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Dengue induced nephritis with concurrent parvovirus infection

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Abstract

Dengue fever induced Hemolytic Uremic Syndrome (HUS) characterized by a triad consisting of hemolytic anemia, thrombocytopenia and Acute Kidney Injury (AKI) has been described in a few patients. Fluid replacement should be performed carefully in these children to avoid overload producing a consequent worsening of intravascular fluid extravasation. The risk of acquiring infections such as parvovirus from blood and blood products is well recognized. However, Parvovirus can induce transient aplastic crisis in children without underlying hemolytic disorders as well and should be kept in mind when a complex picture arises.

Keywords: HUS, AKI, Dengue

Case Presentation

9 year male child, brought by mother came with chief complaints of fever and vomiting since 5 days and cola colored urine since 4 days. On admission, BP was at 90th centile. CBC showed Hb 10.6 TLC 6700 platelets 67,000 Hct 29.8. Dengue NS1 was positive by Rapid antigen test, BUN was 135 with a serum creatinine of 5.3. Liver function tests, PT/INR, serum electrolytes and blood gas were within normal limits. Urine routine showed albumin+, occult blood+, RBC:8-10/HPF, pus cells 10-15/HPF, epithelial cells 4-5/HPF with granular casts 1-2/HPF. Patient was started on oliguric fluid with urine output replacement. BP was persistently greater than 95th centile, hence patient was started on antihypertensives (calcium channel blockers). USG revealed bilateral medical renal disease grade 1 with normal size of kidneys with mild ascites. Serum complement C3 levels were low (77). ASLO titres were weakly positive with a negative throat swab culture. 2D Echo study was normal. CBC monitoring showed improvement in platelet count which was however associated with gradual decrease in Hb. On DOA 3; investigations revealed Hb 8.3 TLC 14,300 PLT 2,92,000 HCT 24.3; RFT : 136/5.6. Minimal fluid replacement was continued with BP and urine output monitoring. On DOA 5; Hb 6.1 TLC 7600 PLT 2,92,000; RFT 89/3.6. Peripheral smear showed fragmented cells with presence of schistocytes; reticulocyte count was 30% with raised LDH levels of 3599. Diagnostic impression of Dengue induced hemolytic uremic syndrome was made. Patient was transfused with PRC @10cc/kg. Pre transfusion vit B12 levels, G6PD enzyme levels were within normal levels,

DCT/ICT was negative and HPLC was also sent from pre transfusion samples which was normal. Post transfusion patient had persistent high grade fever spikes. Fever was associated with pancytopenia. On DOA 15: Hb 5.3 TLC 2100 PLT 1,79,000. Prophylactic antimalarials were given to cover post transfusion malaria. Hep B, Hep C and HIV were negative from pre transfusion. On DOA 17 Hb 4 TLC 1400 PLT 1,43,000; RFT 29/1.1. Patient was investigated for acquired causes of pancytopenia; Mycoplasma IgM was negative. However, Parvo IgM was positive (<0.9 normal, 0.9-1.1: borderline, >1.1: positive). Patients value was 2.0. Patient was managed conservatively with strict monitoring. Patient was discharged after 24 days. On discharge , Hb 5.3 TLC 5300 PLT 6,51,000 RFT 17/0.9.

Investigations

Included in the body of the case report.

Management

Careful assessment of warning signs of severe dengue and the patient's blood volume are crucial for the management of AKI. Amount of infused fluid should be minimum needs to stably maintain the hemodynamic conditions until the increased vascular permeability is reversed. Renal replacement therapy is currently indicated as conventionally used because there are no specific recommendations for proper time to begin treatment, dosing or modality in Dengue patients.

Parvovirus induced transient aplastic crisis may need iv immunoglobulin however success rate is not clear. There can also be spontaneous recovery in healthy patients.

Outcome and Follow UP

Child was discharged after 25 days with plan to follow up regularly with BP monitoring, CBC and RFT monitoring. Follow up with Parvo PCR is essential as its persistence in bone marrow can lead to periodic remissions or severe aplastic anemia, rarely requiring bone marrow transplant.

Discussion

Dengue virus infection induced manifestations are varied, pathogeneses of which include antibody dependent enhancement, virus virulence and IFN gamma/ TNF alpha mediated immunopathogenesis[1]. Aberrant immune responses not only impair the immune response to clear the virus, but also result in overproduction of cytokines. AKI is a lesser known complication of Dengue. Several mechanisms have been proposed to account for etiopathogenesis of Dengue fever induced AKI including the follows[2]:

1) Direct cytopathic effect of viral protein on glomerular and tubular cells.
2) Hemodynamic instability due to cytokine storm leading to shock due to a reduction of renal perfusion and acute tubular injury.
3) Rhabdomyolysis is a rare complication of Dengue and can lead to AKI.
4) Glomeruli may also be affected by Dengue.
5) Hemolytic uremic syndrome can be caused by Dengue.

Parvovirus B19 is widespread and exhibits marked tropism to human bone marrow replicating mainly in erythroid progenitor cells[3]. In immunocompromised host, persistent B19 infection causes pure red cell aplasia and chronic anemia, while volunteer studies of normal subjects have in addition revealed low grade neutropenia and thrombocytopenia. Proteins of Parvovirus B19 receptors are expressed even in marrow cells other than erythroid progenitors where viral propagation takes place[4]. Direct cytotoxicity by these

viral proteins causes destruction of marrow elements. But how vast majority of normal individuals clear the virus and only a few manifest it remain unclear. Case reports in literature show remission of pancytopenia with immunosuppressive therapy. However other case reports show no or minimal improvement with immunosuppression but successful remission with HLA matched Bone Marrow Transplant[4].

Learning Point/Take Home Message

Fluid management in Dengue induced nephritis is very critical and a delicate balance must be maintained.

Acute infection with Parvovirus must be considered a cause of acquired aplastic anemia even in immunocompetent individuals without underlying hemolytic disease.

References

1. Lei H-Y, Yeh T-M, Liu H-S, Lin Y-S, Chen S-H, Liu C-C. Immunopathogenesis of Dengue Virus Infection. J Biomed Sci [Internet] 2001 [cited 2020 Feb 28];8(5):377–88. Available from: http://www.karger.com/doi/10.1159/000054058
2. Oliveira JFP, Burdmann EA. Dengue-associated acute kidney injury. Clin Kidney J [Internet] 2015 [cited 2021 Sep 25];8(6):681. Available from: /pmc/articles/PMC4655808/
3. Bir Çocukta İ, B19 P, İndüklenen T, Kriz GA. Parvovirus B19 Induced Transient Aplastic Crisis in an Immunocompetent Child.
4. Heegaard ED, Madsen HO, Schmiegelow K. Transient pancytopenia preceding acute lymphoblastic leukaemia (pre-ALL) precipitated by parvovirus B19. Br J Haematol [Internet] 2001 [cited 2020 Feb 27];114(4):810–3. http://doi.wiley.com/10.1046/j.132141.2001.