HIV-HCV Coinfection

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Background

Given the common modes of transmission of HIV and HCV, coinfection is common, particularly among intravenous drug users and haemophiliacs. In the EuroSIDA cohort study, 33% of over 3,000 patients with HIV infection had evidence of HCV infection[1]. In the United States, about 30% of the 800,000 individuals with HIV infection are coinfected with HCV[2]. 50 - 90% of persons who acquire HIV from injecting drugs are also HCV-infected. Over 50% of haemophiliacs who were exposed to non-screened, non-heat-treated blood products had HIV-HCV coinfection[3].

Impact of HIV infection on HCV disease progression

The immunodeficiency associated with HIV infection tends to accelerate the course of HCV disease progression. Serum HCV viral load is higher in patients with HIV-HCV coinfection than in patients with HCV infection alone[4]. HCV transmission becomes more efficient both sexually and perinatally. The development of symptomatic liver disease is more common[5], and the rate of fibrosis progression[6] and cirrhosis development appears accelerated[7].

As survival among HIV-infected patients increases with the use of HAART and prophylaxis against traditional opportunistic pathogens, HCV-related morbidity and mortality among HIV-infected patients showed increase. Indeed, HCV-related liver disease has become a major cause of hospital admission and deaths among HIV-infected persons[8].

Impact of HCV infection on HIV disease progression

Co-infection by HCV is also a detrimental prognostic factor for HIV disease. HCV coinfection was associated with a more rapid clinical and immunologic progression among HIV-infected patients with CD4 cell count > 600/mm[3,9]. A recent Swiss HIV cohort study demonstrated that the probability of progression to a new AIDS-defining clinical event or to death was independently associated

with HCV seropositivity[10]. The same study also showed that HCV seropositivity was associated with a smaller CD4 cell recovery after HAART.

HIV-HCV coinfection and HAART-associated hepatotoxicity

HCV is an independent risk factor for hepatotoxicity with HAART. Overall significant liver enzyme elevations are seen in approximately 15% of patients receiving HAART, but severe hepatotoxicity requiring discontinuation of therapy occurs in less than 10% of cases[11].

Drug metabolism may be impaired in patients with chronic liver disease. Hepatotoxicity from accumulation of toxic metabolites becomes more common similar to antituberculous therapy in patients with chronic liver disease. This intrinsic hepatotoxicity, if occurs, is usually dose-related, with delayed onset typically after several months e.g. stavudine, protease inhibitors, also nevirapine. Another mechanism is hypersensitivity reaction which may affect skin and liver, occurring early within a few days to weeks e.g. abacavir, nevirapine. A third mechanism may be immune reconstitution syndrome with flare of transaminases due to dramatic CD4 cell increase after HAART, similar to flare of latent CMV or mycobacterial infections after HAART.

The available evidence suggests that antiretroviral therapy can be safely administered to HIV-infected patients with chronic hepatitis C. However, their serum liver enzymes should be closely monitored.

Management

Management strategies include primary prevention of HCV infection, early detection of HCV coinfection, and measures to modify HCV disease progression in HIV-infected individuals.

Primary prevention of HCV infection

HIV-infected persons should be routinely asked about risk factors for acquiring HCV infection. These include occupational exposure to blood, injection drug use, and high-risk sexual practices like multiple sex partners or history of sexually transmitted disease. Patients with these risk factors should be educated to reduce their

risk of acquiring blood-borne and sexually transmitted infections.

Testing for HCV infection

All HIV-infected persons should be screened for HCV coinfection. Testing for HCV infection should be performed with EIA. Anti-HCV EIA positive patients should undergo confirmatory testing by supplemental antibody testing with RIBA or detection of HCV RNA with reverse transcriptase-PCR.

Certain pitfalls in interpretation of serological tests should be noted. Anti-HCV in patients with acute infection may remain undetectable with EIA for weeks after HCV acquisition. Some individuals with chronic HCV infection have intermittent viraemia, hence a single finding of undetectable HCV RNA should be interpreted cautiously. HIV-infected patients with advanced immunodeficiency may lose anti-HCV activity. Thus the presence of HCV RNA in blood should be assessed when acute or chronic HCV infection is suspected clinically in persons with negative anti-HCV.

Prevention of HCV disease progression

General advice

As for patients with chronic hepatitis C, persons with HIV-HCV coinfection should be advised not to drink excessive amount of alcohol. It might be prudent to avoid alcohol altogether as the harmful effect of occasional moderate alcohol use is unclear in chronic hepatitis C.

