

CHAPTER VI

SUMMARY AND CONCLUSION

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The serum level of phosphate and calcium has been studied in twenty-five children with ALL before, during and after induction of remission. Renal function tests (serum urea, creatinine, and uric acid) were also done concomitant with serum calcium and phosphate.

The findings were compared with twelve normal children of matched age and sex.

Diagnosis of leukemia was based on clinical presentation and hematological analysis including complete blood and bone marrow pictures.

Remission was induced with prednisone and vincristine for a period of 4-6 weeks after which, eighteen cases only (72 % of total) achieved remission.

The biochemical investigations including serum phosphate, calcium, uric acid, urea, and creatinine were conducted to all patients (twenty-five) at time of admission, 72 hours following initiation of treatment and after induction of remission (eighteen cases).

The serum phosphate level in our patients with ALL at time of admission was numerically lower than that of the controls, with no significant difference. 72 hours after initiation of treatment, the serum phosphate became significantly higher than the pre-treatment

level. After induction of remission, there was no significant variation on matching serum phosphate with the pretreatment and control groups.

No disturbances in the serum calcium level were detected in patients at admission in comparison to the controls. 72 hours after initiation of treatment, the serum calcium became significantly lower than the pre-treatment level. Three patients developed manifest tetany. After induction of remission, serum calcium showed no significant difference compared to pretreatment and control groups.

As regards kidney function, the serum urea and uric acid in the pre-treatment group showed a significant elevation compared to controls, while serum creatinine showed no difference in comparison to controls. With initiation of treatment, serum urea and uric acid showed further significant elevation in comparison to the pre-treatment group, and there was no significant variation on matching serum creatinine to pre-treatment group. After induction of remission, serum uric acid and creatinine showed no significant difference in comparison to pretreatment and control groups. Although serum urea was numerically higher than the controls but there was no significant difference when matched with the pretreatment and control group.

The major metabolic abnormalities seen in our leukemic patients after the start of treatment are due to acute tumor lysis syndrome. This syndrome is a consequence of the rapid release of intracellular metabolites (potassium, phosphate, and uric acid) in quantities that exceed the excretory capacity of the kidneys.

Hyperuricemia and hyperphosphatemia with hypocalcemia, were found in most of our patients with ALL who were very sensitive to chemotherapeutic agents. Mild kidney dysfunction evidenced by significant elevation of serum, urea and uric acid might be contributory to the above metabolic disturbance.

Blood transfusion, corticosteroid therapy, hypoproteinemia, hypoparathyroidism, sepsis and hypomagnesemia may have a role in the appearance of hypocalcemia.

Renal function may be impaired by precipitation of urate and phosphate salts in the renal tubules, obstructive uropathy, kidney infiltration by the tumor and pyelonephritis.

Hence, from the results of the present work we can conclude that definite calcium and phosphate disturbances occur in children with ALL chiefly shortly after the initial doses of the cytotoxic therapy.

Although these metabolic disturbances were not so severe as to require specific corrective measures, it is possible that these disturbances might reach

levels that endanger the patients life if not controlled.

Mild renal impairment evidenced by significant elevation of serum urea and uric acid was detected in the studied patients. However, it is our feeling that this is most probably due to a metabolic overload that exceeds the excretory capacity of the kidney rather than being true renal dysfunction. More severe renal impairment may occur due to the variety of the previously mentioned factors contributing to permanent renal dysfunction and damage, leading to vicious circle of metabolic and renal disturbances.

Consequently, it is recommended that close monitoring of serum calcium and phosphate plus assessment of renal function should be included as routine investigations for patients with ALL before therapy is administered. Subsequently, further evaluation should be done shortly after initiation of therapy and appropriate intervention should be instituted to deal with specific anomalies.