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Sero-Prevalence of Hepatitis B and Hepatitis C in District Sialkot

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ABSTRACT

Hepatitis B and hepatitis C are peak overwhelming infectious conditions belonging to liver inflammation. According to the World Health Organization's hepatitis report, 96% of the 1.3 million hepatitis virus-related fatalities annually, of which 720,000 happened at the cirrhosis stage, were the result of chronic infections with the hepatitis B and hepatitis C viruses. **Objectives:** To assess the sero-prevalence of Hepatitis B and Hepatitis C in District Sialkot. **Methods:** A total of 1,737 blood samples were randomly collected from participants who visited the hospital for hepatitis screening from March 2018 to August 2018. All the samples were screened via Immune-chromatographic strip test. Statistical analysis was performed on data using IBM SPSS Statistics (Version 27). **Results:** A total of 15.5% samples tested positive, out of which 12.43% samples were hepatitis C viruses and 2.82% were hepatitis B viruses positive respectively. Overall prevalence was higher in male (16.60%) than female 14.19%. Sero-prevalence was high between the age group of 61-80 years. Likewise, among married persons (17.16%) as compared to unmarried (8.01%). **Conclusions:** It was concluded that a significant association of prevalence of hepatitis with both age and marital status ($p<0.001$) was observed, while no significant effect was on gender.

INTRODUCTION

Viral hepatitis is the most widely spread infectious condition of liver inflammation having a list of serotypes [1]. Both Hepatitis B virus (HBV) and Hepatitis C virus (HCV) infections are significant for causing chronic hepatitis and have become the chief cause of liver cirrhosis and hepatocellular carcinoma. Out of these two, hepatitis C had become severe sequelae. It can develop acute hepatitis, chronic hepatitis or a chronic carrier state and hepatocellular carcinoma [2]. Hepatitis B contamination is caused by the hepatitis B virus (HBV). It belongs to the Hepadenviridae family [3]. It is a member of the Orthohepadnavirus genus [4]. It is a partly double-stranded DNA virus. HBV have a constricted host range, which is restricted to humans and chimpanzees only. Out of these two, humans are the chief natural host for HBV [5]. HBV can cause tarnishing of hepatic cells of the liver and liver failure. At the late phase of infection, HBV may progress to hepatocellular carcinoma (HCC) and cirrhosis. There are two states of HBV infection, acute and chronic. Acute hepatitis frequently happens when the natural resistance is sound. Upon infection to liver cells, HBV has 4-6 weeks to an extensive 6-month incubation phase. Chronic Hepatitis B is the disease state if the perseverance of Hepatitis B (HB) antibodies and HBV exceeds 6 months [6]. It is related to a total 15-25% hazard of premature deaths from liver malignancy [7]. Hepatitis C virus (HCV) belongs to the family Flaviviridae and species of Hepacivirus [8]. It is a miniature, encapsulated virus of 55 nanometers. It is a single-stranded RNA virus and this strand is a positive sense strand [9]. HCV is accountable for liver scarring, liver cancer and liver failure after decades of infection. HCV move into the host cells via receptor-mediated endocytosis [10]. There are two states of hepatitis C first is Acute and second is chronic hepatitis. In the acute

