LETTER TO THE EDITOR

(J Bangladesh Coll Phys Surg 2010; 28: 64-65)

(1). A comprehensive and time honored review on **"Epidemiology of Hepatitis C Virus infection"** of the journal of Bangladesh College of Physicians and Surgeons of September 2009, Vol 27, No 3.

To the Editor in chief: We have gone through the review and we have few important comments on this review.

Acute HCV is asymptomatic in approximately 84% of cases and is usually not recognized clinically.1 Approximately 85% of those infected with HCV will develop chronic infection and 15% will spontaneously clear virus, which is higher in children (45%). 20-30% of chronic HCV patient develop cirrhosis over 10-20 years of infection.1 HCV cirrhosis increases the risk of Hepatocellular carcinoma (HCC) dramatically. It is estimated that between 2 and 6.7% of all patients with HCV cirrhosis will develop HCC over 10 years and annual risk is 1-4%.1 It is rare that HCC will develop in patients without cirrhosis or advanced fibrosis (unlike HBV infection). Global prevalence of chronic hepatitis C is estimated to average 3% (ranging from 0.1-5% in different countries). 2 Intravenous drug uses remains the main mode of transmission, sexual and intra-familial spread is believed to be very low (unlike HIV). 2 The cost of investigation, treatment and monitoring therapy out of reach at the millions of sufferer's worldwide.2

We should pay more attention to prevent infection of HCV. Currently there is no vaccine available. Efficacy of standard immunoglobulin after needle prick, sexual or perinatal exposure to HCV has not been determined and is not recommended.1 All precautions to prevent infection of HCV should target reduction of transmission of the virus, screen of blood donors, treatments of blood products, safe injection practice and strongly discourage illegal drug use will minimize the spread of lethal infection in future.

References:

1. Current medical diagnosis and treatment Gastroenterology, 2nd edition McGraw Hill, 2003, 554-559.
2. Disease of the Liver and Biliary System 11th edition, Sheila Sherlock and James Dooley 2002 Blackwell;305-316.
3. Sultan MT, Rahman MM, Begum S. Epidemiology of Hepatitis C virus infection, J Bangladesh Coll Phy Surg 2009;27;3:160-162.

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Author's Reply

We thank Maj. Gen. (Dr.) M A Moyeed Siddiqui and Dr. Md. Mahmudur Rahman Siddiqui for their interest in our article. We were highly delighted to read their letter with few important comments on "Epidemiology of Hepatitis C Virus Infection". Since we only reviewed the prevalence, genotype data, transmission risk and prevention, we did not mention the elaborate data regarding percentage of asymptomatic infection, virus clearance, cirrhosis of liver and development of hepatocellular carcinoma. However we gladly accept the additional information, they had provided but regarding prevalence, transmission and prevention, all the information discussed in our article are consistent with their comments.

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Ref:

Siddiqui MAM, Siddiqui MMR. Letter to the Editor-Epidemiology of Hepatitis C Virus Infection, J Bangladesh Coll Phy Surg 2010

(2). About **"Familial Hypokalaemic Periodic Paralysis– A Case Report"** of the journal of Bangladesh College of Physicians and Surgeons of September 2009, Vol-27, No-3.

To the Editor in Chief: I have gone through this case report and have few comments. It is the clinical feature and serum electrolyte report which are usually sufficient for the diagnosis of Hypokalaemic Periodic Paralysis (HypoKPP). Though it mimics Guillain Barre Syndrome (GBS), lumber puncture for CSF study is usually not necessary to differentiate HypoKPP from GBS. The CSF findings suggestive of GBS usually appear at the end of first week of illness and by this time patients of HypoKPP improves with the treatment with potassium[1].

In this case report there is a mistake in the arrangement of the ECG tracings in fig: 1(b) and fig: 2(b) as the descriptions mentioned below the figures are opposite to each other[2].

References :

1. Hauser SL, Asbury AK. Guillain Barre syndrome and other immune mediated neuropathies. In Fauci AS, Kasper DL,Longo DL,Braunwald E, Hauser SL,Jameson JL,Loscalzo J. Harrison's Principles of Internal Medicine 17th ed. New York.McGraw-hill Companies Inc. 2008: Vol-2, p 2691-2.
2. Majumder MSH. Familial Hypokalaemic Periodic Paralysis– A case report. J Bangladesh Col Phy Surg 2009:27; 3:166-168

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Author's Reply

I thank Dr. Ahmed Hossain for his comments. I agree that CSF protein level may not rise until the end of the first week in case of Guillain Barre syndrome (GBS). Regarding CSF protein in GBS, it may be mentioned here that tau protein may be elevated within first few days of symptoms; a predictor of residual deficits[1]. However, the reported case, being familial hypokalaemic periodic paralysis, I emphasized on clinical course, electrolytic balance and ECG changes.

I do apologies to you and all other readers on behalf of editorial staff/ press staff regarding the interchange of two ECG tracing scans (a gross mistake) which have been misled the readers by this error[2].

Reference:

1. Hauser SL, Asbury AK. Guillain-Barre syndrome and other immune mediated neuropathies. In Fauci AS, Kasper DL, Longo DL, Braunwald E, Hauser SL, Jameson IL, Loscalzo J. Harrison's Principles of Internal Medicine 17th ed. New York. McGraw-Hill Companies Inc. 2008: Vol-2, p 2669-70.
2. Majumder MSH. Familial Hypokalaemic Periodic Paralysis - A case report. 3 Bangladesh Col Phy Surg 2009:27; 3:166-168.

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