

Case Report:

SIADH IN MDS: A Rare case report

Dr.V.K.Vineeth

Name of the Institute/College: K S Hegde Medical Academy

Corresponding author: Dr.Vijith Shetty

Abstract:

The syndrome of inappropriate secretion of anti diuretic hormone is a disorder of impaired water excretion caused by inability to suppress the secretion of anti diuretic hormone. Most common causes are CNS disturbances, Malignancies, Drugs, Pulmonary diseases. Any CNS disorder like infection, trauma, hemorrhage and psychosis can enhance ADH release.

Ectopic production of ADH by a tumor is most often due to small cell carcinoma of lung. Other rare causes among malignancies are head and neck cancer, olfactory neuroblastoma and extrapulmonary small cell carcinoma.

SIADH is generally not associated with hematological malignancies with mechanism not known. According to literature there is only one case of SIADH in MDS and SIADH with AML.

Keywords: Myelodysplastic syndrome , SIADH

Introduction:

Myelodysplastic syndrome comprises of heterogeneous group of malignant hematopoietic stem cell disorders characterized by dysplastic and ineffective blood cell production. There is a risk of transformation to acute leukemia. Those patients have varying reductions in the production of red blood cells, platelets and mature granulocytes. Diagnosis is based on the following features.

1. Otherwise unexplained quantitative changes in one or more of blood and bone marrow elements.
Haemoglobin<10g/dl, absolute neutrophil count<1800/microL, Platelets<100,000/microL
2. Morphological evidence of significant dysplasia upon visual inspection of peripheral smear, bone marrow aspirate and bone marrow biopsy in the absence of other causes of dysplasia.
3. Blast forms account for less than 20% of total nucleated cells of bone marrow aspirate and peripheral blood.

Case Report:

A 73 Year old male from Mangalore who is known case of systemic hypertension, Type 2 DM on T.Metformin 500mg 1-0-1 and T.Amlodipine 5mg 1-0-1 and a known case of Myelodysplastic syndrome who had finished his 3rd cycle of chemotherapy came with complaints of fever, Fatigue, abdominal pain of 2 days duration. He had diffuse abdominal pain. Initially he was suspected with febrile neutropenia based on history.

On examination his pulse rate was 100/min, BP-160/100mm of Hg, systemic examination showed diffuse tenderness in the abdomen.

Initial reports showed Hb-8.4gm/dl, Total count-3100, with neutrophil predominance, Sodium -111mmol/l , Potassium-4.76mmol/l. Clinically he did not show any signs of hypovolemia. His reflexes were 2+ and his skin turgor was normal. There were no apparent foci of infections.

LFT Showed direct hyperbilirubinemia, elevated alkaline phosphatase. Patient was managed with iv Piperacillin and tazobact, iv fluids, antihypertensives and appropriate doses of insulin. Patient was afebrile after the initiation of antibiotics.

Next day Sodium was 113mmol/l potassium-4.38mmol/l. In spite of continuous hydration patient was in euvolemic state. Hence Urine spot sodium was sent and urine spot sodium was 47mmol/l(elevated).

Clinical suspicion towards SIADH was made and was worked up. Urine osmolality was sent and found to be elevated 1380mosm/kg water confirming SIADH. Patient was started on TAB.TOLVAPTAN 15mg OD for 3 days. Sodium picked up from 113mmol/l to 126mmol/l in 3 days reconfirming our diagnosis. Following patient's fatigue disappeared and sense of well being improved. His sodium levels did not drop during the stay. However he had long stay during which he went into sepsis secondary to acute cholecystitis and went into multi organ dysfunction. He expired after he developed severe metabolic acidosis inspite of receiving one cycle of haemodialysis.

This case holds lot of importance because its one of the rare case where SIADH was picked up quite early. SIADH is not expected to be associated with haematological malignancies.

Discussion:

Hyponatremia from SIADH is a common electrolyte abnormality seen in patients with small cell carcinoma of lung. Usually SIADH is uncommon in blood related malignancies. As per literature there is only one case documented in 2015 in a case of MDS with blast crisis⁽¹⁾. It showed similar clinical status with improvement after starting Tolvaptan.

Accurate drug history is vital to look for the cause of SIADH. In a study titled SIADH in children with Acute lymphoblastic leukemia by Anupama S Borker et al proved that SIADH is common in patients on treatment for ALL especially after 14 days of treatment with Vincristine⁽²⁾.

Initial treatment of hyponatremia is hypertonic saline. However as this patient did not respond to it, he was started on Tolvaptan. Tolvaptan can be initiated at a dose of 15mg OD to maximum of 60mg OD. It blocks the effects of AVP(Arginine vasopressin) in the renal collecting duct to promote aquaresis leading to controlled increase in serum sodium levels by inducing free water excretion without increasing sodium excretion⁽³⁾.

Hence in our case patient responded to the therapy and sodium levels picked up. So it is one rare documented case of SIADH secondary to MDS. It is thought to be caused by ectopic production of AVP by tumour tissues or effect of chemotherapy. No other mechanism has been stated till now.

Conclusion:

This case report is one rare case explaining SIADH secondary to MDS. Before confirming the cause detailed evaluation of other causes is very important. Tolvaptan is a very useful drug in symptomatic cases of SIADH. Early detection and treatment of SIADH secondary to MDS is necessary which otherwise can prove fatal.

This paves way for further research in SIADH in blood related malignancies as it is not reported much.

References:

1. Padmavathi Mali, Sudheer R Muduganti, Rahaman Mujibur, Narayana Murali, Tolvaptan for SIADH in Myelodysplastic syndrome with blast crisis, WMJ April 2015; Volume 114, no 2
2. Anupama S Borker, Sabine Hutchins et al, Syndrome of inappropriate antidiuretic hormone secretion in children with acute lymphoblastic leukaemia, Blood 2006, 108;4474
3. Gheorghide M, Gottlieb SS, Udelson JE, et al, Vasopressin V(2) receptor blockade with tolvapatan versus fluid restriction in treatment of hyponatremia, Am J Cardio, 2006;97;1064-1067