Vaccination against hepatitis A is advised because the risk of fulminant hepatitis appears to be increased[12]. Hepatitis A vaccine is safe for HIV-infected persons, and more than two-thirds of patients with advanced HIV infection can develop protective antibody response[13].

Evaluation of HCV disease

HCV disease severity should be assessed on the basis of medical history, physical examination, serum albumin, prothrombin time and platelet count. Serum ALT and HCV RNA are important to establish ongoing infection. Liver biopsy may provide important information about disease activity, stage of fibrosis, and exclude

alternative causes of liver disease. HCV genotyping may provide useful prognostic information regarding response and duration of therapy. HCV treatment should be recommended on the basis of fibrosis score. Scores of F1 to F4 is generally an indication for treatment. For genotype 2 or 3, treatment without biopsy is acceptable because response rate is very high with combination therapy.

HCV therapy

Patients with HCV-HIV coinfection may benefit from anti-HCV therapy, both to decrease the incidence of HCV-related advanced liver disease and to increase the effectiveness of and tolerance to HAART. When possible, treating HCV before HIV treatment is advocated to avoid the potential drug interactions between HAART and anti-HCV therapy.

Until recently, interferon-a plus ribavirin was the standard therapy for HCV infection. Pegylated interferon (PIFN), a long acting weekly injection preparation, has been shown to have a higher efficacy than standard interferon in the treatment of chronic hepatitis C. PIFN plus ribavirin is currently the preferred option as it has been shown to achieve sustained virological response rates of 42% for genotype 1, 82% for genotypes 2 or 3[14]. This combination was also demonstrated to reduce fibrosis progression in treated patients[15]. Five independent predictors of sustained response to interferon and ribavirin combination therapy were identified. These are genotype 2 or 3, low viral load (< 3.5 million copies/ml), no or only portal fibrosis, female sex and age below 40 years. Large studies with PIFN and ribavirin in HIV-HCV coinfected patients are ongoing. Based on limited data, the treatment of HCV infection in HIV positive patients should be the same as regimens recommended for HIV negative patients.

Additional predictive factors of response related to HIV include CD4 cell count > 500 cells/mm3, plasma HIV RNA levels below 10,000 copies/ml and no alcohol consumption.

Weight-based dosing is important to optimise success and minimise side effects. PIFN a-2b (Peg-lntron) 1.5pg/kg/week or PIFN a-2a (Pegasys) 180pg fixed dose per week plus ribavirin at least 10.6 mg/kg/day are the most effective dosages. When using

ribavirin of >10.6 mg/kg, up to 48% with genotype 1 and 88% with genotype 2 or 3 reached sustained response[14].

Serum HCV RNA by PCR should be performed at least after 24 weeks of treatment. If undetectable, treatment should be discontinued for genotype 2 or 3, and continued for another 24 weeks for genotype 1 or 4. If detectable, treatment should be discontinued in all cases because response is unlikely with further treatment. The benefit of continuing maintenance therapy has to be examined in clinical trials.

CD4 cell count and treatment considerations

Individuals with CD4 cell counts > 500 cells/mm3 regardless of the HIV RNA level are good candidates for treatment. Patients with CD4 cell counts 200 - 500 cells/mm^3 may also benefit from HCV treatment if plasma HIV RNA levels < 5,000 copies/ml. CD4 cell count below 200 cells/mm^3 should be considered a relative contraindication for HCV therapy. Antiretroviral therapy should be the priority in such patients, and HCV therapy reconsidered when CD4 cell count rises above 200 cells/mm^3.

Interferon in HIV patients

HIV patients often develop anaemia and neutropenia. They may suffer from depression, anxiety and irritability. Weight loss of > 10% and fatigue are common. These manifestations are also the common adverse effects of interferon. Treatment is contraindicated in patients with severe depression or suicidal ideation.

Interferon can induce a rapid decline in CD4 cell count in 10-15% of HIV infected patients, usually between 6th to 14th week of therapy. This transient lymphocytopenia is most likely a displacement of circulating cells to lymphoid organs. In a few patients, CD4 lymphocytopenia can be irreversible. Interferon may be contraindicated in subjects with CD4 cell count < 200/ mm^3.

