

Venue: GIZA
23rd August 2007
1515-1630 hr

Symposium 6C: Molecular diagnosis of viral disorders

S6C-1. Molecular diagnosis of viral disorders: HIV

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The advent of highly active anti-retroviral therapy (HAART) has resulted in significant reductions in opportunistic infections and deaths among HIV-positive patients. These benefits continue, but the incidence of treatment failures is increasing, mainly because of the emergence of HIV drug resistance mutations. The good news is that we, as clinical laboratorians, currently have tools for identifying these mutations and aid clinicians to choose the best drugs for a certain patient. HIV genotyping and phenotyping are in the forefront of personalized medicine, on the “virus-side”. But there are also important advances in the “patient-side”, such as pharmacogenetics (PGx) being used to evaluate patient’s polymorphisms and, consequently, patient’s individualized probability to develop adverse reactions prior to administering a certain drug. It is important for the general microbiologist and clinical pathologist (non-HIV expert) to understand some basic principles of HIV drug management. This presentation will focus on addressing the following questions:

- 1) How does HIV acquire resistance to certain drugs?
- 2) What can be done today to predict a particular drug resistance on clinical labs?
- 3) Do we have enough clinical evidence to rely on such tests?
- 4) Is PGx testing in this area clinically useful? In what situations?

S6C-2. Hepatitis C

Balaratti C

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Hepatitis C will be one of the major health problems in this century. About 200 million people have the virus. In the world, the percentage of carriers is approximately between 0.5 to 2.5%. In Brazil, we have 5 million carriers and in some countries in Africa, this problem increased with the co infection with HIV and Hepatitis B. About 90% of HCV carriers do not know the diagnostic and do not have access to the treatment. The latest study shows prevalence increase in IVD (Intra Venous Drugs) users. This lecture will show the new tools to diagnose the HCV, like the recent advanced in serology and molecular biology like real time PCR and another methodology to monitoring the virus.

S6C-3. Review on Hepatitis B and chronic Hepatitis B

Alcantara FFP

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Even though HBV has been extensively studied and characterized since its discovery, in 1967, it is still a large burden for human population. According to WHO, 2 billion people alive were infected by HBV sometime in their lifetime. There are 4 million new cases each year and 1 million chronic cases. Besides, there are 350 million individuals chronically infected with HBV (100 millions in China), producing 1 millions deaths a year. Acute infection in the adult is easily recognized through immunological assays; however, sometimes problems may arise in the interpretation of results in immunocompromised patients or in the transplantation setting. State of the art management of HBV requires in depth knowledge of the viral life cycle: adequate use and follow up with molecular tests for viral load and viral genotyping, as well as their clinical implications. The very high frequency of HBe null mutations (preCore and Core promoter mutations) has precluded this antigen as the sole marker for replication and was replaced by viral load assays. Viral load assays, both commercial and “in house” assays are now widespread and have gained larger acceptance and applications than previously thought. Both amplification and non amplification methods can be used, even if targeting different regions of the genome. Genotypes associated with different clinical outcomes, as seen in HCV, are an area of intense research. Identification of determined viral regions has become necessary. Characterization of the most common mutations affecting viral genes coding for HBs antigen, HBe antigen, and HBV polymerase can be done by several methods. Methods for circumventing the high cost of sequencing, such as, strip hybridization have been popularized. HBV treatment has evolved quickly in recent years and new drugs are now in the pipeline. Their impact on the disease requires careful evaluation of the consequences for viral load titers, mutation susceptibility and ultimately disease progression. Viral escape through resistance mutations (such as YMDD), Lamivudine or Type I Interferon resistance mechanisms have been characterized.