STUDY OF BIOCHEMICAL PARAMETERS IN H1N1 PATIENTS.

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INTRODUCTION
Influenza is an acute respiratory illness caused by infection with influenza viruses. The illness affects the upper and/or lower respiratory tract and is often accompanied by systemic signs and symptoms such as fever, cough, headache, myalgia, and weakness. Influenza virus is a single-stranded ribonucleic acid (RNA) that belongs to members of the Orthomyxoviridae family, of which influenza A, B, and C viruses constitute three separate genera. Swine influenza (H1N1) is caused by influenza A. On June 11, 2009, there was an outbreak of a new strain of influenza A virus subtype H1N1 which resulted in a flu pandemic officially declared by the World Health Organization (WHO) to be the first influenza pandemic of the 21st century.[1] The new strain is thought to be the result of reassortment of strains of influenza A virus subtype (H1N1). Two new capabilities-human to human spread and enhanced virulence. The occurrence of liver involvement during influenza infection is intriguing owing to the virus characteristic of only infecting the epithelial cells of the respiratory tract.[2] Although the liver may not be the primary target organ of viral infection, it can be collateral damaged.[3] Hepatic changes may be a consequence of the immune response to viral antigens with a close topographic association between the presence of viral antigens and the associated inflammatory infiltrates in liver.[4]

AIMS & OBJECTIVE:

- To analyse the serum level of, SGOT, SGPT, ALP, total bilirubin & urea, creatinine in H1N1 patients
- To compare values of, SGOT, SGPT, ALP, total bilirubin, urea and creatinine in diagnosed case of H1N1 and controls.

MATERIALS AND METHODS

- SOURCE OF DATA: All the patients who were H1N1 positive, and H1N1 negative but with similar history, were taken.
- METHODS OF COLLECTION OF DATA: Procedure for collection of blood sample was explained completely to patients and an informed consent was taken, blood sample was collected under proper aseptic care.
- Investigation performed were – Serum SGOT, SGPT, ALP, Total Bilirubin, Urea and Creatinine were done.

RESULT

Biochemical parameters viz LFT – Liver function tests (SGOT, SGPT, ALP and total bilirubin) and KFT – kidney function tests (urea, creatinine) were deranged. P Value found to be <0.05 which is significant.

CONCLUSION

H1N1 might be a cause of transient elevation of liver function tests and kidney function tests. However, further studies are needed, not only to investigate the pathophysiology of hepatitis and nephritis due to H1N1 infection, but also to create a guideline regarding monitoring of multi-organ involvement due to the virus.

KEYWORDS: H1N1/Influenza, LFT, KFT

ABSTRACT

Introduction: H1N1 flu, also known as swine flu Seasonal influenza epidemics, currently involving influenza A(H3N2), A(H1N1) and B strains, affect 10% to 20% of the human population each year. Although most people infected with a seasonal influenza strain recover in less than 2 weeks, young children and adults ≥65 years old, are at higher risk of developing serious influenza complications requiring hospitalization and potentially leading to death.

Aim: To study the biochemical parameters in diagnosed H1N1 patients

Materials and Methods: 100 samples which includes 50 H1N1 diagnosed cases and 50 healthy controls.

1) Serum urea and serum creatinine by kit based method on automated auto analyser.
2) Serum SGOT, SGPT, ALP and total bilirubin by kit based method on automated auto analyser.

Results: Biochemical parameters viz LFT – Liver function tests (SGOT, SGPT, ALP and total bilirubin) and KFT – kidney function tests (urea, creatinine) were deranged. P Value found to be <0.05 which is significant.

Conclusion: H1N1 might be a cause of transient elevation of liver function tests and kidney function tests. However, further studies are needed, not only to investigate the pathophysiology of hepatitis and nephritis due to H1N1 infection, but also to create a guideline regarding monitoring of multi-organ involvement due to the virus.
DISCUSSION
• Total 82 patients, among which 43 diagnosed cases of H1N1 and 39 controls which were H1N1 negative were taken and biochemical parameters viz, SGOT, SGPT, ALP, Total bilirubin, Urea and Creatinine were done.
• Influenza A/H1N1 virus usually affects the respiratory tract, but the pathogenesis is not yet fully understood.
• The incidence of liver & kidney injury in influenza has not been established and the pathogenesis is still not well understood.
• Production of cytokines (e.g., TNF-alpha, IL-6, IL-8, IL-10, and interferon alpha, interferon beta, and interferon gamma) are responsible for the oxidative stress leading to the hepatocyte & renal injury.[5]
• Polakos et al. introduced a so called collateral damage model and revealed that liver lesions occur because of the systemic immune response (SIRS) where, in the absence of viral antigens, the hepatocytes are damaged by viral-specific CD8+ T cells.[4]
• Fislova et al. studied the multi-organ involvement of human strain H1N1 in mice, and concluded that the dissemination of the virus from the lung to other organs is by the process of transient viremia [5].
• The study also concluded that the production of cytokine and chemokine induction might be the cause of multi-organ involvement due to respiratory H1N1 infection [5].
• Bermejo-Martin et al. observed higher levels of pro-inflammatory cytokines in pandemic 2009 influenza A/H1N1, and some of these can cause liver injury [6].
• Considering new emerging evidence showing that liver enzymes are associated with oxidative stress and inflammation, we can presume that influenza promotes a intense SIRS.[7].
• SIRS finally produces perfusion disturbances of the tissue which contributes to hepatocellular damage and hyper transaminemia.

CONCLUSION
• Considering all the results of the above mentioned studies, it becomes evident that influenza A/H1N1 results in multiorgan involvement mainly liver and kidney.
• From our point of view, at least for the time being, probably the safest explanation of the mechanisms leading to liver and renal dysfunction during influenza A/H1N1 is generalized stress due to the multisystemic character of the infection, which is accompanied by a stormy release of cytokines that can cause direct liver and renal injury per se.
• We strongly believe that the close interplay between liver and renal function and the function of the immune system components will stimulate the researchers to re-evaluate the fluctuations of the parameters of liver and kidney and provide a new insight in the multisystemic effects of influenza A/H1N1 viral infections.

BIBLIOGRAPHY