Parvovirus B19 induced fulminant liver failure in pregnancy: A rare incidence

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ABSTRACT

Parvovirus B19 (B19V) is a single-stranded DNA virus of the family Parvoviridae and genus Erythrovirus. Parvovirus B19 infection can present with myriads of clinical diseases and syndromes; liver manifestations and hepatitis are examples of them. The majority of the available literature regarding acute parvovirus B19 induced fulminant hepatic failure has described cases involving children. Acute fulminant liver failure caused by acute parvovirus B19 infection in a pregnant lady is rarely reported in the literature. Here, we report a case of fulminant liver failure in a 28-year-old pregnant lady presenting as a diagnostic dilemma, the etiology of which was eventually detected as acute Parvovirus B19 infection. Since Parvovirus B19 is a potential cause of non-A, non-E fulminant hepatic failure, an early diagnosis is of utmost importance as treatment options are limited.

Keywords: *DNA virus, Liver failure, Parvovirus B19, Pregnancy.*

Acute liver failure is a rare clinical syndrome with an annual incidence of fewer than 10 cases per million population in the developed world [1]. Parvovirus B19 (B19V) is a single-stranded DNA virus of the family Parvoviridae and genus Erythrovirus. Parvovirus B19 infection can present with myriads of clinical diseases and syndromes; liver manifestations and hepatitis are examples of them. Parvovirus B19 is an erythrovirus that can only be replicated in pronormoblasts and hepatocytes, and other cells which have globosides and glycosphingolipids in their membrane can also be affected by direct virus injury [2].

The majority of the available literature regarding acute parvovirus B19 induced fulminant hepatic failure have described cases involving children [3,4]. Parvovirus B19 has been implicated as an important cause of non-A and non-E hepatitis [5,6]. Acute fulminant liver failure caused by acute Parvovirus B19 infection in a pregnant lady is rarely reported in the literature. There is no specific treatment for parvovirus B19 related liver diseases, but triple therapy regimen may be effective consisting of immunoglobulin, dehydrohydrocortisone, and cyclosporine [2].

CASE REPORT

A 28-year-old pregnant lady presented to us with a 1-month history of anasarca, icterus, easy fatiguability, episodic epistaxis, and pruritus. She was a housewife with no personal or family history of known medical ailments or any history of travel. Clinical examination revealed an icteric, afebrile, conscious lady with normal hemodynamic parameters (blood pressure of 110/60 mmHg, pulse rate of 88/min and respiratory rate of 22/min) and abdominal distension (free-fluid present) with no evidence of hepatomegaly/splenomegaly. She had pallor and significant bilateral pitting pedal edema. No skin rashes/bleeding manifestations were noticed. Her chest and cardiovascular examinations were normal and neurological examination revealed no focal deficits or tremors or flaps.

Complete blood counts showed anemia (Hg-8.6 g/dl), thrombocytopenia (platelets- 80,000 cells/mm^3) with evidence of normocytic normochromic RBCs on peripheral smear. Iron studies showing decreased iron levels. Her liver function tests (LFT) showed direct hyperbilirubinemia (total bilirubin - 26, direct bilirubin – 15 mg/dl), modest elevation of enzymes (serum glutamic oxaloacetic transaminase – 114I U/L, serum Glutamic Pyruvic Transaminase – 112.49 IU/L) with marked elevation of serum alkaline phosphatase (331 IU/L). Prothrombin time (PT) was prolonged (22 sec) with deranged International Normalised Ratio (INR-2.1). Kidney function tests (Urea-15mg/dl, Creatinine-0.66mg/dl) were normal. An abdominal ultrasound revealed 25 weeks single live fetus with the gross peritoneal collection and a coarse liver. A diagnostic ascitic tap revealed high serum ascitic albumin gradient (1.8), low protein (1.1 gm%) and lymphocyte - predominant fluid. Viral markers for Hepatitis A, E, B and C were all negative. IgM anti-Epstein Barr virus was negative and IgM anti-parvoB19 was negative. Parvo B19 DNA was detected in serum. Antinuclear antibody (ANA), anti-liver kidney microsomal-1, anti-soluble liver antigen, anti-smooth muscle antibody, and anti-mitochondrial antibody were all negative. Upper gastrointestinal endoscopy revealed evidence of portal gastropathy. Ceruloplasmin was modestly reduced (36.5)

with absent Kayser Fleischer ring on slit lamp exam. Serum bile acids were normal (0.8 mg/dl).

