

**Surveillance of exposure to blood-borne
viruses (HIV, HBV, HCV) and its management
1999 - 2004**

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COMMENTARY

Background

1. Human immunodeficiency virus (HIV) is transmitted by three modes: sexual contact, blood-borne contact and mother-to-child. While blood-borne transmission essentially refers to sharing of needles/syringes among injecting drug users, HIV infection resulting from exposure in health care setting did rarely occur. Small but genuine, the risk of contracting HIV after percutaneous and mucosal exposures to HIV-contaminated blood is 0.3% [1] and 0.09% respectively. [2] Besides, blood-borne hepatitis, notably hepatitis B (HBV) and hepatitis C (HCV), are of concern after occupational exposure.

2. The first case of documented HIV seroconversion after occupational exposure was reported in 1984 in an UK health care worker (HCW). [3] Worldwide, with data censored up to the end of 2002, there were a total of 106 documented and 238 possible occupationally acquired HIV cases. [4] The occurrence of hepatitis B and C related to health care would have been more common, contributed by their higher transmission risk. The risk of HBV infection after a percutaneous injury in health care setting is 6-30% [5] whereas that for HCV is 0-10% after percutaneous or mucosal contact. [6]

3. Risk assessment, counselling and health advice are of utmost importance in post exposure management. This applies for both occupational exposure as well as non-occupational exposure that happen in community settings. Baseline and follow-up blood investigations are necessary to document the incident and its outcome, as well as inform and monitor specific interventions, if any. At present, hepatitis B immunoglobulin (HBIG) and vaccine are available to reduce the risk of HBV transmission after exposure. Antiretroviral drugs can be employed as post exposure prophylaxis (PEP) for HIV but no effective preventive intervention exists for HCV.

Surveillance of occupational and non-occupational exposure

4. In view of the significance and implication of exposure to blood-borne pathogens, many countries have set up various surveillance mechanisms for exposure

to blood-borne viruses (BBV), often focusing on HIV, HBV and HCV. For example, UK started a passive surveillance system for significant occupational exposure to HIV in the mid-1980s, which was enhanced in 1997 together with expanding the coverage to the reporting of HBV and HCV exposures, through the assistance of occupational health departments. [7] The US Centers for Disease Control and Prevention (CDC) also takes stock of the number of HCWs who definitively or probably contracted HIV after a history of occupational exposure.

5. In Hong Kong, the Accident & Emergency (A&E) Department is by far the commonest service provider which clients with potential exposure to BBVs will first attend. However, as post exposure management requires longer term follow up, the clients are often referred to designated institutions after first aid and immediate care at A&E Departments. One such clinic is the Therapeutic Prevention Clinic (TPC) of Integrated Treatment Centre (ITC), Centre for Health Protection of the Department of Health. Since its operation in mid 1999, TPC has been monitoring the characteristics and outcome of clients referred to its care. Albeit far from ideal, the data collected may shed some light on the local pattern of exposure to BBVs.

6. On a referral basis, the TPC aims to provide comprehensive post exposure care to people with documented percutaneous, mucosal or breached skin exposure to blood/body fluids, which could have therapeutic and/or diagnostic implications for HIV or viral hepatitis B and C. Doctors and nurses are the key health professionals staffing the clinic. After the initial consultation and work up, clients are offered clinic revisit at 6 months post exposure to have follow up blood investigations if PEP or HBIg is not indicated. A defaulted case is closed at 8 months after injury per clinic protocol.

7. Integrating into care, three aspects of information are gathered with standard questionnaires by the attending nurse and doctor for each TPC client: (a) first consult assessment of the client and exposure, (b) HIV, HBV and HCV serology, and (c) HIV post exposure prophylaxis. Assessment at first consultation includes demography and occupation of the exposed person, type of the injury/exposure, source person, risk of the exposure, and PEP. All exposures are grouped under two categories: (i) HCW with occupational exposure - category A, and (ii) all other exposure cases - category

B. To determine BBVs seroconversion after the exposure, baseline and subsequent serology are checked as far as possible. The prescription and outcome of HIV PEP is specifically examined, in line with international practice. Collected data are cleaned at quarterly intervals. This Report presents the observations and analysis of data obtained from mid-1999 to 2004.

