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Meeting Highlight

Second International Symposium on Febrile Neutropenia, Brussels, 14–16 December 1995

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FEBRILE NEUTROPENIA is a common complication during the treatment of cancer patients. In the last 30 years much progress has been made in this field by continuous clinical and microbiological research. The mortality rate of febrile neutropenia has decreased from approximately 90% in the early 1960s to below 5% in the 1990s.

The second International Symposium on Febrile Neutropenia, organised by J. Klastersky and his scientific committee, offered an excellent overview of current opinions, recent controversies and future strategies.

In order to deliver and adjust empiric antibiotic therapy and to allow continuous medical surveillance, cancer patients with fever and neutropenia normally remain hospitalised until fever, symptoms and granulocytopenia have resolved. It is obvious that some patients are at a very low risk for delayed response or medical emergency. In order to increase patient comfort and to save costs, efforts have been made to identify subgroups of patients which can be safely treated as outpatients. J. Talcott presented his predication model, used at the Dana-Farber Cancer Institute, to define patients at risk with fever and neutropenia. The risk assessment is based on clinical variables which can be assessed at the first day of presentation. He found three groups of patients at risk for medical complications: inpatients who are already ill before the infection (group I), outpatients with serious concurrent morbidity (group II) and outpatients with uncontrolled cancer (group III). Other patients were considered as low risk (group IV). To validate this risk assessment model, a prospective two-centre study with blinded review of risk factors and endpoints was conducted. A population of 444 consecutive cancer patients treated as inpatients with broad spectrum, intravenous antibiotic therapy was analysed. From 119 serious medical complications, 114 occurred in patients from groups I–III. While multiple complications and death were common among patients in groups I–III, no group IV patient had more than one complication or died. In multiple logistic regression analysis, the predefined risk groups were the strongest independent predictors of complications.

J. Talcott's predictive model seems to be a valid tool for identifying at the onset of infection those patients who are appropriate candidates for early discharge and continuation of treatment in their familiar home environment. As E. Rubenstein stated, patients, family members and care givers must be instructed to comply strictly with the antibiotic therapy, to monitor temperature and to observe symptoms of worsening infection. Close ambulatory surveillance is important, particularly during the first days of infection, in order to assess the response to treatment. It is still controversial if oral antibiotic regimens are equivalent to intravenous regimens. Randomised studies to answer this question are ongoing.

Several posters and oral presentations showed the results of randomised comparisons between newer cephalosporins such as Cefepime and Cefpirome. They might be as effective as Ceftazidim, the most investigated cephalosporin in neutropenic infections. During the discussions about the trends of empiric antibiotic therapy, some provocative questions were raised: Do the aminoglycosides have any significance in the initial treatment? Should the initial regimen be continued without escalation until day 5 or 6, even if fever persists? Can the glycopeptides be withheld until the antibiogram proves the necessity of their use?

Another issue discussed at the meeting was the emerging antibiotic resistance of bacterial pathogens. P. Moreillon showed how micro-organisms have developed protection against antibiotics by several different mechanisms. The resistance mechanisms have been studied in detail for most of the frequently used antiobiotics. A decrease of cell wall permeability has been found to reduce the efficacy of beta-lactams, aminoglycosides and quinolones, where as an active efflux pump has been shown to export erythromycin, tetracyclines and quinolones out of the cell. Most of the antibiotics are vulnerable to inactivation by bacterial modification of the molecular target-structure. A direct enzyme-linked inactivation of the antibiotic has been found to build up resistance against beta-lactams, aminoglycosids, erythromycin and tetracyclines. In addition, bacteria have evolved sophisticated genetic systems which enable them to transfer resistance determinants to other strains. This mechanism can select for resistance against different antibiotics in parallel.

The emerging problems with multiresistant strains of

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Staphylococcus and *Enterococcus* force the reluctant use of vancomycin, and the coordination of all efforts against an expansion of these dangerous pathogens. A prevention of such a dreadful event includes a proper policy of antibiotic usage, strict measures of infection control and a prospective epidemiological follow-up. Patients colonised with multi-resistant strains should be identified, isolated and decolonised. Subsequent hand washing and disinfection of stethoscopes and clinical thermometers are important contributions to prevention.

It is of great importance that cancer therapists consider these subjects and adapt their antibiotic policies to the requirements of resistance prevention. Otherwise the great success of the last 30 years in the treatment of infection in neutropenic patients is in danger, and uncontrolled infections may evolve again to be a life threatening event for cancer patients.