Another Look at Renal Function and the EDTA Treatment Process

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Abstract

Possible renal damage has often been cited as one of the cardinal contraindications to the use of EDTA as a therapeutic modality. In contrast, a few recent research reports suggest that improvement rather than renal compromise may in fact be a consequence of such a treatment process. This report is designed to add to the body of information which supports a relative lack of potential nephrotoxicity. Additionally, the point should be made that this is a unique sample for studying potential nephrotoxicity in that it is composed exclusively of essential hypertension patients. Using traditional statistical procedures, and within the limits of this experiment, it is reasonable to conclude that EDTA chelation does not contribute to nephrotoxicity.

Introduction

A review of the EDTA chelation literature suggests that a significant number of papers have reported research relevant to the issue of whether, and to what degree, one would be concerned with possible nephrotoxicity as a result of EDTA chelation treatment. Two of the most recent and comprehensive reviews, Halstead[1] and Cranton[2], conclude: (a) under certain circumstances there is a potential for renal damage in the use of ED-

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TA, as with the use of many other substances, but (b) where such impairment has been documented it has been traced almost exclusively to excessively rapid EDTA infusion and/or inordinately large doses of the chelate.

Method

The study was catalyzed by a fascinating report in a recent issue of N. Engl. J.Med.[3], which suggested a possible relation between lead toxicity and essential hypertension. The 28 subjects for this study were systematically drawn from 127 volunteers to a study designed to explore the possible effectiveness of EDTA chelation as a treatment for essential hypertension. Criteria for inclusion in the sample group were that the subject be over the age of 40, ambulatory, have a history of hypertension longer than one year, have completed an extensive history packet and screening lab work, demonstrate a 5-fold increase in lead and cadmium as a result of an EDTA challenge chelation, and finally have a fasting serum creatinine score less than 1.7 mg%.

The treatment protocol for all 28 subjects included an extensive battery of biochemical, psychological and physiological tests which will be reported elsewhere. For the purpose of this research report, however, each person was measured three times for serum creatinine and blood urea nitrogen (BUN) levels, which are two of the most commonly recognized minimally invasive estimates of renal function[4]. The latter will be discussed in a subsequent report. Specifically, blood samples were drawn subsequent to the first, tenth and twentieth EDTA infusions. By this

Table 1
Distribution of Serum Creatinine Scores
Before and After Ten and Twenty Chelation Infusions with (Percentages)

Creatinine Groups*		Pretreatment	After Ten	After Twenty
0.6-0.8	6	(21.5)	3 (10.7)	4 (14.2)
0.9-1.1	11	(39.3)	19 (67.9)	16 (57.2)
1.2-1.4	9	(32.1)	5 (17.9)	**7** (25.1)
1.5-1.7	2	(7.1)	1 (3.6)	1 (3.6)
TOTALS	28	(100)	28 (100)	28 (100)
Mean Scores	:	1.08	1.06	1.05
SD		.24	.17	.21
Minimum /Maximum		0.6/1.5	0.7/1.5	0.7/1.7**

* Laboratory coefficient of variation is 17.4% at the 1.0 mg% level. ** Attention is directed to the one subject whose creatinine level rose from 1.4 mg% at pretreatment to 1.7 mg% at post 20 chelations. A review of other available clinical data about the patient gives no readily apparent clues to the meaning of the above change.

procedure it became possible to assess the serial effects of the infusions on renal functioning.

The therapeutic treatment regimen consisted of a series of 20 EDTA infusions administered at approximately weekly intervals, where possible, over a period of 20 weeks. The maximum time span for a very few subjects ran up to 38 weeks. Each infusion consisted of 3 gms EDTA (Keylate: Edetate Disodium: The Key Co.), 15 gms ascorbic acid buffered in sodium bicarbonate (Bronson Pharmaceuticals), 800 mg magnesium chloride, 40 mg procaine, and 1000 units heparin delivered in 500 cc sterile deionized water and intravenously infused over a period of 3-5 hours. Additionally, each subject was given three Insurance Formula (Bronson Pharmaceuticals) tablets per day.