Ribavirin in HIV patients

Adequate dose of ribavirin, particularly at the start of therapy, is linked to an increased likelihood of sustained virological response. The main concerns are dose-dependent haemolytic anaemia and

drug-drug interactions. For ribavirin-induced anaemia, erythropoietin may be an alternative strategy to dose reduction, and prospective studies are examining this approach.

Ribavirin, a guanosine nucleoside analogue, inhibits intracellular phosphorylation of zidovudine, stavudine, and zalcitabine in vitro. This may cause anti-HIV antagonism in vivo. Alternatively, ribavirin enhances phosphorylation of didanosine. This may increase anti-HIV effect, but also may increase mitochondrial toxicity. Clinical manifestations of mitochondrial toxicity include pancreatitis, steatohepatitis, myopathy, peripheral neuropathy and lactic acidosis. Stavudine and didanosine were the nucleosides most commonly associated with lactic acidosis. Clinicians should consider routine monitoring of serum lactate and amylase levels in HIV-HCV coinfected patients exposed concomitantly to ribavirin and nucleoside analogues.

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HIV Infection in Women: Contemporary Issues

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Since the first reported case of AIDS in 1982, the disease has quickly reached a global epidemic. Over the past 20 years, we witnessed tremendous changes in epidemiology and management of HIV infection. In particular, there is a whole body of evidence to suggest that HIV infection behaves differently in many aspects in women when compared with their male counterparts: epidemiology, disease progression, response to treatment, etc. This article serves to review the recent advances in the understanding of the characteristics of HIV infection in women.

Epidemiology

In the year of 2001, 213 persons were found be serologically positive in HIV antibody tests in Hong Kong, bringing the cumulative total number of reported HIV infections to 1755[1]. Of these213 patients, there were 158 male patients and 55 female patients, with the male to female ratio of 2.9 to 1.124 cases acquired the infection via heterosexual exposure and 43 cases via homosexual or bisexual contacts. Two other cases acquired the infections perinatally and 11 other cases involved injecting drug use. The route of transmission of the remaining 33 cases was undetermined. 60 AIDS cases were reported in the same year, bringing the total number of confirmed AIDS cases to 560. Among them, there were 48 male patients and 12 female patients, with the male to female ratio of 4 to 1. Around 80% of them acquired the infection through heterosexual contact. The prevalence of AIDS among different ethnic groups was remarkably different between two sexes: 47.8% of female patients were non-Chinese Asians, while Chinese females accounted for another 41.9% of cases. In contrast, 75.3% of infected males were Chinese.

Coupling these findings with the secular trend of the epidemiological data locally and from overseas, several observations could be made. First, there was a gradual narrowing of male to female ratio in recent years, while incidence of disease is increasing for both sexes, i.e., women constitutes a faster growing population in acquiring the HIV infection. Second, heterosexual contact becomes the most important route of

transmission, and this is more apparent in people of younger age. Similar findings were observed in other countries like the USA. Despite the ever-increasing incidence of HIV infection in women, their chance of accessing proper management, especially the highly active antiretroviral therapy (HAART), does not commensurate with that of men. This is exemplified by the work of Carten et al[2]. They reported a study for a cohort of HIV infected persons who were under their care in a county hospital from Chicago. Despite the similar baseline characteristics, the utilization rate of HAART was significantly lower among the female patients. This and numerous similar studies have demonstrated that women are less likely to receive HAART, even when they have the same care providers and similar HIV characteristics as compared with men. Such a finding has been demonstrated in the USA and other countries, and appears to be independent of financial considerations.

Viral kinetics

As more longitudinal follow-up data are now available, differences of viral kinetics between the two sexes become more apparent. The ACTG 175 study demonstrated clearly that HIV infected women had a lower baseline viral load compared with men[3]. Subsequently, ALIVE study, reported in 1998, further revealed that among those who developed AIDS, women had their viral load measuring 38 to 65% of men[4]. When comparing with men who had matched viral load at baseline, women had 1.6 times greater risk of progressing to AIDS. However, such a difference in viral load diminished among patients with advanced disease, as evidenced by the result from Viral Activation Transfusion Study[5]. This finding was further substantiated by the result of a meta-analysis performed from the pooled data of four large cohorts of patients in the USA (HERS, ASD, HOPS, VLSP), which comprised 2467 men & 1309 women[6]. The differences in longitudinal course of viral load were best exemplified by the report from Sterling et al, which showed that while the first post seroconversion median viral load was significantly lower in women when compared with men[7], it increased more rapidly over time in women and the two groups subsequently converged. The significance of these findings in using the viral load as a reference for the initiation of antiretroviral therapy and planning of follow-up in women remains to be elucidated.

