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Postmortem Vitreous Humor in Fatal Acute Iron Poisoning

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ABSTRACT: The postmortem level of iron in the vitreous humor was assayed in a case of treated fatal iron poisoning. An antemortem serum iron level was 9060 μg/dL and postmortem blood level was 2920 μg/dL. A postmortem vitreous humor specimen collected by a standard technique had an iron level of 80 μg/dL whereas 23 controls had a mean of 12.8 ± 4.2 μg/dL (range, 6 to 21 μg/dL). Although this elevated value could reflect some serum contamination, it may be a reflection of the toxic state.

KEYWORDS: toxicology, vitreous humor, poisons, iron

Fatal poisoning resulting from the sudden ingestion of iron is uncommon in Dade County, Florida. Since the inception of the Dade County Medical Examiner Department in 1956, only two cases were found, both young children. In the following case report, samples including postmortem vitreous humor were drawn for iron assay. To the best of our knowledge, no articles have been published on iron levels in postmortem vitreous humor in a case of acute iron toxicity. In addition, a range of normal values is established for iron in the vitreous humor after death.

Case Presentation

A 21-month-old black child developed sudden onset of lethargy and limping after vomiting his breakfast. The mother noted white discoloration around the mouth and correlated this observation with a nearly empty bottle of 325-mg ferrous sulfate tablets. Sixty tablets had been prescribed to the mother because of pregnancy and she had taken ten pills previously. The bottle, which had been left on the bedroom dresser the night before, was found on the living room floor with seven to ten pills remaining. Subsequent police investigation revealed that it was possible for the child to have obtained the tablets by himself.

The patient was admitted to a local hospital within 45 min from the onset of the symptoms. Ipecac was given and followed by nasogastric lavage and charcoal. Iron tablets were

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recovered from the lavage and an abdominal X-ray revealed six to eight tablets within the small intestine. The diagnosis of iron intoxication was evident and treatment with deferoxamine, 250 mg/h, was initiated. Hypotension and bradycardia ensued, after which the child was transferred to Jackson Memorial Hospital in Miami. Upon admission the patient was semicomatose and hypotensive with metabolic acidosis. Severe blood loss occurred through the stools and nasogastric tube. Despite intensive treatment with respirator therapy, vasopressors (epinephrine and dopamine), and deferoxamine, hypotension persisted and renal output ceased. Bradycardia eventually progressed to asystole and death was pronounced approximately 17 h from the onset of symptoms.

At autopsy, examination revealed a well-developed, well-nourished black child with acute gastroenteritis and a large amount of blood in the gastrointestinal tract.

Method

Antemortem blood and postmortem blood as well as postmortem vitreous humor were collected for iron content analysis. In addition, 25 vitreous samples were obtained from adult subjects who had died within 24 h without obvious hematologic disease. The latter specimens were obtained to establish a normal range. All samples were taken from subjects whose deaths were investigated in accordance with the Florida Statute 406 and were, therefore, under the jurisdiction of the Dade County Medical Examiner Department. The fluids were collected in Monojet® Vacutainers® and were then transferred to serum tubes (Becton-Dickinson Co., Rutherford, NJ) except for the postmortem vitreous specimen in the case of the fatal toxicity. The latter was collected in a sodium fluoride-potassium oxalate tube (Becton-Dickinson Co.).

The iron determinations for the serum and ocular fluids were performed on a Ferrochem® Model 3050 (Environmental Sciences Associates, Inc., Bedford, MD). This instrument uses an electrochemical technique called "controlled potential coulometry" [*1*]. In this method, there are two test electrodes with two different potentials. At one electrode, the ferric ions are reduced and at the other, the ferrous are oxidized. The current allowed to flow from these reactions is proportional to the iron concentration. This technique has a special advantage in that various chelating agents such as deferoxamine do not interfere in the determination [*1*]. The assays for sera were done according to the manufacturer's specifications. The ocular fluids were tested with one minor change. After standardization, the electrodes were primed with ocular fluid instead of serum before the electrodes were analyzed. The study was done under conditions that could be found in most general hospitals.

Results

The mean concentration for 23 of the 25 postmortem ocular fluids was 12.8 $\pm$ 4.2 μg/dL (range, 6 to 21 μg/dL). Two samples were not included; one was grossly bloody and the second had particulate matter that was probably tissue. This latter sample gave erratic values in the 30- to 50-μ/dL range.

The highest antemortem serum level for our case of acute iron toxicity was 9060 μg/dL whereas the postmortem concentration was 2920 μg/dL. The latter value no doubt reflects the deferoxamine therapy. The postmortem vitreous concentration was 80 μg/dL. No gross blood or particulate matter was demonstrable in the patient's vitreous humor.

Discussion

The values for ocular iron are in general agreement with those done on bovine ocular fluid [*2*]. The values, as expected, are lower than those for sera and any error from contamination would only have falsely elevated the results. Interestingly, the patient had an elevated ocular

fluid iron level compared to the others. The value, however, is much lower than the serum value and could reflect some extremely small contamination that might have occurred during collection. The collection of the vitreous in a sodium fluoride-potassium oxalate tube would have no effect on the values because sample tubes from the lot number involved were checked and no iron contamination was found. Also, the contents of that type of tube were added to normal sera and ocular fluid and shown to have no significant effect on the results. The postmortem vitreous iron concentration is immensely lower than the blood iron concentration in this case of fatal acute iron toxicity. However, the postmortem vitreous result is four times the upper limit of normal for our control population. This is probably a reflection of the patient's toxic state. However, a slight contamination from blood during the collection could account for the elevated vitreous value. This contamination would have to be on the order of 3% and, since no visible hemoglobin color was noted, this seems to be unlikely. The blood and vitreous data collected in this case show that any prospective study of vitreous iron values must pay special attention to collection techniques.

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