
The importance of follow-up biopsies of clinically suspicious lymphadenopathy in nodular lymphocyte predominant Hodgkin’s lymphoma

Although nodular lymphocyte predominant Hodgkin’s lymphoma (NLPHL) is an indolent disorder, clinically suspicious lymphadenopathy commonly develops during follow-up. In our series of 100 cases of NLPHL, fifteen cases with sequential biopsies were identified. The vast majority showed reactive changes only, emphasizing the importance of histologic sampling of lymphadenopathy in NLPHL patients.


Letters to the Editor

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References


Thalidomide plus oral melphalan for advanced multiple myeloma: a phase II study

Thalidomide exerts synergistic or additive effects when combined with other drugs. This study reports the toxicity and efficacy of the combination of thalidomide plus oral melphalan in 27 patients with advanced multiple myeloma. We found that this combination induces a high response rate and a long progression-free survival without significantly increasing thalidomide-related toxicity.

There are few therapeutic options available for patients with relapsed-refractory multiple myeloma (MM). Since the first report by Singhal et al., attention was focused on thalidomide or thalidomide combined with dexamethasone or chemotherapy. Unfortunately, there was a significant incidence of side effects, mainly deep venous thrombosis and myelosuppression, in association with combination therapy.2-3 We report our experience using thalidomide and oral melphalan in patients with advanced MM.

From May 2000 to July 2002 in our tertiary care institute and in the main medical institutions of the Marche region (Italy) 27 patients with relapsed-resistant MM were treated with thalidomide plus melphalan. Patients with poor performance status and/or cardiopulmonary, renal and liver diseases were not excluded whereas patients with severe mental disorders or severe peripheral or central neuropathy were not enrolled. All patients signed a written informed consent form. The starting dose of thalidomide was planned to be 100 mg p.o. daily at bedtime, escalated weekly by 100 mg increments up to a maximum dose of 600 mg, in the absence of severe side effects. Thalidomide was stopped only because of severe side effects or disease progression. Melphalan was administered intermittently at a dose of 0.20 mg/kg/day p.o. for four days every 28 days for at least one course after greatest response was achieved or until severe toxicity developed. No patients received antithrombotic prophylaxis. Responses to therapy were assessed as reductions of paraprotein in serum and/or urine of at least 25%, 50% and 75% without the appearance of new skeletal lesions or an increase in bone marrow plasma cells. Complete response (CR) was defined according to EBMTR/IBMTR criteria.4 Toxicity was assessed according to the Word Health Organization (WHO) criteria.

Forty percent of patients were aged >70 years; more than 2 prior regimens had been administered to 56%, prior high-dose therapy with stem cell support to 41% and prior therapy with melphalan to 96% of patients.2,3 Microglobulin concentration > 3 mg/L in 63% and the disease had been present for longer than 3 years in 30% of patients.

Paraprotein decreases of ≥50% and ≥75% were obtained in 59% and 15% of patients, respectively (Table 1). Remarkably, 3 out of 4 patients who had a maximal response had no monoclonal paraprotein detectable by immunofixation. The median time to remission was 6 weeks. The main side effects were constipation (82%), somnolence (41%), fatigue (22%), sensory peripheral neuropathy (56%), deep venous thrombosis (11%) and grade 3 leukopenia (30%). However, no severe infections occurred. After a median follow-up of 15 months (range 6-32), 9 patients (33%) had disease progression and 6 (22%) had died.

The 2-year progression-free survival (PFS) and overall survival (OS) were both 61%.

As a single agent thalidomide produces an overall response rate of 30% and a 2-year event-free survival (EFS) of 20% in patients with heavily pretreated MM. Some studies have demonstrated that thalidomide may restore the sensitivity of myeloma cells to apoptosis induced by drugs, preventing the interaction between tumor cells and stromal cells.5-6 We found