Adverse reaction to treatment

Difference in sexes of the rate of adverse reaction to HAART is expected because of the difference in pharmacokinetic profile. In particular, weight appears to be a significant parameter leading to the difference. In fact, antiretrovirals are rarely dosed by weight, and when they are, the cut-offs are often problematic. This issue was examined by an analysis of ACTG 175 data[8]. Women appeared to reduce dosage and discontinue didanosine-containing regime more frequently than men. However, the gender difference became insignificant when adjustment of the difference in weight was made. On the other hand, risk of some adverse effects, e.g. lactic acidosis, increases with body weight. While a higher incidence of adverse reaction is generally observed in women, here we will focus on a number of special adverse events of practical interest.

(1) Lactic Acidosis

Lactic acidosis is a well-recognised adverse reaction secondary to the use of nucleoside analogues. It is due to the mitochondrial toxicity and the phenomenon is potentially fatal. Boxwell et al reviewed the 60 reports of lactic acidosis from FDA. Out of the twenty fatal cases, seventeen of them were women[9]. Among them, eleven were obese. In this series, hepatic steatosis and pancreatitis were identified in 71% and 29% of patients respectively. Results of other case series also came to the same conclusion of female sex and obesity being the risk factors for development of lactic acidosis and related complications.

(2) Lipodystrophy

Lipodystrophy was first reported among the HIV infected patients after the introduction of protease inhibitors. Although this class of drugs was once thought to be the causative agent, current evidence suggests that other components of HAART may also play a role. Galli et al reported the frequency and risk factors for the development of lipodystrophy in a cohort of 2258 patients, for which about 30% were women[10]. In this series, 33.2% of patients developed at least 1 morphological change associated with lipodystrophy during follow-up and female gender was found to be the strongest independent risk factor. Changes usually occurred within the first 12 to 18 months of the therapy and appeared stabilized afterwards.

(3) Nevirapine Induced Skin Rash

Nevirapine is a non-nucleoside reverse transcriptase inhibitor which has proven its value in the prevention of maternal to child transmission of HIV infection. Skin rash is one of the common adverse reactions to this agent. Mazhude et al reviewed the characteristics of 285 nevirapine-experienced patients concerning the risk factors and prevalence of this adverse event11. According to this report, 7.4% of patients developed rash following the use of this agent, and two-third of them required cessation of therapy. Median time of onset of rash was 17 days. Relative risk for rash in women was 11.7. Three cases of Stevens Johnson Syndrome were identified, and two of which were women.

Issues related to pregnancy

Prevention of maternal to child transmission (MTCT) of HIV is certainly one of the most important aspects in the management of HIV infected women. During the antenatal period, the most vulnerable period for MTCT was found to be the end of second trimester and the beginning of third trimester; whereas in the intrapartum period, blood or vaginal secretions may come in contact with foetal conjunctiva, gastrointestinal tract, or a break in skin, and this accounts for 65% of non-breastfeeding cases of HIV infection in newborns[12].

Breastfeeding is yet another important route of MTCT, which accounts for 75% of transmission occurring during the first 6 months of life in newborns. Factors that increase the risk of MTCT during breastfeeding period included younger maternal age, seroconversion during breastfeeding, mastitis and breast abscess[13].

One of the important breakthroughs in management of MTCT is the universal antenatal screening of HIV antibody. The rationale for this policy is obvious: in Hong Kong, about 50% the mothers of perinatally exposed children were diagnosed of HIV infection only after delivery. Since effective treatment is available to reduce MTCT, this argues strongly for testing all antenatal mothers for HIV. A formal Universal Antenatal HIV Screening Programme was jointly implemented by the Department of Health and the Hospital Authority in Hong Kong

on 1/9/2001. A total of 10238 tests were performed in the first three months of the programme and six expectant mothers were detected positive to the test.

Use of antiretroviral therapy (ART) is another important strategy in reducing the incidence of MTCT. The efficacy of this approach has been clearly documented. In the standard regimens, e.g. the PATCG 076 protocol, which comprises the use of zidovudine (ZDV) beginning as early as 14 weeks of pregnancy, continuing through labour by intravenous administration, and followed by treatment of the newborn for 6 weeks[14]. This results in a two-third reduction in risk of MTCT from the baseline of about 25%. Combination of ZDV and lamivudine (3TC), which was delivered in the same way as in the PATCG 076 protocol, was evaluated in PETRA study[15]. This proved to further enhance the efficacy in reducing MTCT.

