"RECOVERY" OF In-111 CHLORIDE UPTAKE IN SPLEEN AND BONE MARROW, AFTER CHEMOTHERAPY FOR ACUTE LEUKEMIA

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ABSTRACT

A man with acute myelogenous leukemia, treated with chemotherapy, had a decrease in circulating formed blood elements. Bone marrow biopsy revealed necrosis. Imaging with In-111 chloride demonstrated activity in the liver, but not in the spleen or bone marrow. Eighteen days later, the circulating blood count had risen; a repeat bone marrow biopsy showed the return of blood cell precursors. Follow-up In-111 chloride study at that time showed uptake in both the spleen and vertebrae, in addition to the liver. Hence, the spleen and bone marrow had "recovered" function. In the present study, potential causes of this change are discussed. Iranian J Nucl Med. Summer 1996.

Key words: myelogenous leukemia; chemotherapy; In-111 chloride

INTRODUCTION

Bone marrow necrosis has been found in association with diverse clinical entities, such as sepsis, chemotherapy, sickle cell crisis, tumor metastatic to marrow (1), and in the adult hemolytic-uremic syndrome (2). We have encountered a patient, with proven bone marrow necrosis, who had "failure" of the spleen, as well as judged by disruption of uptake of In-111 chloride.

CASE REPORT

A 41-year-old man was admitted for evaluation of thrombocytopenia. The diagnosis of acute myelogenous leukemia had been made on the basis of bone marrow biopsy which showed both blast cells and monocytic cell lines. A course of chemotherapy was instituted. This involved daunarubicin, ARA-C and 6-thioguanine over a period of 10 days. Pancytopenia developed and a second bone marrow biopsy was performed. This revealed bone marrow necrosis and a fibrotic component.

To localize any sites of functional marrow, In-111 chloride was injected intravenously and images obtained at 24 hours; this was 9 days after the bone marrow biopsy. Posterior abdominal imaging demonstrated hepatic uptake of In-111 with little activity in the spleen or vertebral marrow (Fig. 1). Further chemotherapy was withheld; the peripheral white cell count increased from 600 to 6,800 and platelet from 111,000 to 346,000. A repeat bone marrow biopsy demonstrated functional marrow with megakaryocytes present, as well as erythroid and myeloid cell lines. No leukemic cells were noted. A second In-111 chloride image was obtained 18 days after

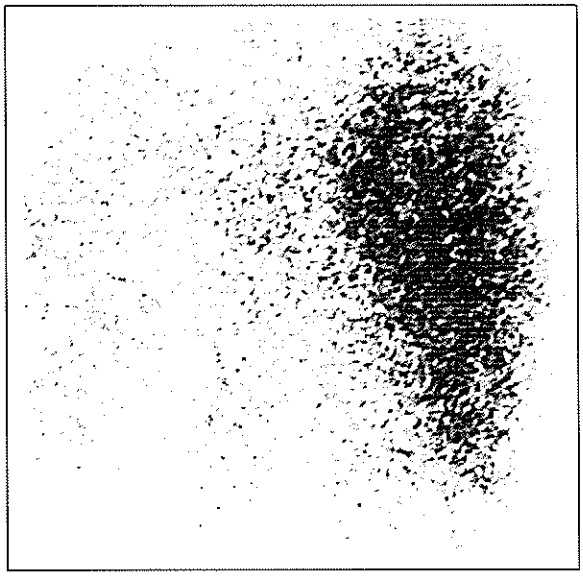

Fig. 1. This posterior image was obtained at 24 hours after intravenous administration of In-111 chloride. The liver can be delineated, while little activity is noted in the spleen or vertebral bone marrow.