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hepatitis C infection state, the onset of disease with clear symptoms ranges from 3-12 weeks after first exposure [11]. In chronic hepatitis C viral RNA remained persistent in blood. It occurs after at least 6 months or longer afterwards the inception of severe infection [12]. Approximately 2 to 10% of individuals with chronic HCV infection have co-infection with hepatitis B. This co-infection accelerates liver infection and increases the risk of liver cirrhosis, HCC, and even death [13]. Symptoms of HBV and HCV infection include jaundice, tiredness, anorexia, low appetite, malaise and severe weakness [11]. The basic treatment of HB viral infection is vaccination against the virus which has been accessible since 1982. This inoculation comprises a recombinant HBs Ag protein which is derived from yeast. This protein is effective in more than 95% of Immuno-competent receivers [14]. Up to now, the vaccine is not available for HCV [15]. Two commonly adopted HCV therapies are utilized for treating hepatitis. Firstly, is standard or PEGylated interferon Alfa and the second one is ribavirin. These two therapies ensure effectiveness in 40%–50% of treated suffering ones [16]. Among all the serotypes of hepatitis, HBV is the major necro-inflammatory agent in developing and under-developing countries. Approximately 248 million individuals are chronically septic by hepatitis B virus worldwide [17]. Around 780, 000 deaths occur per year due to HBV. Chronic infection is responsible for 650,000 deaths annually. Wisely, 130,000 deaths occur due to acute hepatitis [18]. There are about 370 million carriers of HBV [19]. The prevalence of Hepatitis B is at a peak of 6.2% in the Pacific Regions of West [7]. The World Health Organization has associated hepatitis C with a 'viral time bomb'. This time bomb could be the chief cause of billions of deaths around the world. About 350, 000 to 500, 000 individuals are expected to expire each year due to hepatitis C virus. WHO demonstrated that about 3% of the world's population, about 180 hundred thousand people is disease-ridden by HCV. HCV is accountable for 50-76% of entire liver malignancy cases [7]. Pakistan is currently facing the overwhelming infectious condition of hepatitis with a prevalence rate between 0.3-33 percent [20]. It is assessed that approximately 150, 000 individuals are world widely diseased with hepatitis C virus [21]. One study predicted 10% frequency of HBV and 6.7% of HCV among women in Pakistan [22]. Records from diverse regions of Pakistan revealed a 2.4% frequency of HBV. Temperate to the heavy dominance of HBV infection was observed in Punjab, Baluchistan, Sindh and (KPK) Khyber Pakhtunkhwa. About 29% of chronic liver disease cases and 8% of hepatocellular carcinoma cases are HCV seropositive in Pakistan [23]. This study aims to assess the sero-prevalence of Hepatitis B and Hepatitis C in District Sialkot.

METHODS

This cross-sectional study was conducted in district Sialkot, located at Latitude 32°30'N and Longitude 74°31'E in the northeast province of Punjab, Pakistan. From March 2018 to August 2018, 1,737 blood samples were randomly collected from Abdul-Sattar lab's free hepatitis screening camp. The minimum sample size (385) was calculated with the online tool "Sample Size Calculator" at 5% error. All Individuals from District Sialkot, both married and unmarried, infected with HBV+/HCV+, aged (0->80) years, and any gender (male and female) who visited Abdul-Sattar lab's free hepatitis screening camp. Patients with any other infection and having HAV+ were excluded. Distinct information including the history, sex, age and marital status of each patient was obtained by interviewing the patient via a questionnaire. For screening of Hepatitis, a standard diagnostic kit was used (Bioline kit). The presence of the control line and test line confirms a positive test. Faint test lines were regarded as weak positive. The presence of the control line alone confirms negative results. Statistical analysis (Chi-square) was applied to data using IBM SPSS Statistics (Version 27).

RESULTS

Out of 1,737 samples, 265 (15.25%) were positive for hepatitis B and C. While 49 (2.82%) were HBV positive and 216 were HCV positive (12.43%). Out of 765 males 127(16.60%) were positive for hepatitis. A total of 3.52% were HBV and 13.07% were HCV positive. Among 972 females 2.26% were HBV positive and 11.93% were HCV positive. Non-significant difference (p=0.204) was observed between the prevalence of hepatitis in two genders (Table 1).

Table 1: Gender Wise Prevalence of HBV+ and HCV + Patients

Gender	Total Patients	Positive Patients (%)	HBV+ Patients (%)	HCV+ Patients (%)	p-value*
Male	765	127 (16.60%)	27 (3.52%)	100 (13.07%)	0.204
Female	972	138 (14.19%)	22 (2.26%)	116 (11.93%)	
Total	1737	265 (15.25%)	49 (2.82%)	216 (12.43%)	

Distribution of HBV and HCV in different age groups from 0-20, 21-40, 41-60, 61-80 and above 80 years of age are mentioned in Table 2. The highest HBV and HCV were noted in the 61-80 years' age group. No positive patients above 80 years were noted for HBV infection while the lowest HCV infection was noted in the 0-20-year age group. The chi-square test showed a significant difference (p=0.0001) between the prevalence of hepatitis in different age groups (Table 2).

