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Case Study

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CASE REPORT- AUTOIMMUNE HAEMOLYTIC ANAEMIA WITH SEPTIC SHOCK WITH RIGHT PROXIMAL TIBIA STRESS FRACTURE

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ABSTRACT

Autoimmune haemolytic anaemia (AIHA) is an acquired autoimmune disorder causing production of antibodies directed against RBC antigens causing shortened erythrocyte survival.[1]

Classification of AIHA include warm AIHA, cold agglutinin syndrome, paroxysmal cold hemoglobinuria, mixed type AIHA and drug induced AIHA.[2]

Management of AIHA is based mainly on empirical data on small, retrospective, uncontrolled studies. Certain therapeutic options available are monoclonal antibodies and complement inhibitory drugs[3] though the mainstay of treatment remains steroids, immunosuppressants and in some cases splenectomy.

This case report demonstrate the importance of comprehensive health screening and management of a case of AIHA with septic shock and right proximal tibia stress fracture.

CASE DESCRIPTION

A 39 year old housewife presented to the hospital with chief complaint of pain in right lower limb followed by swelling and tenderness since 1 week.

She was apparently alright 6 months back when she had a fall which in turn lead to trauma to her right leg. Though the initial X Ray was not suggestive of fracture, due to lack of rest and continuous stress, she was later diagnosed with right proximal tibia stress fracture. It was managed using Ilizarov ring fixator and regular follow ups were scheduled.

The patient started developing redness, swelling and yellowish discharge from the ilizarov pin site due to irregular treatment.

On further enquiry history of high grade fever associated with chills and 4 episodes of vomiting, containing food particles, non projectile, non bilious, not blood stained was deciphered.

On examination the patient was drowsy, disoriented and not following commands. She was febrile with a temperature of 103 F, tachycardic (pulse-130/min), BP 110/70mmhg and oxygen saturation of 99% on RA. She was admitted in the ICU and kept on NIV (30/5/18) for 3 days i/v/o severe tachypnea.

Pallor was present.

Local examination revealed red, warm, and tender right leg with swelling present.

Automated cell counter of peripheral blood showed leucocytocis (12.9 UL) and a platelet count of 429UL. Evaluation of blood parameters revealed features of severe anaemia with Haemoglobin of 3.1gm/dl, MCV 64fl MCHC 10.4gm/dl. Iron studies was done, Iron was 6.3, TIBC was 466, Ferritin >2000 & retic count of 14.

PT/INR was 13.8/ 0.97. Ther blood parameters were SGOT- 60mg/dl, SGPT- 15mg/dl, Bilirubin (Indirect+ Direct) – 2.70mg/dl (1.40+1.30). Creat was 0.83 and Alkaline phosphatase was 161.

Based on a provisional diagnosis of severe anaemia with right proximal tibial stress fracture post ilizarov fixation, blood transfusion was planned. The blood samples came out to be DCT positive. This further confirmed the diagnosis of AIHA.

The management protocol followed was IVIG 1 gm/kg for 24hours before the blood transfusion. Patient was not started on Inj MPS as she was in septicemia. Following

IVIG blood transfusion was done. 3 pint blood was transfused.

Post 3 pint of blood transfusion the Hb raised to 6.6 Gm/dl with platelet count of 207UL (after 48hours).

Further investigations were done to identify the etiology. ESR and CRP values were significantly raised. To rule out other infective etiology urine routine, MP/MAT, Dengue NS1, Leptospira, WIDAL &VDRL were performed. The results were insignificant.

Pus culture was done for the pin tract infection which revealed staphylococcus aureus. ANA report didn't show any significant changes.

The patient was started on Inj PCM (1gm) for fever. Inj Meropenem , Inj Linezolid and Inj Clindamycin were started to combat the bacterial colonisation. Nutritional supplement was added in the form of Inj optineuron.

As of today, the patient has improved and septicemia resolved without any fever spike. Patient was discharged on tab. prednisone 1mg per kg.

DISCUSSION

Hemolytic Anaemia results due to intrinsic and extrinsic factors. AIHA is one such group of acquired haemolytic anaemia. It is a rare finding.

Diagnosis is based on evidence of haemolytic anaemia consisting of anaemia, jaundice, splenomegaly, raised serum bilirubin and a positive DCT.[4]

AIHA is known to be associated with infection, malignancy or any other autoimmune condition, though the majority cases seen are idiopathic.[5]

There have been studies showing a link of AIHA and SLE, hence necessitating screening of all patients of AIHA for clinical evidence of SLE.[6]

Corticosteroid therapy and blood transfusion remain the mainstay of treatment for AIHA. Transfusions are of transient benefit but are required initially due to the severity of anaemia. Cross matching and blood grouping due to the presence of agglutinins become troublesome.[7] However with the advancing science many patients do not require repeated blood transfusions and are managed solely on corticosteroid therapy and they show dramatic response. Other modalities of treatment available are immunosuppressant therapy and plasmapheresis.

The patient is still in the path of recovery and has shown promising signs of improvement. Further course of management would depend on the recovery process and stringent follow ups with the patient.

REFERENCES

1. Liebman HA, Weitz IC. Autoimmune Hemolytic Anemia. Med Clin North Am, Mar, 2017; 101(2): 351-359. doi: 10.1016/j.mcna.2016.09.007. Epub 2016 Dec 14. PMID: 28189175.
2. Gehrs BC, Friedberg RC. Autoimmune hemolytic anemia. Am J Hematol, Apr, 2002; 69(4): 258-71. doi: 10.1002/ajh.10062. PMID: 11921020.
3. Sève P, Philippe P, Dufour JF, Broussolle C, Michel M. Autoimmune hemolytic anemia: classification and therapeutic approaches. Expert Rev Hematol, Dec, 2008; 1(2): 189-204. doi: 10.1586/17474086.1.2.189. PMID: 21082924.
4. Grunchy GC. The hemolytic anaemias.In: Firkin FC, Chesterman CN, Penington DG, Rush BM, Editors De Gruchy's clinical haematology in medical practice.5. Edinburgh: Blackwell Science Ltd, 1989; 172-215. doi: 117.239.25.194
5. Gupta V, Shukla J, Bhatia BD. Autoimmune hemolytic anemia. Indian J Pediatr, May, 2008; 75(5): 451-4. doi: 10.1007/s12098-008-0071-0. Epub 2008 Jun 8. PMID: 18537006.
6. VIDEBAEK A. Auto-immune haemolytic anaemia in systemic lupus erythematosus. Acta Med Scand, Feb, 1962; 171: 187-94. doi: 10.1111/j.0954-6820.1962.tb04180.x. PMID: 13925815.
7. Leverger G, Bancillon A, Schaison G. Traitement des anémies hémolytiques auto-immunes de l'enfant [Treatment of autoimmune hemolytic anemias in children]. Ann Pediatr (Paris), Oct, 1989; 36(8): 519-23. French. PMID: 2683950.