Leukocyte superoxide dismutase activity in patients with chronic myeloid leukemia

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Introduction

Chronic myeloid leukaemia (CML) is a myeloproliferative disorder of the hematopoietic cell and is associated with a characteristic chromosomal translocation called the Philadelphia chromosome. Clinically, CML is often divided into three phases; CML typically begins in the chronic phase (CML-CP) and in the absence of intervention and over the course of several years progresses to an accelerated phase (CML-AP) and ultimately to blast crisis. Blast crisis is the terminal phase of CML and clinically behaves like acute leukemia[1]. Free radicals can be key contributory agents in a number of human diseases, including cancer and leukaemia[2,3]. These free radicals can be generated within the cell not only by external sources of radiation but also within the body as a by-product of normal metabolic processes which include the electron transport chain, drugs, pollutants, and chemicals including toxins, collectively termed as xenobiotics. Thus, antioxidants which balance the oxidative stress state represent a major line of defense in regulating the overall true state of health[2]. Antioxidant enzymes such as superoxide dismutase (SOD) can directly counter the oxidants and may protect cells against oxidative stress. Studies show that cancer cells have abnormal activities of SOD enzymes when compared to an appropriate normal counterpart. There are no available publications regarding the relationship between leukocyte SOD enzyme activity in CML and its progression. The present study was planned to propose leukocyte SOD activity as a possible biomarker for oxidative stress in CML and its correlation with the progressive phases of CML.

Methods

This study included 83 CML patients and 50 age- and gender-matched healthy volunteers. Leukocyte SOD activity was measured by the spectrophotometric method[4].

Results

At the time of diagnosis, the mean leukocyte SOD activity in CML, CML-CP and CML-AP were significantly higher (p-value < 0.001, p-value < 0.01 and p-value < 0.001, respectively) than in healthy volunteers (Table 1). CML-CP patients who converted into CML-AP had significantly higher (p-value < 0.05) leukocyte SOD activity than CML-CP patients who did not convert into CML-AP (Table 2).

Table 1 - Leukocyte superoxide dismutase activity in CML, CML-CP, CML-AP patients and healthy subjects

Groups	n	Leukocyte SOD activity (U/mg protein)
Healthy Subjects	50	11.69 ± 4.34
CML	83	16.99 ± 7.61**
CML-CP	62	15.94 ± 7.52***
CML-AP	21	19.94 ± 7.26**

CML: Chronic myeloid leukaemia; CML-CP: Chronic myeloid leukaemia - chronic phase; CML-AP: Chronic myeloid leukaemia - accelerated phase; SOD: superoxide dismutase
Values are mean ± SD; **p value < 0.0001; ***p value < 0.001

Conflict-of-interest disclosure:
The authors declare no competing financial interest

Submitted: 8/31/2012
Accepted: 9/4/2012

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DOI: 10.5581/1516-8484.20120097

Table 2 - Progression of CML-CP patients to CML-AP and leukocyte superoxide dismutase activity

	n	Leukocyte SOD activity (U/mg protein)
CML-CP not progressed to CML-AP	34	15.14 ± 6.26
CML-CP progressed to CML-AP	15	19.83 ± 9.75*

CML: Chronic myeloid leukaemia; CML-CP: Chronic myeloid leukaemia - chronic phase; CML-AP: Chronic myeloid leukaemia - accelerated phase; SOD: superoxide dismutase
Values are mean ± SD; *p value< 0.05

Discussion and conclusion

The mean leukocyte SOD activity was significantly different in CML, CML-CP and CML-AP patients when compared to controls (Table 1). CML-CP patients who converted into CML-AP had higher leukocyte SOD activity than CML-CP patients who did not convert into CML-AP (Table 2). Superoxide dismutase is a major cellular defense device against superoxides in the cells[5]. A SOD enzyme is dimeric antioxidant enzyme responsible for the quenching of superoxide radicals which are released in various metabolite pathways. Altered leukocyte SOD activity has been reported in various cancers including leukemias[2,3,6]. The elevated leukocyte SOD activity may be due to altered gene expression in hematopoietic cells. It could be concluded that leukocyte SOD activity reflects oxidative stress in CML patients, and may be used as an indicator for oxidative stress related to disease progression. Further studies are needed to confirm the precise role of the SOD enzyme in oxidative stress in CML pathobiology and its progression.

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