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Table 2: Age-Wise Distribution of HBV+ and HCV+ Patients

Age (Years)	Total Patients	Positive Patients	HBV+ Patients	HCV+ Patients	p-Value
0-20	151 (8.69%)	10 (6.62%)	4 (2.64%)	6 (3.97%)	0.0001
21-40	1,012 (58.26%)	127 (12.54%)	29 (2.86%)	98 (9.68%)	
41-60	452 (26.02%)	95 (21.01%)	12 (2.65%)	83 (18.36%)	
61-80	110 (6.33%)	30 (27.27%)	4 (3.63%)	26 (23.63%)	
>80	12 (0.69%)	3 (25%)	0 (0.00%)	3 (25%)	
Total	1737	265 (15.25%)	49 (2.82%)	216 (12.43%)	

Out of 1,375 married patients, 236 (17.16%) were positive overall. Among married positive patients 2.69% were HBV+ and 14.47% were HCV+. While unmarried patients were 8.01% positive, out of it 3.31% were HBV+ and 4.69% were HCV+. A significantly high (p=0.000016) prevalence of hepatitis was observed in married persons (Table 3).

Table 3: Prevalence of HBV+ and HCV+ According to Marital Status of Patients

Marital status	Total Patients	Positive Patients (%)	HBV+ Patients	HCV+ Patients	p-Value
Married	1,375 (79.15%)	236 (17.16%)	37 (2.69%)	199 (14.47%)	0.0-00016
Unmarried	362 (20.84%)	29 (8.01%)	12 (3.31%)	17 (4.69%)	
Total	1737	265 (15.25%)	49 (2.82%)	216 (12.43%)	

Among 765 male samples, 0.13% were weak HBV+ and 0.26% were HCV+. While in female samples 0.61% were weak positive only for HCV. No significant (p=0.516) difference was noted gender-wise in weak hepatitis-infected patients (Table 4).

Table 4: Gender-Wise Distribution of Weak HBV+ and HCV+

Gender	Total Patients	Weak +ve Patients (%)	Weak HBV+ Patients (%)	Weak HCV+ Patients (%)	p-Value
Male	765 (44.04%)	3 (0.39%)	1 (0.13%)	2 (0.26%)	0.516
Female	972 (55.95%)	6 (0.61%)	0 (0.00%)	6 (0.61%)	
Total	1737	9 (0.51%)	1 (0.05%)	8 (0.46%)	

There are some exceptional cases of showing weak positive results with testing regarding age. Among age groups of 0-20, 21-40, 41-60, 61-80 and above 80 years, weak HBV was noted only in the 61-80 years' age group. Likewise, weak HCV prevalence was in the age group of 0-20, 21-40, and 41-60 years. A significant difference (p=0.015) was observed between the prevalence of hepatitis in different age groups with weak infection (Table 5).

Table 5: Age-Wise Distribution of Weak HBV+ and HCV+ Patients

Age (Years)	Total Patients (%)	Weak +Ve Patients (%)	Weak HBV+ Patients (%)	Weak HCV+ Patients (%)	p-Value
0-20	151 (8.69%)	1 (0.66%)	0 (0.00%)	1 (0.66%)	0.015
21-40	1,012 (58.26%)	4 (0.39%)	0 (0.00%)	4 (0.39%)	
41-60	452 (26.02%)	3 (0.66%)	0 (0.00%)	3 (0.66%)	
61-80	110 (6.33%)	1 (0.90%)	1 (0.90%)	0 (0.00%)	
>80year	12 (0.69%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	
Total	1737 (100%)	9 (0.51%)	1 (0.05%)	8 (0.46%)	

According to the marital status, 9 samples showed weak positivity out of whom 8 were married and 1 was a bachelor. No significant (p=0.47) difference was noted in married and unmarried weak hepatitis-infected patients (Table 6).

Table 6: Prevalence of Weak HBV+ and Weak HCV+ Patients According to Martial Status of Patients

Marital status	Total Patients (%)	Weak Positive Patients (%)	Weak HBV+ Patients (%)	Weak HCV+ Patients (%)	p-Value
Married	1,375 (79.15%)	8 (0.58%)	1 (0.07%)	7 (0.50%)	0.47
Unmarried	362 (20.84%)	1 (0.27%)	0 (0.00%)	1 (0.27%)	
Total	1,737 (100%)	9 (0.51%)	1 (0.05%)	8 (0.46%)	

There were also some unique cases in which some patients were suffering from HBV and HCV at the same time. Out of 1,737 patients, 0.28% of samples had shown their positivity to both HBV and HCV screening tests. Among these 5 patients, 3 were male and 2 were female. All of these 5 patients belong to 21-40 years of age group and were married.