Types and pattern of exposure

8. Since mid-1999, TPC saw some 300-500 clients referred for post exposure management each year. Of which, about one-third were health care workers who sustained exposure in the health care settings. As shown in Table 2, the biggest number of HCW referred for care was nurses, which may be related to their higher frequency of having occupational exposure and/or a larger size of the profession. Dental professionals came second while ward/clinic ancillary staff were the next commonest category of HCW with occupational exposure. On the other hand, cleansing staff were the most frequent distinct worker amongst other exposure cases. The proportions contributed by disciplinary staff and institution staff were about the same. No obvious trend change was observed over the last few years regarding the profession of TPC clients.

9. The exposed persons spanned a wide range of age. For HCW with occupational exposure, they were mostly (over 60%) in the age group of 25-44 years (Table 3). Clients belonging to all other exposures were older, with about half between 35 and 54 years of age. This is expected as the latter clients were heterogeneous, including those from the general public while the former was in the health care workforce. More females were implicated in attendance for post exposure management, in particular for category A clients.

10. Over half of both categories of clients sustained exposure from 8am to 4pm of a day (Table 4). Not surprisingly, more clients in category B had injury in other time periods, especially for 12 midnight to 8 am, when compared to category A clients. Again, both groups first attended for medical consultation between 8am to 4pm, with slightly more category B clients seeking consultation for the exposure in other time periods. Over 70% of the HCW with occupational exposure worked in public hospitals/clinics/laboratories (Table 5). For all other exposure cases, most sustained

exposure in their workplace or public areas. Percutaneous injury is the commonest type of exposure, accounting for nearly 90% in category A and over 60% in category B clients (Table 6). Human bite was also a common mode of exposure in category B clients, accounting for about 30% of all cases. Mucosal exposure was exceedingly uncommon in our clients, occurring in less than 2% of HCW with occupational exposure. Over 80% of all exposures were assessed to be superficial, similar for both categories of clients. Some 80% of category A clients had their exposure source identified, which was higher than the 46% for category B clients (Table 8). At the time of first consultation at TPC, about 7% and 5% of the identified sources were known to be HBsAg positive for category A and category B clients respectively. The known anti-HCV and anti-HIV positivity rates were 1.5% and 3.0% among identified sources of category A clients, which were also higher than the corresponding figures in category B clients. Nevertheless, it was unlikely that they represented the underlying prevalence of blood-borne pathogens in the populations, due to the biased sampling and small number of cases.

11. Amongst HCW who sustained occupational exposure, over 70% occurred under four situations: (a) blood-taking/intravenous catheter insertion, (b) injection including recap, (c) other bedside/treatment room procedures (which was the commonest), and (d) cleansing/tidying up after procedures (Table 9). About 45% of category A exposures were with blood or blood-contaminated fluids. Nearly half were implicated with hollow-bore needles while lancet and dental instrument were the other common specific technical device relating to exposure. The frequency of respective activity/procedure contributing to the exposures in medical/dental health professionals were similar to the overall scenario (Table 10). For the nursing professionals, unexpectedly cleansing/tidying up after procedures was not an important activity/procedure leading to exposure (Table 11). The specific settings of injury/exposure in the health professionals somewhat reflect their work nature.

12. Taking reference of the case-control study which identified risk factors associated with higher likelihood of HIV transmission after percutaneous injury, [8] we set five factors under higher risk exposure: deep percutaneous injury, involving procedures with device placed in a blood vessel, involving a hollow-bore needle, device which is visibly contaminated with blood and source person with AIDS. On a

second level which is considered lower risk but still risky exposure, there are also five factors: moderate percutaneous injury, mucosal contact, contact with deep body fluids other than blood, source person is HIV infected but not or not sure to the stage of AIDS and other reasons contributing to increased risk. Under such assessment framework, about 55% of all HCW with occupational exposures was classified to be higher risk over the years (Table 12). Some 13% belonged to lower risk. A vast majority of the clients with either higher or lower risk exposure had one risk factor out of the five (Table 13). When examined against the procedures, only blood-taking/intravenous catheter insertion and sharps disposal had ever resulted in client exposures with 3 factors. The mean number of risk factors was the highest at 1.39 (95% confidence interval, 1.25-1.53) for the former activity/procedure (Table 14). Just over half of the HCWs sustaining occupational exposure had worn glove as a precaution (Table 15). Judging from the most common activities/procedures that ensue in occupational exposure which normally require precaution with glove, there could be rooms for improvement in infection control measures.

