# Letter to editor

**CML: Imatinib mesylate (Glivec) or something else?**

Dear Sir,

Chronic Myeloid Leukaemia (CML) is characterized by uncontrolled proliferation of myeloid cells. It accounts for 15.0% of Leukaemias and occurs most often in middle age with a slight male predominance. CML is best differentiated from other myeloproliferative disorders by detection of the t(9;22) Philadelphia (Ph) translocation. The translocation results in the formation of a *bcr-abl* fusion gene that gives rise to the expression of a BCR-ABL fusion protein (210-KD) with tyrosine kinase activity. This leukaemia specific fusion protein is necessary and sufficient to drive the leukaemia process in early chronic phase, though additional genetic lesions are important in disease evolution. It has also been reported that *bcr-abl* sequences occur even in normal population.[1,2]

The traditional curative therapy for CML was bone marrow transplantation but recently introduction of imatinib mesylate (Glivec) has become the first line therapy for the disease at all ages. Imatinib mesylate, a specific tyrosine kinase inhibitor has revolutionized the management of CML. It is more effective than the previous gold standard treatment of alpha-interferon. The drug is also effective in patients in accelerated phase and blast crisis.

However, the results of imatinib therapy beyond 30 months are not known and increasing number of patients are becoming resistant to imatinib. Thus, a supplementary therapy is required in patients who are unsuitable for or unresponsive to imatinib treatment. Existing literature has demonstrated that cytotoxic T cells can be generated against BCR-ABL peptides in normal HLA-A3+ subjects and CML patients.[3,4] In this context, further exploitation of the immune system will improve understanding of the potential benefits of BCR-ABL peptide immunization of CML patients.

**REFERENCES**


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