DISCUSSION

HBV and HCV viruses are widespread in South East Asia including Pakistan. It is expected that nearly 10,00, 000 people are living with hepatitis in Pakistan. Several studies have been conducted to estimate the seroprevalence of HBV and HCV in Pakistan and reported different rates of infection from different areas. In 2008, a report presented 7.3% HCV infection and 2.2% HBV infection at Sir Ganga Ram Hospital, Lahore [24]. In 2006, 1.7% to 5.5% HBV prevalence was reported among children of Karachi, Pakistan [25]. In 1996, 1.18% prevalence of HCV and 2.48% prevalence of HBV among blood donors in southern Pakistan was reported [26]. In the Punjab Regiment Centre Mardan, sero-prevalence of HCV reported was 3.69% and HBV was 3.24% [27]. Our study also reported a low prevalence of Hepatitis B (2.82%) in contrast to Hepatitis C (12.43%) indicating people are more susceptible to Hepatitis B or there may be differences in the mode of transmission of both type of infections. In a study, 7.56% Seroprevalence of HCV with a male predominance of 10.84% was reported in Fauji Foundation Hospital, Rawalpindi [28]. Our results are significant to these results with the predominant prevalence of HCV and HBV in males. In Rawalpindi and Islamabad, the dominance of HBV over HCV was reported as 2.6% [29]. These results of HBV prevalence are by our results showing 2.82% HBV prevalence but HCV is dominant in Sialkot as compared to the HBV in Rawalpindi and Islamabad. A study reported a 3.5% prevalence of HCV in District Mansehra. The incidence of HCV infection was high 4% in males as compared to females 2% [30]. These results of our study also show a higher prevalence of HCV among males. Gender-wise incidence was documented to be higher 19.1% in male than in female 12.7% from the district Peshawar is

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analogous to our results [31]. Our study shows high surveillance of HCV and HBV among males and married people. The main routes of transmission of HBV and HCV are blood transfusion and, the use of contaminated syringes, and razors. Males are more susceptible to infection due to having insecure sexual relationships with numerous partners and using contaminated syringes at barber shops.

CONCLUSIONS

It was concluded from this research work that there is an elevated incidence of HCV than HBV in District Sialkot, Pakistan. A high prevalence of HBV and HCV is seen in male, in people belonging to the 60-80 years' age group and in married ones. Massive caution should be employed regarding modes of transmission of HBV and HCV. Awareness campaigns for HBV and HCV should be conducted to control the infection.

Authors Contribution

Conceptualization: AWQ
Methodology: AWQ, FJ, MM
Formal analysis: AWQ
Writing review and editing: AWQ, FJ

All authors have read and agreed to the published version of the manuscript

Conflicts of Interest

All the authors declare no conflict of interest.

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REFERENCES

[1] Weinbaum CM, Mast EE, Ward JW. Recommendations for Identification and Public Health Management of Persons with Chronic Hepatitis B Virus Infection. Hepatology.2009 May; 49 (S5): S35-44. doi: 10.1002/hep.22882.

[2] 2Basit A, Rahim K, Ahmad I, Shafiq M, Mushtaq S, Shaheen H *et al.* Prevalence of Hepatitis B and C Infection in Pakistan. Journal of Infection and Molecular Biology.2014 May; 2(3): 35-8. doi: 10.14737/jimb.2307-5465/2.3.35.38.

[3] Zanetti AR, Van Damme P, Shouval D. The Global Impact of Vaccination Against Hepatitis B: A Historical Overview. Vaccine.2008 Nov; 26(49): 6266-73. doi: 10.1016/j.vaccine.2008.09.056.

