SPOROZOITE INFECTIVITY

To the Editor: We have reported that a mouse monoclonal antibody directed against the circumsporozoite antigen and serum of mice immunized with recombinant and synthetic circumsporozoite peptides strongly inhibit the entry and development of *Plasmodium falciparum* sporozoites in hepatocyte culture.[1] Nevertheless, even though this inhibitory activity is often pronounced in culture, it is very rarely complete.

Since it can be argued that in vitro results are often of questionable relevance to an in vivo situation, we attempted to assess the extent of correlation between our in vitro results and observations made in patients in endemic areas. To examine this relationship, we collected serum from three persons living in three holoendemic areas in West Africa (Cameroon, Congo, and Mali). The samples had anti-sporozoite antibody titers that were the highest observed among samples from several hundred subjects studied thus far. The titers, directed against the sporozoite surface as determined by reactivity with "wet" preparations in indirect fluorescence assay,[2] ranged from 1:50,000 to 1:100,000, which is as high as or higher than corresponding titers of adults receiving as many as three infective bites per day (Druilhe P, et al.: unpublished data) and approximately 10 times higher than titers of mice with a high response to artificial peptides with Freund's complete adjuvant.

The subjects' serum samples were tested for their ability to block entry and inhibit development of *P. falciparum* sporozoites in a human hepatocyte culture system[3,4] and under the technical conditions described elsewhere.[1] Despite the high level of sporozoite surface–specific reactivity, the inhibitory activity of these samples in vitro was only 82 to 88 percent, indicating that 12 to 18 percent of the parasites in an inoculum were unaffected by the antibody.

The presence of *P. falciparum* ring forms in blood films of one of the subjects at the time that serum was obtained demonstrates that some sporozoites are able to evade the protective action of naturally acquired antibodies in vivo as well as in vitro, even when these antibodies are present at high levels. Therefore, these specific antibodies do not consistently protect against disease determined by invasion and multiplication of parasites in erythrocytes.

Antibodies elicited in humans by synthetic or recombinant peptides may be more effective than those produced in mice. Whether total protection will be achieved by vaccination with these preparations, in contrast to the incomplete protection observed under natural conditions of immunization, must await vaccine trials in humans. However, our results do suggest that an antigen or antigens specific to a single stage of the parasite may be inadequate as a vaccine designed for complete prophylaxis.

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