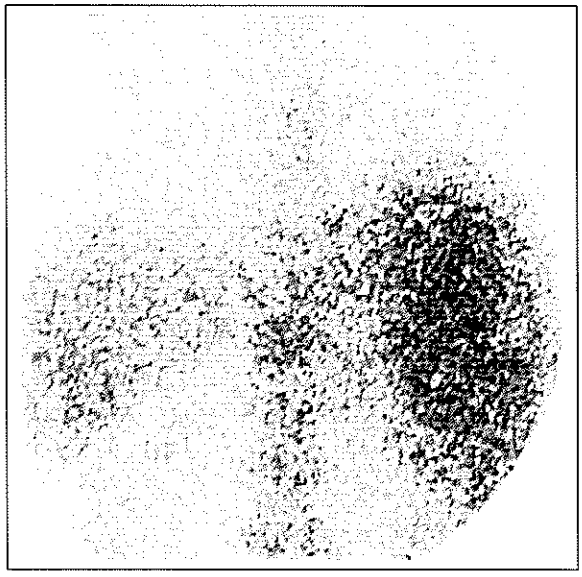

Fig. 2. This posterior image, also at 24 hours after In-111 chloride, was obtained 18 days after the first study. Activity in the spleen and vertebral marrow, as well as in the liver, can now be appreciated. In addition, faint uptake can be noted in at least 2 ribs.

the prior study (Fig. 2). This showed activity not only in the liver, but in the spleen and vertebral marrow as well.

DISCUSSION

The radiopharmaceutical In-111 chloride distributes by a dual mechanism. Much of the label, being an iron analogue, binds to transferrin in the blood stream. A portion of the injected material forms a radiocolloid, which then goes principally to the reticuloendothelial system. In our case, with demonstrated bone marrow necrosis, the initial failure of In-111 chloride to enter the vertebral marrow was thus not unexpected. However, the failure of the spleen to accumulate the radiolabel was surprising. While it is difficult to prove etiology in a single case, we can hypothesize at least 3 scenarios for splenic failure of uptake of In-111 chloride, followed by "recovery" :

1. Chemotherapy may have been cytotoxic to spleen cells. There were few available data to allow us to compare splenic susceptibility to that of other sites of reticuloendothelial cells;
2. Dissolution of leukemic cells, during chemotherapy, may have presented sufficient particulates and "debris" to saturate splenic reticul reticloendothelial function. However in a familial disorder with massive reticuloendothelial uptake of red blood cells and presumptive phagocytic cell engorgement (erythrophagocytic lymphohistio-cytosis), hepatic radiocolloid uptake was depressed but not that in the spleen (3);
3. Destruction of leukemic cells, by chemotherapy, may have relieved intrasplenic pressure from the leukemic infiltrate, and permitted the return of reticuloendothelial function.

The occurrence of bone marrow necrosis after chemotherapy has been well documented (1,4). Maisel and coworkers have presented a tabulation of causes of bone marrow necrosis for various age groups (5). Carloss and coworkers described 2 cases of bone marrow necrosis (one with acute leukemia) in which Tc-99m-sulfur colloid was used to demonstrate the bone marrow abnormality. While the liver could be noted to have uptake of radiocolloid in both cases, no images of the spleen were presented. We suggest that, in cases of bone marrow necrosis, radiocolloid or In-111 chloride be used to define patterns of uptake and whether splenic congruence or noncongruence has any significance for clinical management.

REFERENCES

1. Brown CH III. Bone marrow necrosis, a study of seventy

cases. Hopkins Med J. **131:** 189-203; 1972.

2. Hicks CB, Redmind J H. Adult hemolytic-uremic syndrome and bone marrow necrosis. Western J Med. **141:** 680-681; 1984.

3. O'Brien RT, Schwartz AD, Pearson HA, Spencer RP. Reticuloendothelial failure in familial erthyrophagocytic lymphohistiocytosis. Pediatries **81:** 543-545; 1972.

4. Mason BA, Klug PP, Cohen P. Bone marrow necrosis during chemotherapy for lymphoma. J Am Med Assn. **239:** 1158; 1978.

5. Maisel D, Lim JY, Pollock WJ, Yatini R, Liu PI. Bone arrow necrosis: and entity often overlooked. Ann Clin Lab Science **18:** 109-115; 1988.

6. Carloss H, Winslow D, Kastan L, Yam LT. Bone marrow necrosis: diagnosis and assessment of extent of involvement by radioisotope studies. Arch Internal Med. **137:** 863-866; 1977.