

Original Articles

LIVER IRON CONCENTRATION IN ADULT MALES

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Abstract

Iron was estimated in 64 specimens of liver tissue obtained from autopsies of adult males who died from various causes. The values ranged from 13 to 474 ug per 100 mg dry weight of tissue with a mean of $118 \pm \text{S.E. } 10.3$. The highest values were found in subjects who died from malignant diseases and the lowest in those who died from trauma. Only 5(8.2%) subjects were found to have liver iron below the minimum reported control values.

Introduction

Measurement of liver iron provides a direct estimate of the iron stores present in the body. As no data on tissue iron was available in Pakistan a pilot study was conducted to evaluate the iron stores by estimating iron from autopsies of adult males.

Material and Methods

A total of 64 liver specimens were obtained from a similar number of autopsies of adult males. Their ages and cause of death were recorded. Liver iron was estimated by the method described by Barry and Sherlock (1971). This method measures total liver iron, expressed as ug per 100 mg dry weight of tissues.

Results

The mean age of the 64 subjects from whom the samples were obtained was 28.5 years. Three subjects were less than 20 years of age, 39 were between 20 and 30 years, 15 were between 31 and 40 years and in 4 subjects the age ranged between 41 and 50 years.

The causes of death included cardiovascular diseases, trauma, acute and chronic infections, visceral congestion, malignancy and liver diseases. The number of cases grouped according to their cause of death are listed in table I.

The mean $\pm \text{S.E.}$ liver iron concentration in 64 liver specimens was 118 ± 10.3 ug per 100 mg dry weight. The values found in 10 control subjects by the authors of the method adopted in present study (Barry and Sherlock, 1971), ranged between 34.4 and 136.4 ug per 100 mg dry weight, with a mean of 77.7 ± 9.7 .

Table I: Number of Cases According to Cause of Death

Cause of Death	No. of Cases
Cardiovascular Diseases	15
Infection: Chronic	6
Acute	6
Trauma	10
Visceral Congestion	14
Malignancy	6
Liver Disease	4
Unknown	3
Total:	64

The frequency distribution of the values obtained for liver iron is shown in table II. Of the 64 cases, 5(8.2%) were found to have liver iron less than the minimum of the control range, (that is 34.4 ug/100 mg) and in 17 (26.5%) cases the liver iron was above the maximum of the control values (i.e. 136.4 ug/100 mg). The five cases with low liver iron were those who died of pneumonia, hemorrhage and shock, perforated gastric ulcer with peritonitis, tuberculosis with osteogenic sarcoma and bleeding. The cause of death in the fifth case could not be ascertained.

Table II: Frequency Distribution of Liver Iron

Liver Iron (ug/100 mg dry weight)	No. of Cases
Less than 50	8
50—70	7
71—90	16
91—110	8
111—130	6
131—150	6
151—170	3
171—190	2
191—210	1
211 and above	7
Total:	64

Of the 17 cases with high levels of liver iron, the cause of death in 5 was cardiovascular disease, 4 visceral congestion, 3 malignancy, 2 chronic infection, one trauma and one liver disease. The cause of death was unknown in one case. Table III shows the mean $\pm \text{S.E.}$ liver iron in various groups classified according to the cause of death. The highest mean liver iron was found in cases who died of malignancy and cardiovascular disease. The lowest mean value was observed in cases who died of trauma.

All groups except those who died of trauma, had mean liver iron higher than that reported for controls. In 12 cases who died of infection, the mean liver iron was 98.0 ± 10.8 ug/100 mg. Of these 12, the mean in 6 cases who died of chronic infection was 113 ± 27.3 and in 6 cases who died of acute infection was 83 ± 11.3 .

Table III: Mean Liver Iron in Cases with Various Causes of Death

Cause of Death	Liver Iron ($\mu\text{g}/100 \text{ mg}$) dry weight		
	Mean \pm	SD \pm	SE
Cardiovascular Diseases	140 \pm	110	\pm 28.4 (15)
Infection	98.0 \pm	51.42	\pm 14.8 (12)
Chronic Infection	113 \pm	67	\pm 27.3(6)
Acute Infection	83 \pm	27.8	\pm 11.3(6)
Trauma	68.0 \pm	34.6	\pm 11.5 (9)
Malignancy	145 \pm	117	\pm 47.8 (14)
Liver Disease	133.7 \pm	78.5	\pm 39.2 (4)

Discussion

The size of the storage iron depots provide an important pointer to the previous iron nutrition of an individual. Iron nutrition in its turn is dependent on the balance between the quantities of available dietary iron and body requirements.

The size of the body stores can be assessed in a number of ways. Some of these are highly cumbersome like the use of repeated phlebotomies, while others like estimation of serum ferritin requires very expensive reagents which are not widely available.

The liver is a particularly useful organ for estimation of the iron stores since it contains up-to one third of the total body storage iron and sufficiently large samples can be obtained to permit accurate chemical estimation of non-haem iron.

However, the limitation on such an approach is the problem of obtaining specimens from presumably healthy persons, or from autopsies, where the underlying cause of death did not interfere with the quantity or distribution of iron stores.

The present study was undertaken as a pilot study in order to determine the range of liver iron in an unselected autopsy material. Charlton et al. (1970) found no correlation between age and liver iron concentration in males. They reported that there was no difference between the values in individuals dying from cardiovascular diseases and those killed by acute trauma. This was not found in present study, where the subjects killed by trauma had lower values as compared with subjects who died from cardiovascular diseases.

The high values of liver iron reported in subjects dying from malignancy (Charlton et al., 1970) were also found in this study. This could be due to "trapping" of iron in the reticuloendothelial cells. This type of metabolic disorder may also occur in chronic infections (Bothwell and

Finch, 1962). High values were also found in subjects dying from chronic infection in the present study.

The values reported in controls (Agha et al., 1978) were quite similar to those found in subjects dying from acute infection in the present study. Considerable variation was noted in the values in subjects dying from various causes of death. However, the traumatic deaths in this study probably represent control values because these were sudden and there was no chance of mobilization of liver iron.

Previous work on measurement of transport iron in apparently healthy males (Hashmi et al., 1977) had indicated that the depletion of iron stores as estimated by the transferrin saturation below 15% was 11.7%. In this study, the iron stores below the minimum reported values were found in 8.2% subjects.

It appears from this preliminary study that the size of iron stores in the majority of male population was within the normal range and the estimation of transport iron provides a valid measurement of iron stores.

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