Care, HIV post-exposure prophylaxis and outcome

13. Cumulatively, 55% of category A clients attended medical consult within 2 hours of exposure; another 30% between 2 and 12 hours (Table 16). Seeking consultation for the exposure was comparatively less prompt in category B clients. The median time lag was 1.55 hours and 2.3 hours for category A and category B clients respectively. Baseline blood check was done to discern the susceptibility of clients to BBVs, necessity of intervention and document status prior to injury. As expected, hepatitis B was the common infection present before exposure. Some 7 % and 9% of category A and B clients respectively had positive HBsAg at baseline (Table 17). In addition, another 50-66% of the two categories had anti-HBs. It was not surprising that HBV markers were more prevalent in HCWs, likely as a result of hepatitis B vaccination and increased exposure risk from work. Hepatitis C antibody was only found in 3 (1.5%) of category B clients but not HCWs. None of the clients were HIV positive at baseline (Table 17). Follow up testing of the cases did not show seroconversion to become HBsAg positive, anti-HCV or anti-HIV positive.

14. Over the 5-and-a-half years, a total of 46 subjects had been put on HIV PEP, corresponding to 3.3% and 1.5% of category A and B clients respectively (Table 18).

The proportion prescribed HIV PEP fluctuated more each year for the group of HCWs who sustained occupational exposure. Administration of PEP to category A clients was in general prompt, with an overall median time of 3 hours after exposure in the 24 subjects (Table 19). Over 40% and all were given PEP within 2 hours and 24 hours after exposure respectively. Time lag of PEP initiation in category B exposures was greater, with a median of 16.7 hours in 22 clients. Two clients were prescribed PEP greater than 24 hours. Comparatively, more category A clients were continued on HIV PEP than category B clients (Table 20). The phenomenon could be contributed by several factors, including further assessment and counselling of exposure risk and willingness to continue treatment in individual subject.

15. A majority of the clients in both categories who were continued on PEP had known outcome as they attended TPC for follow up. Of these subjects, most were put on 3 drugs (two nucleoside reverse transcriptase inhibitor, NRTI, backbone plus one protease inhibitor, PI) instead of 2 NRTIs alone – 87.5% in category A and 66.7% in category B (Table 22). Occurrence of toxicity was common, irrespective of the number of drugs of the PEP regimen (Table 23). Experience of adverse effects of PEP appeared to be commoner in category A clients (100%) as compared to category B clients (66.7%). Overall, moderate to severe drug toxicity was encountered in over half of category A clients and one-third of category B clients. Due to a variety of reasons, completion rate of PEP was poor (Table 24). The rate was even lower for HCWs sustaining occupational exposure, which was related to their more frequent experience of drug toxicities. Seven HCWs stopped PEP because of adverse effects and 6 were due to source confirmed HIV negative (Table 26). On the contrary, only one category B client did not complete PEP due to drug toxicity. With more subjects completing PEP in category B, their median duration of intake of drugs was longer. It has to be cautioned, however, that the small number of subjects precluded definitive conclusion on HIV PEP regarding differences between different clients and drug side effects.

16. Four category A and 2 category B clients who were continued on PEP with known outcome had an exposure source confirmed HIV infected over the years (Table 27). All of the six subjects were prescribed HIV PEP, with a median of 2.3 hours in category A and 17.2 hours in category B respectively. All were given PEP within 24

hours of exposure, and 2 of the HCWs were intervened within 2 hours. Three of category A clients and one of category B clients completed their PEP. The health care worker stopped PEP because of side effects.