The most important prerequisite for the success of the fore-mentioned regimens was to withhold breastfeeding. This is obviously not practical in resource-poor countries. HIVNET-012 study evaluated the efficacy of nevirapine in an abbreviating regimen for the prevention of MTCT[16]. In this study, 200 mg of the drug was offered to the mother at the onset of labour; then another dose of drug at 2 mg/kg was given to newborn at 48 to72 hours after delivery. 95% of the newborns received breastfeeding during follow-up. Transmission rate was 13.1% at 14 to 16 weeks of follow-up. Rapid emergence of drug resistance in mother is a concern, as this will limit the options of drugs available for use in future.

As a rule of thumb, alternative antiretroviral prophylaxis should be administered in special circumstances where the standard regimen is considered not practicable. Apart from the use of antenatal screening and ARTs, delivery by Caesarean section would be another way to reduce MTCT. This was first suggested by the European Collaborative Study Group[17] in 1992, although the result of their study did not reach statistical significance. The International Perinatal HIV Group subsequently reported in 1999 a meta-analysis of North American & European studies[18], which undoubtedly showed the superiority of Caesarean section in reducing the incidence of MTCT compared with vaginal delivery. However, controversy exists regarding in the role of Caesarean section in patients with

undetectable viral load. Furthermore, the risk of complications associated with operation should not be overlooked[19]. Prolonged rupture of membranes (especially if more than 4 hours), invasive foetal monitoring, and instrumental vaginal delivery should be avoided to reduce the risk of MTCT.

In general, a woman who is diagnosed HIV positive in the course of pregnancy should be counselled on the long term care plan, informed of the efficacy of prophylaxis against MTCT, and evaluated for the appropriate antiretroviral treatment. In cases where HAART is not indicated for maternal HIV infection, standard ZDV regimen is recommended for prophylaxis against MTCT. If HAART is indicated, ZDV should be incorporated in the regimen unless contraindicated. Apart from the usual parameters in non-pregnant patients, the choice of other components of a HAART regimen should also be based on potential toxicity to mother and foetus, altered pharmacokinetics in pregnancy, and compliance. If there is intolerance to ZDV, then the nevirapine regimen may be substituted. Efavirenz is contraindicated because of its teratogenicity in animal models. Toxicity including teratogenicity to the foetus would be greatest in the first trimester. It is therefore acceptable that treatment be postponed until 10 to 12 weeks of gestation[20].

In mothers who become pregnant while receiving antiretroviral therapy, evaluation of the treatment should be made regarding antiretroviral potency, potential toxicity to the mother and foetus, and prophylactic efficacy against MTCT. The rationales of alteration or continuation of therapy should be fully explained to the mother to facilitate the decision. ZDV should be added or substituted even if the mother has had prior experience with the drug. If there is intolerance to ZDV, the nevirapine regimen may be used for prophylaxis against MTCT. Optimal control of maternal HIV disease is essential in reducing MTCT as both the magnitude of viral load and CD4 count are related to the risk of MTCT. A viral load result near term is preferable to help determine the mode of delivery[20].

The option of termination of pregnancy should be thoroughly discussed with patients, taking into account the patient's own wish, her health status, risk of MTCT, currently available management strategies and potential side effects of the antiretroviral therapy to both the mother and the fetus.

For the newborns, one should continue the prophylactic regimen as described and look for possible congenital defects or other consequences as a result of exposure to antiretroviral therapy[20]. Mothers shall be advised against breastfeeding whenever possible.

Conclusion

The incidence of HIV infections is increasing among women, and heterosexual contact is becoming the commonest mode of transmission. Lower level of HIV-RNA was observed in women during the early stages of infection compared with men. This difference remains unexplained. Clinicians should be aware of this in considering the timing for initiation of HAART and when planning follow-up for those women who would like to defer treatment because of low viral load. There is a growing interest concerning the impact of gender and weight on the pharmacokinetics of ARTs and adverse reactions to therapy. Antenatal screening for HIV is now offered to all pregnant ladies in Hong Kong. For those with HIV infection, management should be directed towards optimisation of maternal health, initiation of HAART if indicated & reduction of MTCT.

