

A CASE OF IDIOPATHIC HYPOALBUMINEMIA TURNED AS MYELOMONOCYTIC LEUKEMIA

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ABSTRACT

Hypoalbuminemia is a common cause of generalized edema. Although the underlying cause can usually be identified but very rarely we may come across a case with no detectable cause either clinically or by routine investigations. These cases may be labeled as having idiopathic hypoalbuminemia. These are challenging cases for diagnosis and usually lead to treatment errors exposing the patient to side-effects of unnecessary medications. We present an adult patient with idiopathic hypoalbuminemia who presented with generalized edema and had no evidence of renal, hepatic, cardiac, or thyroid abnormality. A year later he developed severe thrombocytopenia three days after taking sodium valproate for headache. Astonishingly his bone marrow examination revealed chronic myelomonocytic leukemia. His BCR ABL was negative. Corticosteroids and supportive care with platelet concentrates and whole blood was given but he could not make.

KEY WORDS: Hypoalbuminemia; Albumin; Edema; Myelomonocytic leukemia.

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INTRODUCTION

Albumin has several important functions in the body like transport of various substances including bilirubin, fatty acids, metals, ions, hormones, and drugs. It is also responsible for the normal colloid oncotic pressure and therefore severe hypoalbuminemia causes generalized edema. Hypoalbuminemia is a frequently observed condition and it can be associated with several different diseases, including cirrhosis, malnutrition, nephrotic syndrome and sepsis.¹⁻³

Idiopathic hypoalbuminemia is different from systemic capillary leak syndrome (SCLS) which presents with recurrent acute episodes of vascular leakage manifesting as severe hypotension, hypoalbuminemia, hemoconcentration, and generalized edema.⁴

Although the underlying cause of hypoalbuminemia can usually be identified in clinical practice

but very rarely we may come across a case with no detectable cause either clinically or by routinely available investigations. These cases may be labeled as having idiopathic hypoalbuminemia. These are the challenging cases for diagnosis and usually lead to treatment errors exposing the patient to side-effects of unnecessary medications.

CASE PRESENTATION

A 40 years old gentleman presented with swelling of both the lower limbs. Clinical examination showed emaciated look, pallor, and gross pitting edema of both lower limbs up to the thighs. He also had clinically demonstrable ascites with no organomegaly. His pulse was 72 beats/min, BP 100/60 mm Hg and neck veins were not engorged. Chest and cardiovascular examination was unremarkable. Prior to this problem he worked in UK as a security guard for few years with no health problem while abroad. He had no bowel problem and had normal dietary habits, except while in UK his diet was lacking meat and mostly contained dairy products. On investigations FBC showed Hemoglobin 10.5 g/dl, with normal total and differential counts. Blood sugar, urea, creatinine, ALT, CPK levels were normal. HBsAg, Anti-HCV and Anti-HIV were negative. Urine proteins were repeatedly negative. Ultrasonography of abdomen showed mild ascites with normal liver, spleen and portal vein diameter and doppler study for lower limb vessels was unremarkable. Serum protein estimation showed hypoalbuminemia with total proteins 4.71 g/dl, albu-

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min 2.58 g/dl and globulin 2.13 g/dl.

He was referred to a tertiary care centre where he was thoroughly investigated by two physicians with no detectable cause for hypoalbuminemia. His investigations repeatedly showed urine proteins qualitatively negative and quantitatively within normal limits, and serum albumin significantly low. His serum cholesterol was reported as low (52 mg/dl). Creatinine clearance was normal. Ascetic fluid was transudative. Serum protein electrophoresis was normal apart from hypoalbuminemia. (Fig. 1) Thyroid function tests were normal and autoantibody screen was negative. Doppler of lower limb veins was normal with normal d-dimers levels. Chest x-ray, ECG and echocardiography were also unremarkable. CT scan abdomen just showed ascites, CT chest minimal pleural effusion, and CT angiogram of lower limbs was unremarkable.