Discussion

17. Transmission of BBVs to HCWs is an occupational hazard which can lead to serious consequences. Fortunately, the risk is extremely low with appropriate precautions and work practices. The advent of pre-exposure vaccination and post exposure prophylaxis further decrease the risk for some of the infections. Overseas, guidelines/recommendations were developed to guide the management, including use of PEP, after occupational exposure to HIV, HBV and HCV in health care settings. [9-11] Recently the European authorities [12] as well as US CDC [13] had also issued new recommendations on non-occupational exposure for HIV. Locally, the importance of prevention and transmission of BBVs after exposure is all along recognized. Guidelines and recommendations have been specifically developed and made in place, to assist patient management. [14, 15]

18. It is prudent to keep track of the extent and impact of exposure to BBVs in occupational and non-occupational settings. Over the last 6 years, as a surveillance effort, we have tried to gauge the incident of exposure, baseline and follow up BBV serologic markers and details of PEP for the clients who attended TPC. The objectives are to follow the trend of exposure, discern characteristics of client and exposure, and hopefully contribute to preventive measures to reduce and better manage such incidents in future. While the exposure in health care setting was more clear-cut, exposure in the community setting is often less easily defined and characterized. The need and efficacy of intervention such as HIV PEP are also less well proven and documented for non-occupational exposure. Nevertheless, the basic principles of risk assessment of the exposure, counseling, psychological support and investigations apply for all kinds of exposure. Just like managing other clinic conditions, it is necessary to have case-by-case assessment and tailored management for post exposure clients.

19. We found that a variety of health care workers were implicated in occupational exposure to potential BBVs, albeit a greater number of some professions

were involved. Percutaneous injury was the most frequent exposure, which conceivably is associated with the highest risk amongst different sorts of exposure. Sharps handling was the most important element linked to exposure, which can occur during patient procedures or after care. In general, clients sustaining exposure attended consultation promptly. Being the most important first time carer of clients with exposure to BBVs, the A&E Departments are with the strength of having a standardised guidelines in providing its management. [15] Nevertheless, frontline staff of the departments of different hospitals may have different practice when delivering their care. Prescription of HIV PEP was found to be uncommon in our cohort. Stock of starter pack antiretrovirals at A&E Department improves the promptness of treatment of significant exposure when indicated. Among those initiated on HIV PEP, a significant proportion of them did not complete the 28-day course of treatment. Similar to overseas reports, drug intolerance and toxicity was a key factor leading to discontinuation of PEP.

20. There were several limitations to our findings. Our data comes from a single centre only, and is based on secondary referral. There are many factors influencing whether clients attend or not attend our clinic. Hence, it is sure that the data did not cover or represent all clients who sustained exposure in the same period of time. As a result, the exact dimension of the issue cannot be ascertained basing on our data. Moreover, we examined the characteristics and pattern of exposure from the absolute cases seen. We have not factored in the denominator, i.e. number of HCWs of each health care workforce in our analysis. The extent of the problem within different health professions would be better analysed in such way. Also, even though data has been collected with standardised template, the presence of inter-observer and intra-observer bias is likely, which may affect data consistency.

21. We examined all clients and not only those exposed to positive BBVs sources in most of the analysis. The overall risk could thus be an under estimate for some of the clients. The fact that the status of BBVs in many sources being unknown adds to the difficulty. Thus, in contrast to the UK HPA [7] whose surveillance only includes HCWs exposed to HBV surface antigen, HCV or HIV positive source, or where the source status is unknown but HIV PEP is started, our monitoring covers all clients who attended care at TPC. This is, however, similar to the surveillance effort of Italy

which includes all occupational exposure and not restrict to known sources with BBVs. [16] Also, the inclusion of insignificant exposure rather than only significant exposure would have made the overall risk estimate lower. The relative small number of clients and especially those with known positive sources made it impossible to draw conclusions for many aspects. Nevertheless, our data has the strength of completeness as default of the clients was infrequent.

