Hepatitis C and Human Immunodeficiency Virus Infection Following Ozone Autohaemotherapy

In the past three years, our institute has been consulted on three occasions involving patients who developed severe infectious complications associated with so-called ozone autohaemotherapy. Two patients developed hepatitis C virus (HCV) infection and one patient, a 35-year-old woman, acquired not only HCV but also the human immunodeficiency virus (HIV) following autohaemotherapy. This woman had attended a private practice because of menstrual cycle-dependent migraine, and the doctor recommended ozone autohaemotherapy. The woman was otherwise healthy and, in particular, had no risk factors for the acquisition of blood-borne viruses. All three patients received ozone autohaemotherapy over many months.

Ozone autohaemotherapy is a procedure in which 50–100 ml of blood is withdrawn from the patient and immediately treated with a gaseous mixture of oxygen and ozone. Subsequently, the blood is promptly reinfused. It is a form of therapy not officially sanctioned by the medical community, and scepticism considering its supposed usefulness in many different disorders is justified (1). Depending upon how the ozonization of blood is performed, there is a considerable risk of transmission of blood-borne viruses associated with the procedure.

In the three cases described here, patients' blood was withdrawn through a peripherally inserted intravenous catheter and was passed directly via a connecting piece into an empty infusion bottle. The oxygen-ozone mixture was taken from the ozone apparatus by pressing the tip of a great glass syringe onto the port of the apparatus. Subsequently, the gas mixture was passed into the infusion bottle containing the patient's blood using a large disposable injection needle. During the procedure the blood bubbles up, and sometimes the tip of the syringe is then visibly contaminated with blood.

The hygiene problem in the reported cases was that although a sterile infusion bottle, a sterile connecting piece, and a sterile disposable needle were used in every patient, the glass syringe was changed only one or two times per day or in the case of visible contamination with blood. Therefore, the blood of several patients is frequently ozonized with the same, possibly contaminated syringe. Contamination can occur during the passage of the gas mixture into the infusion bottle; it is also possible that the syringe tip can become contaminated when it is pressed onto the port of the apparatus, which, in turn, could have been contaminated through a contaminated syringe. Cleaning and disinfection of this port is extremely difficult, if not impossible.

We are convinced that the HCV infections in two patients and the HCV-HIV co-infection in the third case were caused by the fact that the glass syringe had not been changed between patients. Therefore, all of these infections could have been completely avoided if simple and inexpensive standard infection control practices had been followed.

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Reference


Septicemia due to Susceptible Enterococcus faecalis despite Prophylaxis with Trimethoprim-Sulfamethoxazole

Enterococci are becoming increasingly important nosocomial pathogens, commonly causing urinary tract infections, sepsis, and intra-abdominal and pelvic infections (1). The efficacy of treatment with trimethoprim-sulfamethoxazole (TMP/SMX) against enterococci that have shown in vitro susceptibility to this antimicrobial agent is a matter of controversy.

We present the case of a 20-year-old male who had received bone marrow transplantation because of a lymphoblastic non-Hodgkin’s lymphoma. After discharge from hospital, he received oral TMP/SMX (160 mg TMP plus 800 mg SMX; Roche, Germany) twice daily for three consecutive days per week for prophylaxis of Pneumocystis carinii pneumonia. During a prophylaxis cycle started ten weeks after bone marrow transplantation, the patient developed fever of 39°C and remained febrile over three days. Pancytopenia with a total leuko-
cyte count of 2500/µl (normal, 4000–11,000/µl) was noted, but there were no clinical signs of peritonitis or urinary tract infection.

*Enterococcus faecalis* was isolated from a single blood culture drawn from a central venous catheter approximately 12 hours after the last dose of TMP/SMX. As prophylaxis had been maintained during the febrile period, it was suspected that TMP/SMX was not effective against this *Enterococcus* isolate. However, the isolate proved susceptible to TMP/SMX when tested in Mueller-Hinton broth (BBL, USA) using the broth microdilution method according to NCCLS criteria. The minimum inhibitory concentration (MIC) was 0.0625 µg/ml for TMP tested in a TMP:SMX ratio of 1:19 (bacteria with TMP MICs ≤ 2 µg/ml are regarded as susceptible when tested in this ratio). By the agar disk diffusion test (Diagnostic Sensitivity Test Agar; Oxoid, UK), the isolate was shown to be susceptible to TMP/SMX, ampicillin, piperacillin, and vancomycin. Antibiotic therapy was switched to intravenous piperacillin and vancomycin, which led to the patient's rapid recovery.

Methicillin- and TMP/SMX-resistant *Staphylococcus epidermidis* had also been isolated from the same blood culture. *Staphylococcus epidermidis* has been described as a frequent cause of catheter-related bacteremia (2), although this pathogen belongs to the physiological skin flora and isolates from blood cultures are often contaminants. In contrast, *Enterococcus* species are part of the intestinal flora (1), and blood culture isolates are much more likely to represent the etiologic agents of septicemia than contaminants. It seems probable, therefore, that our *Enterococcus faecalis* isolate was likely to have caused the patient's state of fever. However, the possibility that the *Staphylococcus epidermidis* isolate was also involved in pathogenesis cannot be ruled out.

One early study demonstrated susceptibility of enterococci to TMP/SMX in Mueller-Hinton broth in vitro but also found that this agent's activity varied considerably with the manufacturer of the broth media (3). Other studies have demonstrated that addition of thymidine, thymine, and "folates" to testing media leads to reduction of the activity of TMP/SMX in vitro (4–6). Another study found that TMP/SMX was not reliably bactericidal against enterococci in vitro (7).

Different conclusions have been drawn from these results. Some authors have expressed doubt about the reliability of in vitro susceptibility testing of enterococci to TMP/SMX (4, 5, 7). Inhibition of the activity of TMP/SMX has been found at concentrations of folinic acid that are achievable in serum or urine (4). However, another study concluded that normal "folate" concentrations in serum and urine were not sufficient to cause a therapeutically important reversal of trimethoprim activity (6).

The results of two studies in animals seem to favour the first result. Therapy of infection due to enterococci susceptible in vitro to TMP/SMX was not effective in a rat model of experimental endocarditis (8) or in a mouse model of peritonitis (9), despite adequate TMP/SMX levels in serum. The results of these animal studies are supported by Goodhart's (10) clinical report of two patients who developed bacteremia due to in vitro TMP/SMX-susceptible enterococci, despite therapy with TMP/SMX that had been administered because of uncomplicated urinary tract infections. In one of these patients, isolation of enterococci from urine and sputum specimens had led to the administration of TMP/SMX; the other patient had developed urinary tract infections on several occasions, once due to enterococci.

The oral dosage of 160 mg TMP combined with 800 mg SMX twice daily leads to serum levels of both substances that far exceed the determined MIC of our *Enterococcus faecalis* isolate (11). Our case corresponds to the two clinical cases reported by Goodhart (10), suggesting that TMP/SMX is not adequate for prophylaxis and therapy of serious enterococcal infections.

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References