The patient was managed conservatively with iron and folate supplementation, ursodeoxycholic acid, syrup lactulose, potassium supplementation, Rifaximine and Torsemide. She had been transfused with 6 units of fresh frozen plasma and also received Injection Vitamin K.The patient had a gradual downhill clinical course with Model for End-Stage Liver Disease (MELD) score deteriorating from 23 to 27 (Fig. 1). Attendants were counseled for intravenous Immunoglobulin, plasmapheresis and liver transplantation. The patient had a massive upper gastrointestinal bleed (probably due to deranged INR) with alteration of sensorium (hepatic encephalopathy) and went into irreversible shock. Unfortunately, she could not be revived despite all supportive measures.

DISCUSSION

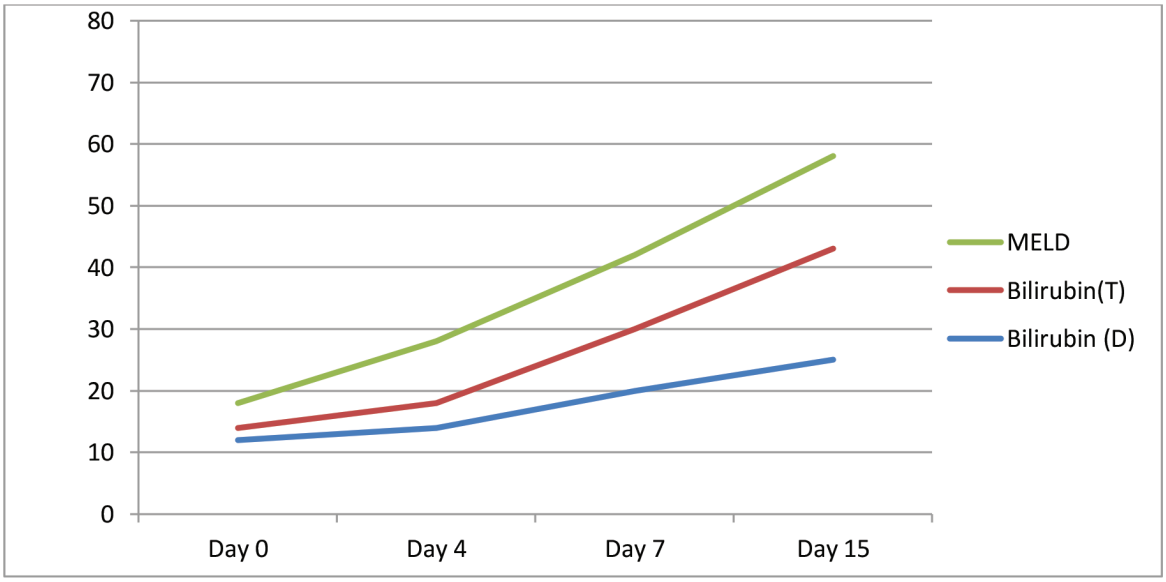
Parvovirus B19 infection is a common infection in childhood as well as in pregnancy. The clinical presentations can be benign to life-threatening as was in our case.As the majority of patients with liver failure are diagnosed as "indeterminate", a sound knowledge of Parvovirus B19 associated liver disease is limited. The mechanism by which Parvovirus B19 infection may result in hepatic injury is not clear. Hepatic cell damage related to direct viral invasion is one possibility.Alternatively, an injury may result as an indirect consequence of the immune response directed against the virus [7].

Parvovirus B19 infection has been reported as a rare cause of fulminant liver failure in children requiring liver transplantation [8]. A case of non-A-E fatal acute liver failure in an immunocompetent child with B19 infection had also been described [9]. Our knowledge of Parvovirus B19 as a potential cause of non-A, non-E acute liver failure in a pregnant woman is limited. Although B19 IgM antibodies are rarely found in those patients that are not associated with an outbreak and without an illness suggesting parvovirus infection, there is always a possibility of false-positive results among these cases, even when using highly accurate tests [10]. Parvo B19 DNA by polymerase chain reaction (PCR) was positive in serum in our case. Detection of ParvovirusB19 DNA and VP1 antigen detection in a liver biopsy specimen would confirm the presence of active viral infection [10]. A liver biopsy was planned but could not be done in our case because of rapid clinical deterioration of the patient. Leptospirosis, Scrub Typhus and malarial hepatitis (Falciparum) were easily ruled out due to lack of fever and negative smear examination and serologies. A meticulous drug history revealed no specific hepatotoxic drug ingestion.Testing for rare causes of non-A, non-E Hepatitis like SEN Virus, Toga virus, and Herpes Simplex virus were not done.