22. There have been no cases of documented seroconversion after occupational exposure to HIV, HBV or HCV from this cohort. Notwithstanding, given the significance and potential implications, regular monitoring of the situation, exposure and outcome is needed. To have more complete and representative sampling, information collection from the primary caring institution, i.e. A&E Departments, would be useful. Data collected at secondary care level is limited by that not all exposure cases are referred to designated service and some of the referred clients may not attend. It has to be cautioned, however, the importance of maintaining confidentiality irrespective of the sites and points of data collection. Due to the sensitive nature of these communicable BBVs, protection of personal particulars has to be ensured especially for services with small number of respective HCWs. Collection of person identifying information would be unnecessary in surveillance efforts with a view for general improvement relating to the prevention of transmission of blood-borne pathogens in health care settings. Adherence to infection control measures is imperative to the prevention of exposure in health care settings which runs an excess risk of acquiring and transmitting blood-borne pathogens. Standard precaution should be practised; training and education to staff has to be implemented. Prompt and appropriate management in case of exposure can ease the fear of the injured person and reduce the chance of infections.

**Tabulated results on cases with exposures attending
Therapeutic Prevention Clinic**

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Table 1. Number of incidents with possible viral exposure and referred to attend Therapeutic Prevention Clinic, ITC, CHP, DH (mid 1999-2004)

	A. Health care workers with occupational exposure	B. All other exposure cases	Total
1999 (Jul-Dec)	30	99	129
2000	122	266	388
2001	167	380	547
2002	154	295	449
2003	157	231	388
2004	97	222	319
Total	727	1493	2220

Table 2. Profession of the exposed persons

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure							
Nursing profession	21 (70.0%)	51 (41.8%)	72 (43.1%)	64 (41.6%)	65 (41.4%)	26 (26.8%)	299 (41.1%)
Dental profession	6 (20.0%)	26 (21.3%)	41 (24.6%)	29 (18.8%)	29 (18.5%)	39 (40.2%)	170 (23.4%)
Medical profession	1 (3.3%)	5 (4.1%)	12 (7.2%)	9 (5.8%)	11 (7.0%)	4 (4.1%)	42 (5.8%)
Ward/clinic ancillary staff	0 (0.0%)	25 (20.5%)	23 (13.8%)	29 (18.8%)	27 (17.2%)	13 (13.4%)	117 (16.1%)
Others	2 (6.7%)	15 (12.3%)	19 (11.4%)	23 (14.9%)	25 (15.9%)	15 (15.5%)	99 (13.6%)
B. All other exposure cases							
Cleansing staff	28 (28.3%)	67 (25.2%)	77 (20.3%)	76 (25.8%)	63 (27.3%)	45 (20.3%)	356 (23.8%)

Disciplinary staff	18 (18.2%)	29 (10.9%)	36 (9.5%)	29 (9.8%)	38 (16.5%)	25 (11.3%)	175 (11.7%)
Institution staff	7 (7.1%)	18 (6.8%)	26 (6.8%)	35 (11.9%)	33 (14.3%)	49 (22.1%)	168 (11.3%)
Others	46 (46.5%)	152 (57.1%)	241 (63.4%)	155 (52.5%)	97 (42.0%)	103 (46.4%)	794 (53.2%)