[4] Norder H, Couroucé AM, Coursaget P, Echevarria JM, Lee SD, Mushahwar IK *et al.* Genetic Diversity of Hepatitis B Virus Strains Derived Worldwide: Genotypes, Sub-Genotypes, and HBs Ag Subtypes. Intervirology.2004 Nov; 47(6): 289-309. doi: 10.1159/000080872.

[5] Rehermann B and Nascimbeni M. Immunology of Hepatitis B Virus and Hepatitis C Virus Infection. Nature Reviews Immunology. 2005 Mar; 5(3): 215-29. doi: 10.1038/nri1573.

[6] Seeger C and Mason WS. Molecular Biology of Hepatitis B Virus Infection. Virology. 2015 May; 479: 672-86. doi: 10.1016/j.virol.2015.02.031.

[7] World Health Organization. Global Health Sector Strategy On Viral Hepatitis 2016-2021. Towards Ending Viral Hepatitis. World Health Organization. 2016.

[8] Ferri C, Sebastiani M, Giuggioli D, Colaci M, Fallahi P, Piluso A *et al*. Hepatitis C Virus Syndrome: A Constellation of Organ and Non-Organ Specific Autoimmune Disorders, B-Cell Non-Hodgkin's Lymphoma, and Cancer. World Journal of Hepatology.2015 Mar; 7(3): 327. doi:10.4254/wjh.v 7. i3.327.

[9] Adams MJ, Lefkowitz EJ, King AM, Harrach B, Harrison RL, Knowles NJ *et al*. Changes to Taxonomy and the International Code of Virus Classification and Nomenclature Ratified by the International Committee on Taxonomy of Viruses (2017). Archives of virology.2017 Aug; 162(8): 2505-38. doi: 10.1007 /s0 0705-017-3358-5.

[10] Tang H and Grisé H. Cellular and Molecular Biology of HCV Infection and Hepatitis. Clinical Science.2009 Jul; 117(2): 49-65. doi: 10.1042/CS20080631.

[11] Thimme R, Oldach D, Chang KM, Steiger C, Ray SC, Chisari FV. Determinants of Viral Clearance and Persistence During Acute Hepatitis C Virus Infection. The Journal of Experimental Medicine.2001 Nov; 194 (10): 1395-406. doi: 10.1084/jem.194.10.1395.

[12] Alter MJ, Kruszon-Moran D, Nainan OV, McQuillan GM, Gao F, Moyer LA *et al*. The Prevalence of Hepatitis C Virus Infection in the United States, 1988 Through 1994. New England Journal of Medicine. 1999 Aug; 341(8): 556-62. doi: 10.1056/NEJM199908193410802.

[13] Jamma S, Hussain G, Lau DT. Current Concepts of HBV/HCV Coinfection: Coexistence, But Not Necessarily in Harmony. Current Hepatitis Reports. 2010 Nov; 9: 260-9. doi: 10.1007/s11901-010-0060-4.

[14] Sheldon J and Soriano V. Hepatitis B Virus Escape Mutants Induced by Antiviral Therapy. Journal of Antimicrobial Chemotherapy.2008 Apr; 61(4): 766-8. doi: 10.1093/jac/dkn014.

[15] Umar M and Bilal M. Hepatitis C, A Mega Menace: A Pakistani Perspective. Journal of Pioneering Medical Sciences.2012 Jun; 2: 68-72.

[16] Thomas DL, Astemborski J, Rai RM, Anania FA, Schaeffer M, Galai N *et al*. The Natural History of

DOI: https://doi.org/10.54393/pjhs.v5i12.990

Hepatitis C Virus Infection: Host, Viral, and Environmental Factors. Journal of American and Medical Association.2000 Jul; 284(4): 450-6. doi: 10.1001/jama.284.4.450.

[17] Schweitzer A, Horn J, Mikolajczyk RT, Krause G, Ott JJ. Estimations of Worldwide Prevalence of Chronic Hepatitis B Virus Infection: A Systematic Review of Data Published Between 1965 and 2013. The Lancet. 2015 Oct; 386(10003): 1546-55. doi: 10.1016/S0140-67 36(15)61412-X.

[18] Edmunds WJ, Medley GF, Nokes DJ, Hall AJ, Whittle HC. The Influence of Age On the Development of the Hepatitis B Carrier State. Proceedings of the Royal Society of London. Series B: Biological Sciences. 1993 Aug; 253(1337): 197-201. doi: 10.1098/ rspb .19 93 .0102.

