

Implications of High Grade Lymphoma Arising in Chronic Myeloid Leukaemia

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Letter to the Editor

There have been several reports of different types of Philadelphia (Ph) negative lymphomas arising in chronic myeloid leukaemia (CML) [1-5]. These are usually of T- cell origin but also include B- cell lymphomas ranging from mantle cell lymphoma, follicular lymphoma and diffuse large B- cell lymphoma(DLBCL).These however have all been described in the pre- tyrosine kinase (TKI) era and the lymphomas have been of extramedullary origin.

I present the case of a 64 year old man with CML who had achieved a major molecular response after one year of imatinib therapy. He then presented with cachexia and obstructive jaundice. The blood count was stable WBC 2.9×10^9 Neutrophils $1.1 \times 10^9/l$ Hb 11.4 g/l Platelets $241 \times 10^9/l$. The BCR/ABL ratio was 0.4% on a marrow sample. The bilirubin was 97 $\mu\text{mol/l}$ GGT 1182 U/l ALT 242 U/l ALK 1320 U/l. Liver and spleen metastases, multiple skeletal lytic lesions, mesenteric and paraortic lymphadenopathy were detected on CT scan. There were also bilateral pleural effusions and he later developed gross ascites. The marrow architecture was completely effaced by a diffuse infiltrate of immature lymphoma cells. The liver biopsy revealed infiltration by DLBCL and lymphoma cells were present in the pleural fluid. The lymphoma cells in the marrow demonstrated the t 2:18 and there was no evidence of t 9:22 by FISH. A diagnosis of denovo stage IV DLBCL with marrow involvement was made.

The patient therefore had two unrelated malignancies even though prima facie the presentation was typical of a lymphoid transformation of CML. It is well known that TKI therapy can select resistant clones and a focal clonal escape from the original CML population in the marrow resulting in the lymphoma could have occurred. If this were a lymphoid transformation, the TKI would have had to be changed to a

more potent TKI besides inducing with acute leukaemia- type therapy and backing this up with an allogeneic stem cell transplant. However, since these were two distinct malignancies, imatinib was continued and the lymphoma was treated with R-HyperCVAD/MA although the patient eventually succumbed.

A hypothesis is that a) the original malignant clone had arisen in a very early cell not committed to either lineage b) that part of the clonal population had acquired a t 9:22 and had then undergone differentiation to develop myeloid characteristics consistent with CML c) that another part of the original clone had acquired the t 2:18 and subsequently differentiated along the lymphoma route.

References

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