Table 3. Gender and age of the exposed persons

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure							
Male	7 (23.3%)	25 (20.5%)	36 (21.6%)	36 (23.4%)	45 (28.7%)	34 (35.1%)	183 (25.2%)
Female	23 (76.7%)	97 (79.5%)	131 (78.4%)	118 (76.6%)	112 (71.3%)	63 (64.9%)	544 (74.8%)
Age (Years)							
<15	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
15-24	3 (10.0%)	22 (18.0%)	33 (19.8%)	36 (23.4%)	25 (15.9%)	20 (20.6%)	139 (19.1%)
25-34	8 (26.7%)	39 (32.0%)	56 (33.5%)	45 (29.2%)	46 (29.3%)	25 (25.8%)	219 (30.1%)
35-44	16 (53.3%)	38 (31.1%)	55 (32.9%)	39 (25.3%)	56 (35.7%)	40 (41.2%)	244 (33.6%)
45-54	3 (10.0%)	20 (16.4%)	22 (13.2%)	28 (18.2%)	26 (16.6%)	10 (10.3%)	109 (15.0%)
>55	0 (0.0%)	3 (2.5%)	1 (0.6%)	6 (3.9%)	4 (2.5%)	2 (2.1%)	16 (2.2%)
B. All other exposure cases							
Male	54 (54.5%)	135 (50.8%)	177 (46.6%)	123 (41.7%)	100 (43.3%)	101 (45.5%)	690 (46.2%)
Female	45 (45.5%)	131 (49.2%)	203 (53.4%)	172 (58.3%)	131 (56.7%)	121 (54.5%)	803 (53.8%)
Age (Years)							
<15	5 (5.1%)	20 (7.5%)	19 (5.0%)	14 (4.7%)	0 (0.0%)	3 (1.4%)	61 (4.1%)
15-24	10 (10.1%)	54 (20.3%)	86 (22.6%)	38 (12.9%)	34 (14.7%)	33 (14.9%)	255 (17.1%)
25-34	25 (25.3%)	40 (15.0%)	77 (20.3%)	60 (20.3%)	54 (23.4%)	57 (25.7%)	313 (21.0%)
35-44	32 (32.3%)	68 (25.6%)	81 (21.3%)	74 (25.1%)	52 (22.5%)	60 (27.0%)	367 (24.6%)
45-54	17 (17.2%)	56 (21.1%)	72 (18.9%)	76 (25.8%)	74 (32.0%)	53 (23.9%)	348 (23.3%)
>55	10 (10.1%)	28 (10.5%)	45 (11.8%)	33 (11.2%)	17 (7.4%)	16 (7.2%)	149 (10.0%)

Table 4. Timing of exposure and medical consultation

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
Time of injury							
A. Health care workers with occupational exposure							
12MN - 8am	4 (13.3%)	11 (9.0%)	20 (12.0%)	11 (7.1%)	14 (8.9%)	2 (2.1%)	62 (8.5%)
8am - 4pm	21 (70.0%)	76 (62.3%)	96 (57.5%)	93 (60.4%)	98 (62.4%)	67 (69.1%)	451 (62.0%)
4pm - 12MN	5 (16.7%)	35 (28.7%)	51 (30.5%)	50 (32.5%)	45 (28.7%)	28 (28.9%)	214 (29.4%)
B. All other exposure cases							
12MN - 8am	12 (12.1%)	49 (18.4%)	64 (16.8%)	42 (14.2%)	37 (16.0%)	29 (13.1%)	233 (15.6%)
8am - 4pm	54 (54.5%)	140 (52.6%)	185 (48.7%)	149 (50.5%)	119 (51.5%)	111 (50.0%)	758 (50.8%)
4pm - 12MN	33 (33.3%)	77 (28.9%)	131 (34.5%)	104 (35.3%)	75 (32.5%)	82 (36.9%)	502 (33.6%)
Time of first medical consultation							
A. Health care workers with occupational exposure							
12MN - 8am	1 (3.3%)	8 (6.6%)	7 (4.2%)	11 (7.1%)	12 (7.6%)	2 (2.1%)	41 (5.6%)
8am - 4pm	25 (83.3%)	78 (63.9%)	95 (56.9%)	87 (56.5%)	86 (54.8%)	55 (56.7%)	426 (58.6%)
4pm - 12MN	4 (13.3%)	36 (29.5%)	65 (38.9%)	56 (36.4%)	59 (37.6%)	40 (41.2%)	260 (35.8%)
B. All other exposure cases							
12MN - 8am	10 (10.1%)	22 (8.3%)	45 (11.8%)	31 (10.5%)	27 (11.7%)	18 (8.1%)	153 (10.2%)
8am - 4pm	46 (46.5%)	138 (51.9%)	194 (51.1%)	133 (45.1%)	109 (47.2%)	96 (43.2%)	716 (48.0%)
4pm - 12MN	43 (43.4%)	106 (39.8%)	141 (37.1%)	131 (44.4%)	95 (41.1%)	108 (48.6%)	624 (41.8%)