Parvo B19 infection in pregnancy is quite common and can cause a wide range of clinical outcomes ranging from fetal hydrops and intrauterine fetal death to maternal arthritis, transient aplastic crisis, myocarditis or asymptomatic [11,12]. The association of Parvovirus B19 and fulminant hepatitis in adults is well-established in the literature [13,14] but is rarely reported in pregnancy. In our case, with all other possible etiologies of fulminant hepatitis being ruled out, acute Parvovirus B19 induced liver failure seems to be the most logical diagnosis.Liver transplantation would have been the ideal treatment modality in such cases of fulminant liver failure as was reported in a 2009 study [15].

CONCLUSION

Since Parvovirus B19 is a potential cause of non-A, non-E fulminant hepatic failure, early diagnosis is of utmost importance as treatment options are limited. The physician should keep Parvovirus B19 in differentials while ordering tests for acute hepatitis as timely diagnosis and management could alter the patient's clinical course.



T: Total; D: Direct; MELD: Model for End Stage Liver Disease Score

Figure 1: Line diagram showing progressive derangement of liver function

REFERENCES

1. Bernal W, Auzinger G, Dhawan A, Wendon J. Acute liver failure. Lancet. 2010;376:190-201.
2. Bihari C, Rastogi A, Saxena P, Rangegowda D, Chowdhury A, Gupta N, *et al*. Parvovirus B19 Associated Hepatitis. Hepat Res Treat. 2013:1-9.Article ID 472027.
3. Young NS, Brown KE. Parvovirus B19. N Engl J Med. 2004;350:586-97.
4. Sokal EM, Melchior M, Cornu C, Vandenbroucke AT, Buts JP, Cohen BJ, Burtonboy G. Acute parvovirus B19 infection associated with fulminant hepatitis of favourable prognosis in young children. The Lancet. 1998;352:1739-41.
5. Brennan PN, Donnelly MC, Simpson KJ. Systematic review: non A-E, seronegative or indeterminate hepatitis; what is this deadly disease?. Aliment Pharmacol Ther. 2018;47(8):1079-91.
6. Ho JK, Tha SP, Coupland R, DalalBI, BowieWR,Sreenivasan GM, et al. Parvovirus B19 in an immunocompetent adult patient with acute liver failure: an underdiagnosed cause of acute non-AE viral hepatitis. Can J Gastroenterol Hepatol. 2005;19(3):161-2.
7. Hatakka A, Klein J, He R, Piper J, Tam E, Walkty A. Acute hepatitis as a manifestation of parvovirus B19 infection. J Clin Microbiol. 2011;49(9):3422-4.
8. Islek A, Keskin H, Agın M, Aksungur N, Korkut E, Ozturk G. Parvovirus B19 Infection as a Rare Cause of Fulminant Liver Failure: A Case Report. Transplant Proc. 2019;51(4): 1169-1171.
9. Leon LA, Alves AD, Garcia RD, Melgaço JG, de Paula VS, Pinto MA. Parvovirus B19 infection in a fatal case of acute liver failure. Pediatr. Infect. Dis. J.2017;36(12):e355-8.
10. Pinho J.R.R., Alves V.A.F., Vieira A.F., Moralez M.O.S., Fonseca L.E.P., Guz B. *et al*. Detection of human parvovirus B19 in a patient with hepatitis. Braz J Med Biol Res. 2001;34(9): 1131-1138.
11. Ergaz Z, Ornoy A. Parvovirus B19 in pregnancy. Reprod Toxicol. 2006;21(4):421-35.
12. Miller E, Fairley CK, Cohen BJ, Seng C. Immediate and long term outcome of human parvovirus B19 infection in pregnancy. BJOG: An International Br J Obstet Gynaecol. 1998;105(2):174-8.
13. Diaz F,Collazos J. Hepatic dysfunction due to parvovirus B19 infection. J Infect Chemother. 2000;6(1): 63-4.
14. Langnas AN, Markin RS, Cattral MS, Naides SJ. Parvovirus B19 as a possible causative agent of fulminant liver failure and associated aplastic anemia. Hepatology. 1995;22(6): 1661-5.
15. Krygier DS, Steinbrecher UP, Petric M, Erb SR, Chung SW, Scudamore CH, Buczkowski AK, Yoshida EM. Parvovirus B19 induced hepatic failure in an adult requiring liver transplantation. World J Gastroenterol: WJG. 2009; 15(32):4067.

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