Letter to the Editor

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Superoxide Dismutase as a Prognostic Criterion in Nephrotic Syndrome

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Dear Sir,

It has long been speculated that certain types of human renal disease, in particular those affecting the glomerulus, appear to be mediated by immunological mechanisms. However, little is known about the particulars of this mediation. There is evidence that phagocytes accumulate in the glomeruli and are the source of the mediators that are responsible for much of the glomerular injury. What these mediators are, on the other hand, is not clear. Recently, oxygen radicals have been reported to be responsible for glomerular injury and subsequent proteinuria [1, 2]. Several recent studies indicated that the superoxide radical (O_2^-) played a major role in the inflammatory process, and that the enzyme, superoxide dismutase (SOD) might prove to be highly effective in the management of glomerulonephritis [3, 4]. Superoxide (O_2^-) is an oxygen radical, which is formed by the addition of one electron to the oxygen molecule. Some superoxide is formed 'accidentally', when certain molecules in the body react directly with oxygen to form superoxide. Examples include the catecholamines, tetrahydrofolates etc. In addition, some superoxide is formed as a by-product of certain immunological processes. For instance, activated phagocytes generate large amounts of superoxide as a part of the mechanism by which foreign organisms are killed. During chronic inflammation, this normally protective mechanism becomes damaging, and is in part responsible for the tissue damage which accompanies the inflammatory process. As is well known, SOD converts superoxide to hydrogen peroxide (H_2O_2).

$$2\,O_2^- + 2\,H^+ \xrightarrow{SOD} H_2O_2 + O_2$$

Based on the above, we postulated that SOD levels could be used as a measure of inflammation and consequently as a prognostic criterion of the nephrotic syndrome. In this communication the preliminary data from our study are presented.

The study group consisted of 20 children with nephrotic syndrome in relapse. Ten children were steroid-responsive with good prognosis and formed the first group. The remaining 10 children, which were steroid-resistant, formed the second group. When we compared the SOD levels of these groups, we found a statistically significant difference ($t = 2.17$, $p < 0.05$) as shown in table 1.

Our preliminary data clearly demonstrate that SOD levels in the steroid-resistant group are higher than in the steroid-responsive group. In 1984, Halliwell and Gutteridge [5] emphasized that oxidative damage could be a consequence as well as a cause of tissue injury. With this in mind, our data suggest that oxidative damage is important in the pathogenesis of the nephrotic syndrome and that, if oxidative damage is excessive, SOD will increase as a compensation and protection. Furthermore we also suggest that in the nephrotic syndrome with steroid resistance, oxidative damage is higher than the steroid responsive type and consequently SOD may be used as a prognostic criterion of steroid response in the nephrotic syndrome.

Table 1. Erythrocyte SOD levels in groups 1 and 2

Patient No.	Erythrocyte SOD (U/g Hb) levels of group 1	Erythrocyte SOD (U/g Hb) levels of group 2
1	2,040	2,800
2	3,880	1,351
3	1,555	3,890
4	1,493	1,000
5	2,486	1,567
6	1,555	2,631
7	1,836	5,600
8	1,763	4,117
9	1,557	5,932
10	1,475	3,500
Mean ±SD	1,964.0 ±742.9	3,238.8 ±1,700.4

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