[19] Bostan N, Naeem M, Afzal MS, Shah ZH, Mustafa I, Arshad M *et al*. Sero-Prevalence of Hepatitis B and C Virus from Rural Areas of Northern Punjab (Sargodha District), Pakistan. Tropical Biomedicine. 2016; 33(4): 599–607.

[20] Mahmood M, Anwar MA, Khanum A, Zaman N, Raza A. Distribution and Clinical Significance of Hepatitis B Virus Genotypes in Pakistan.BioMed Central Gastroenterology.2016 Dec; 16: 1-9. doi: 10.1186/s128 76-016-0513-5.

[21] Parlati L, Hollande C, Pol S. Treatment of Hepatitis C Virus Infection. Clinics and Research in Hepatology and Gastroenterology.2021 Jul; 45(4): 101578. doi: 10 . 1016/j.clinre.2020.11.008.

[22] Khan JK, Lone DS, Hameed A, Munim R, Bhatti M, Khattak AA *et al*. Evaluation of the Performance of Two Rapid Immune-Chromatographic Tests for Detection of Hepatitis B Surface Antigen and Anti-HCV Antibodies Using ELISA Tested Samples. Annals of King Edward Medical University.2010; 16(1 SI). doi: 10.21649/akemu.v16i1%20SI.166.

[23] Tong MJ, El-Farra NS, Reikes AR, Co RL. Clinical Outcomes After Transfusion-Associated Hepatitis C. New England Journal of Medicine.1995 Jun; 332(22): 1463-6. doi: 10.1056/NEJM199506013322202.

[24] Ali SA, Donahue RM, Qureshi H, Vermund SH. Hepatitis B and Hepatitis C in Pakistan: Prevalence and Risk Factors. International Journal of Infectious Diseases.2009 Jan; 13(1): 9-19. doi: /10.10 16/j. ijid. 20 08.06.019.

[25] Jafri W, Jafri N, Yakoob J, Islam M, Tirmizi SF, Jafar T *et al*. Hepatitis B and C: Prevalence and Risk Factors Associated with Sero-Positivity among Children in Karachi, Pakistan. BioMed Central Infectious Diseases. 2006 Dec; 6: 1-0. doi: 10.1186/1471-2334-6-101.

[26] Kakepoto GN, Bhally HS, Khaliq G, Kayani N, Burney IA, Siddiqui T *et al*. Epidemiology of Blood-Borne Viruses: A Study of Healthy Blood Donors in Southern Pakistan. The Southeast Asian Journal of Tropical Medicine and Public Health. 1996 Dec; 27(4): 703-6.

[27] Mirza IA, Mirza SH, Irfan S, Siddiqi R, Tariq WU, Janjua AN. Sero-Prevalence of Hepatitis B and C in Young Adults Seeking Recruitment in Armed Forces. Pakistan Armed Forces Medical Journal.2006 Jun; 56(2): 192-7.

[28] Chaudhary IA, Khan SS, Majrooh MA, Alvi AA. Sero-Prevalence of Hepatitis-B and C among the Patients Reporting in Surgical OPD at Fauji Foundation Hospital, Rawalpindi: Review of 5-Year Literature. Pakistan Journal of Medical Sciences.2007 Jul; 23(4): 514.

[29] Waheed Y, Shafi T, Safi SZ, Qadri I. Hepatitis C Virus in Pakistan: A Systematic Review of Prevalence, Genotypes and Risk Factors. World Journal of Gastroenterology.2009 Dec; 15(45): 5647. doi: 10.3 7 4 8/wjg.15.5647.

[30] Ali A, Ahmad H, Ali I, Khan S, Zaidi G, Idrees M. Prevalence of Active Hepatitis C Virus Infection in District Mansehra Pakistan. Virology Journal.2010 Dec; 7: 1-5. doi: 10.1186/1743-422X-7-334.

[31] Ilyas M and Ahmad I. Chemiluminescent Microparticle Immunoassay Based Detection and Prevalence of HCV Infection in District Peshawar Pakistan. Virology Journal.2014 Dec; 11: 1-5. doi: 10.1186/1743-422X-11-127.