Table 5. Setting/location of the exposure

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure							
Public hospital	6 (20.0%)	49 (40.2%)	69 (41.3%)	73 (47.4%)	83 (52.9%)	25 (25.8%)	305 (42.0%)
Private hospital	1 (3.3%)	0 (0.0%)	2 (1.2%)	0 (0.0%)	2 (1.3%)	2 (2.1%)	7 (1.0%)
Public clinic/laboratory	20 (66.7%)	45 (36.9%)	58 (34.7%)	36 (23.4%)	39 (24.8%)	35 (36.1%)	233 (32.0%)
Private clinic/laboratory	1 (3.3%)	5 (4.1%)	13 (7.8%)	11 (7.1%)	5 (3.2%)	15 (15.5%)	50 (6.9%)
Others	2 (6.7%)	23 (18.9%)	25 (15.0%)	34 (22.1%)	28 (17.8%)	20 (20.6%)	132 (18.2%)
B. All other exposure cases							
Workplace of the exposure	63 (63.6%)	130 (48.9%)	156 (41.1%)	160 (54.2%)	138 (59.7%)	136 (61.3%)	783 (52.4%)
Home	6 (6.1%)	22 (8.3%)	26 (6.8%)	30 (10.2%)	22 (9.5%)	19 (8.6%)	125 (8.4%)
Public area	28 (28.3%)	107 (40.2%)	185 (48.7%)	90 (30.5%)	54 (23.4%)	46 (20.7%)	510 (34.2%)
Other	2 (2.0%)	7 (2.6%)	13 (3.4%)	15 (5.1%)	17 (7.4%)	21 (9.5%)	75 (5.0%)

Table 6. Nature of the exposure

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure							
Percutaneous	27 (90.0%)	96 (78.7%)	144 (86.2%)	142 (92.2%)	142 (90.4%)	92 (94.8%)	643 (88.4%)
Mucosal excluding sexual	0 (0.0%)	3 (2.5%)	2 (1.2%)	1 (0.6%)	5 (3.2%)	2 (2.1%)	13 (1.8%)
Non-intact skin	0 (0.0%)	7 (5.7%)	6 (3.6%)	2 (1.3%)	2 (1.3%)	0 (0.0%)	17 (2.3%)
Human bite	2 (6.7%)	9 (7.4%)	7 (4.2%)	9 (5.8%)	7 (4.5%)	2 (2.1%)	36 (5.0%)
Other	1 (3.3%)	7 (5.7%)	8 (4.8%)	0 (0.0%)	1 (0.6%)	1 (1.0%)	18 (2.5%)
B. All other exposure cases							
Percutaneous	56 (56.6%)	165 (62.0%)	240 (63.2%)	179 (60.7%)	144 (62.3%)	131 (59.0%)	915 (61.3%)
Mucosal excluding sexual	0 (0.0%)	2 (0.8%)	1 (0.3%)	3 (1.0%)	5 (2.2%)	7 (3.2%)	18 (1.2%)
Non-intact skin	2 (2.0%)	8 (3.0%)	13 (3.4%)	4 (1.4%)	3 (1.3%)	4 (1.8%)	34 (2.3%)
Human bite	37 (37.4%)	84 (31.6%)	118 (31.1%)	102 (34.6%)	71 (30.7%)	72 (32.4%)	484 (32.4%)
Other	4 (4.0%)	7 (2.6%)	8 (2.1%)	7 (2.4%)	8 (3.5%)	8 (3.6%)	42 (2.8%)

Table 7. Severity of the exposure

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure							
Superficial	29 (96.7%)	85 (69.7%)	119 (71.3%)	131 (85.1%)	142 (90.4%)	89 (91.8%)	595 (81.8%)
Moderate	1 (3.3%)	32 (26.2%)	43 (25.7%)	20 (13.0%)	8 (5.1%)	5 (5.2%)	109 (15.0%)
Deep	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	(0.0%)	0 (0.0%)
B. All other exposure cases							
Superficial	87 (87.9%)	224 (84.2%)	313 (82.4%)	238 (80.7%)	189 (81.8%)	188 (84.7%)	1239 (83.0%)
Moderate	11 (11.1%)	40 (15.0%)	50 (13.2%)	46 (15.6%)	29 (12.6%)	26 (11.7%)	202 (13.5%)
Deep	1 (1.0%)	0 (0.0%)	0 (0.0%)	2 (0.7%)	0 (0.0%)	1 (0.5%)	4 (0.3%)