Results

Table 1 summarizes the distribution of creatinine scores at three major time points in the study; pretreatment and after 10 and 20 EDTA chelation infusions. A number of points become evident as a result of a brief overview of these data. First, on the basis of traditional norms (0.5-1.5 mg%) *no* subject in this group could be described as suffering from obvious renal impairment.

However, according to Duarte[4], a more useful set of nearly physiologically ideal ranges appear to be closer to 0.8-1.2 and 0.6-0.9 mg% for males and females respectively. Using the latter cutoff points, as many as 30 percent of the patients in this group could clearly be regarded as demonstrating decreased renal efficiency. To be more precise, Duarte supports his use of more narrow ranges by noting that for every 0.1 mg% above 1.0 mg% there is approximately a 7.5% renal impairment. This might suggest that 1.0 mg% could be conceptualized as a hypothetical "ideal score." Viewed in this light, Table 1 shows a number of subjects with varying levels of renal impairment.

Finally, utilizing traditional comparison methodologies of mean differences between groups followed by t tests of significance, these data indicate that treatment with EDTA chelation as described does not significantly compromise renal function as measured by serum creatinine. Specifically, a comparison of the pretreatment and post 10 treatment serum creatinine means demonstrates no significant differences exist ($t=.39$, $p >.20$) between those groups. The findings are essentially the same between post 10 and post 20 chelations ($t=.68$, $p >.20$), and between pre and post 20 ($t = .38$,

p>.20). Accordingly, the chelation procedure described above appears to live up to a basic tenet of medicine, "do no harm."

Discussion

In evaluating the nephric effects of EDTA chelation, the point should routinely be underscored that we are dealing with a multi-factorial treatment modality, which uses vitamins, minerals, a local anesthetic, an isotonic carrier, and EDTA. Obviously research utilizing such a multifactorial therapy has the disadvantage of making it difficult to identify and rank order the active ingredients in the treatment in terms of their effects on the patient. Having said the above, these data as reported seem to indicate that the weekly infusion of the EDTA combination solution described above is safe in a group of hypertensive patients. It is reassuring to note that such a conclusion is consistent with the data of previous research done on a large group of over 300 private practice patients as reported by McDonagh et.al.[3]. While one of the most convincing designs for documenting treatment effectiveness is obviously the double-blind approach, the above methodology would appear to be quite adequate since the tenor and philosophy of this report is to check for potential nephrotoxicity.

Having documented that the treatment as described appears not to be nephrotoxic, is there evidence to suggest that it may in fact be salutary? Using Duarte's more narrow physiologically ideal range as a point of departure, we conclude that, after ten treatments, these patients would appear to be (renally) healthier than before treatment as demonstrated by the decrease in the variability of the creatinine scores. Viewed another way, scores of patients that were below 1.0 mg% at pretest tended to move up toward 1.0 mg% after 10 treatments, and scores above 1.0 mg% to move down toward 1.0 mg%. (Table 1) This trend is demonstrated by the drop in SD from 0.24 at pretest to 0.17 following ten infusions. The somewhat less familiar general test for the equality of variances suggested by Choi and Wette (1972)[6] even more dramatically demonstrates the decrease in variance between pretest and post 10 chelations by suggesting that the decrease in variance is significant to the .025 level with a correlation of .39. In other words, these data suggest that renal function is actually improved by EDTA chelation up to 10 infusions.

The same movement toward Duarte's ideal serum creatinine score of 1.0 mg% does not continue between ten and twenty chelations as evidenced by a statistically insignificant increase in the standard deviation from 0.17 to 0.21. This lack of significant change is again borne out by the Choi and Wette test for equality of variances, where the correlation is 0.15 (p> .05).

Acknowledgements

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