



Introduction

H. Joensuu*

*Department of Oncology, Helsinki University Central Hospital, Haartmaninkatu 4,
FIN-00029 Helsinki, Finland*

This supplement presents the proceedings of a symposium held in Helsinki, Finland, on 7–8 September 2001 that focused on the treatment of gastrointestinal stromal tumors (GISTs) with imatinib, formerly known as STI571 or CGP57148, a tyrosine kinase inhibitor. The first patient to receive imatinib for treatment of GIST (March 2000) showed excellent response with minimal toxicity [1]. This single-patient proof-of-concept study was soon followed by larger studies, which confirmed that imatinib is the first effective systemic therapy for metastatic and locally inoperable GIST [2,3].

Fifteen to 50% of GISTs are metastatic at presentation with the liver and the abdominal cavity the most common sites. The median survival has been only about 10 to 20 months in metastatic disease [4]. Unlike leiomyosarcomas, GISTs are notoriously resistant to conventional chemotherapy, and no effective systemic therapy has been available for GISTs.

We begin with presentations concerning the significance of tyrosine kinases in solid tumors, with discussions focused on the molecular and cellular activity of these enzymes. These are followed by discussions on the structure, molecular design, and pharmacology of imatinib, and how this new tyrosine kinase inhibitor specifically affects malignant cells. The clinical presentation of GIST is discussed, including pathology and diagnostic criteria. The first clinical results of imatinib treatment are described, as is the role of positron emission tomography in oncology, and specifically, GIST. Following an overview of related issues by the experts present at the symposium, the use of imatinib for chronic myelogenous leukemia is discussed along with a review of the first 3 years of investigations

with imatinib. Finally, there is discussion of the use of imatinib in advanced soft tissue sarcoma.

Although many important questions remain open, it is already clear that imatinib is a significant step forward in the systemic treatment of GIST. Imatinib is an excellent example of rational and highly targeted drug design. It supports the hypothesis that very effective, well-tolerated anti-cancer drugs can be developed through modern technology for even the most therapy-resistant human cancer types, based on a detailed understanding of their molecular pathogenesis.

Since the meeting in Helsinki, the ongoing clinical experience with imatinib has demonstrated that it is now possible to successfully manage patients with GIST with a new oral medication [2,3]. As our understanding of GIST rapidly evolves and we continue to explore novel treatment strategies for it, the prospect of sharing new findings with our colleagues is an exciting one indeed.

References

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* Tel. +358-9-471 73208; fax +358-9-471 74202.

E-mail address: heikki.joensuu@hus.fi