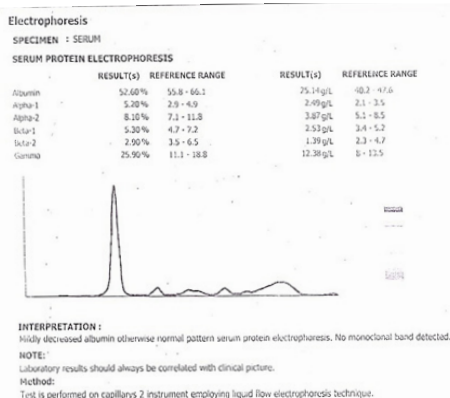


Figure 1: Serum proteins electrophoresis of patient with idiopathic hypoalbuminemia.

During his shift-over from one physician to another he was given antibiotics, corticosteroids, azathioprine, diuretics, and proton pump inhibitors from time to time.

The patient was labeled as Idiopathic hypoalbuminemia, all unnecessary medications mentioned above were stopped except Spiromide (Furosemide + Spironolactone) and he was given dietary advice to have high protein diet. On follow-up he was feeling better, had no ascites, but was still having pitting edema of his lower limbs.

A year later he developed severe thrombocytopenia three days after taking sodium valproate for headache. Astonishingly his bone marrow examination revealed chronic myelomonocytic leukemia. His BCR ABL was negative. Corticosteroids and supportive care with platelet concentrates and whole blood was given but he could not make

DISCUSSION

Idiopathic hypoalbuminemia with no clinical or laboratory evidence of renal, hepatic, cardiac, or

thyroid abnormality is a rare disorder in which the patient may present with generalized edema.

Watkins et al⁵ described an Italian family with analbuminemia, a very rare inherited syndrome in which the subjects produce little or no albumin because of mutation in the albumin gene. These patients usually get moderate edema and few related symptoms due to compensatory increase in other plasma proteins. The mother in this family was homozygous for the trait with serum albumin level <0.01 g/dl while the daughter was heterozygous with nearly normal albumin value.

Weinstock et al⁶ described a case of idiopathic hypoalbuminemia in whom albumin kinetics and morphologic observation of hepatocellular alterations suggested decreased albumin synthesis. There was abnormally large postural shift of intravascular fluid into the extravascular compartment. Effect of albumin infusion on these physiologic and biochemical abnormalities suggested that most were secondary to hypoalbuminemia.

Hsu et al⁴ described six children with SCLS from the United States, Australia, Canada, and Italy. Serum cytokines from these subjects and 10 healthy children were analyzed. It was found that children with SCLS had at least one acute, severe episode of hypotension, hypoalbuminemia, and hemoconcentration in the absence of underlying cause for these abnormalities. Infectious triggers precipitated most episodes in children and elevated levels of various cytokines were observed in these patients as compared to controls.

CONCLUSION

With the availability of better diagnostic facilities idiopathic hypoalbuminemia has become almost obsolete but cases do present from time to time in whom routine clinical and laboratory investigations may not be able to determine the cause of hypoalbuminemia and resulting edema. These cases of idiopathic hypoalbuminemia should be identified to avoid the unnecessary medications and their side-effects.

REFERENCES

1. Gatta A, Verardo A, Bolognesi M. Hypoalbuminemia. Intern Emerg Med 2012 Suppl 3:S193-9.
2. Losowsky MS, Alltree EM, Atkinson M. Plasma colloid osmotic pressure and its relation to protein fractions. Clin Sci 1962; 22:249.
3. Gill JR, Waldmann TA, Bartter FC. Idiopathic edema: 1. The occurrence of hypoalbuminemia and abnormal albumin metabolism in women with unexplained edema. Am J Med 1972; 52:444-51.
4. Hsu P, Xie Z, Frith K, Wong M, Kakakios A, Stone KD, et al. Idiopathic systemic capillary leak syndrome in children. Pediatrics 2015; 135:e730-5.
5. Watkins S, Madison J, Gallianott M, Minchiotti L,

- Putnam FW. A nucleotide insertion and frameshift cause analbuminemia in an Italian family. *Proc Natl Acad Sci* 1994; 91:2275-9.
6. Weinstock JV, Kawanishi H, Sisson J. Morphologic, biochemical and physiologic alterations in a case of idiopathic hypoalbuminemia (analbuminemia). *Am J Med* 1979; 67:132-9.

CONFLICT OF INTEREST
Authors declare no conflict of interest.
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