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Respiratory Tract Infections in Adults

(A report from Seminar in Infectious Disease - Focus on Community - acquired ENT and Respiratory Infections, 20 July 2002, Princess Margaret Hospital)

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Viral Respiratory Infections (VRI)

VRI is a big socio-economic burden and is an important cause of admission into HA and private hospitals. Rhinoviruses cause a majority of these infections. Many of the cases of upper respiratory infections are due to viral causes but antibiotics have been frequently prescribed unnecessarily. Studies in the US showed that the strongest predictor of receipt of antibiotics for acute respiratory infection is the doctor's perception of patient expectation. Indiscriminate use of antibiotics not only increases healthcare cost but also increases the risk of antibiotics resistance. After careful explanation to the patient, the doctor should decline the use of antibiotic when it is not indicated.

Community-acquired Pneumonia (CAP)

Overseas studies showed that the common causes of CAP were Streptococcus pneumoniae (10-40%), viruses (5-20%), Haemophilus influenzae (5-15%), Legionella (3-15%), Mycoplasma pneumoniae (2-15%), Chlamydia pneumoniae (5-10%), unknown (30-50%). Previous studies showed that Streptococcus pneumoniae and Mycobacterium tuberculosis were common causes of CAP in HK. However, more recent study showed that H. influenzae (13.7%) was the commonest cause of CAP, followed by Mycoplasma pneumoniae (11.8%) and Streptococcus pneumoniae (5.9%). The cause of the change in pattern of etiologic agent was not known.

Over the past 10 years, there was also an increasing trend of penicillin resistant S. pneumoniae (PRSP) in HK. Latest figures

showed that up to 80% of the S. pneumoniae isolates were PRSP. The clinical significance of the high incidence of PRSP was not certain. A retrospective study of 248 patients with positive isolates of PRSP showed that the organism was associated with the extremes of age and recent antibiotic use. Most of these patients only had minor chest x-ray abnormalities. There was no difference in mortality compared to those with penicillin sensitive strains. The study concluded that in most cases the PRSP were likely to be colonizers of the respiratory tract and only in a few patients was it clinically significant.

Surveillance of antibiotic resistance in HA hospitals showed that there was an increasing trend of antibiotic resistance due to extended spectrum beta-lactamase producing organisms, PRSP, methicillin resistant Staphylococcus aureus and Pseudomonas aeruginosa.

The IMPACT guidelines for antibiotic use in CAP were:

	Preferred regimen
1. CAP, not hospitalised	PO Augmentin/ Unasyn + Macrolide
2. CAP, hospitalised in general ward	IV Augmentin/ Unasyn + macrolide
3. CAP, hospitalised in ICU or serious pneumonia	IV Tazocin/ Cefotaxime/ Ceftriazone + Macrolide

Supraglottitis in Prince of Wales Hospital
(A report from Seminar in Infectious Disease - Focus on Community - acquired ENT and Respiratory Infections, 20 July 2002, Princess Margaret Hospital)

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"Epiglottitis" is perhaps more popular amongst clinicians, nevertheless, "supraglottitis" is a more accurate description of the condition. In the Prince of Wales Hospital, the ENT Unit has managed 95 cases of "supraglottitis" between August 1986 and June 2002. Adult cases dominated the picture with only 4 paediatric cases. There was a marked variation in annual and seasonal incidence. There were slightly more male than female cases. The age incidence plateaued between the 4^{th} and 6^{th} decades. All the paediatric patients presented with stridor while the clinical picture of the adult patients was much more variable including sore throat (100%), fever (47%), dysphagia (47%) dyspnoea (39%).

In contrast to the experience in the West where Haemophilus influenzae type b is the most important cause, there were no predominant micro-organisms identified from the patients in our series. *Streptococcus milleri* was the commonest pathogen identified. Adequate control of the airway remained the most important aspect of management. All paediatric patients required endotracheal intubation and were managed in the Intensive Care Unit (ICU). Adult patients with respiratory distress, however mild, were observed in ICU. Those with more severe respiratory obstruction had their airways secured by either endotracheal intubation or tracheostomy. Overall, 70% patients required ICU admission. The average ICU and hospital stay were 3.2 and 12.3 days respectively. Septicaemia and deep neck space abscesses were more likely to occur in the older patients. There were two airway related and one non-airway related deaths in the present series.

In conclusion, supraglottitis is still associated with significant mortality and morbidity. A standardised protocol for management is necessary for a satisfactory outcome.