Table 8. Reported status of blood-borne infections in identified sources of the incidents of exposure at initial consult

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure							
Source identified	n= 24	n= 95	n= 142	n= 123	n= 135	n= 78	n= 597
Known HBsAg positive	0 (0.0%)	11 (11.6%)	11 (7.7%)	5 (4.1%)	8 (5.9%)	6 (7.7%)	41 (6.9%)
Known HCV positive	0 (0.0%)	2 (2.1%)	4 (2.8%)	2 (1.6%)	1 (0.7%)	0 (0.0%)	9 (1.5%)
Known HIV positive	0 (0.0%)	3 (3.2%)	9 (6.3%)	0 (0.0%)	5 (3.7%)	1 (1.3%)	18 (3.0%)
B. All other exposure cases							
Source identified	n= 38	n= 96	n= 149	n= 141	n= 131	n= 133	n= 688
Known HBsAg positive	1 (2.6%)	6 (6.3%)	7 (4.7%)	7 (5.0%)	6 (4.6%)	5 (3.8%)	32 (4.7%)
Known HCV positive	0 (0.0%)	1 (1.0%)	1 (0.7%)	0 (0.0%)	1 (0.8%)	2 (1.5%)	5 (0.7%)
Known HIV positive	1 (2.6%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	2 (1.5%)	1 (0.8%)	4 (0.6%)

Table 9. Activity/procedure involved, contact specimen and the implicated device in health care workers with occupational exposure

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
Activity/procedure							
Blood-taking/IV catheter insertion	4 (13.3%)	15 (12.3%)	26 (15.6%)	23 (14.9%)	29 (18.5%)	10 (10.3%)	107 (14.7%)
Injection including recap	6 (20.0%)	20 (16.4%)	29 (17.4%)	26 (16.9%)	18 (11.5%)	13 (13.4%)	112 (15.4%)
Other bedside/treatment room procedures	9 (30.0%)	34 (27.9%)	47 (28.1%)	28 (18.2%)	29 (18.5%)	15 (15.5%)	162 (22.3%)
Cleansing/tidying up after procedures	4 (13.3%)	16 (13.1%)	20 (12.0%)	30 (19.5%)	22 (14.0%)	21 (21.6%)	113 (15.5%)
Other	7 (23.3%)	37 (30.3%)	45 (26.9%)	47 (30.5%)	57 (36.3%)	34 (35.1%)	227 (31.2%)
Contact specimen							
Blood/blood products	12 (40.0%)	33 (27.0%)	51 (30.5%)	53 (34.4%)	54 (34.4%)	44 (45.4%)	247 (34.0%)
Blood-contaminated fluid	9 (30.0%)	38 (31.1%)	32 (19.2%)	5 (3.2%)	0 (0.0%)	1 (1.0%)	85 (11.7%)
Saliva/urine	6 (20.0%)	29 (23.8%)	56 (33.5%)	25 (16.2%)	9 (5.7%)	7 (7.2%)	132 (18.2%)
Other/unknown	3 (10.0%)	22 (18.0%)	28 (16.8%)	71 (46.1%)	94 (59.9%)	45 (46.4%)	263 (36.2%)
Technical device							
Hollow-bore needle	13 (43.3%)	53 (43.4%)	80 (47.9%)	82 (53.2%)	83 (52.9%)	47 (48.5%)	358 (49.2%)
Lancet	7 (23.3%)	13 (10.7%)	24 (14.4%)	21 (13.6%)	20 (12.7%)	10 (10.3%)	95 (13.1%)
Dental instrument	5 (16.7%)	12 (9.8%)	22 (13.2%)	16 (10.4%)	16 (10.2%)	19 (19.6%)	90 (12.4%)
Other	2 (6.7%)	20 (16.4%)	20 (12.0%)	23 (14.9%)	23 (14.6%)	17 (17.5%)	105 (14.4%)

Nil	3 (10.0%)	24 (19.7%)	21 (12.6%)	12 (7.8%)	15 (9.6%)	4 (4.1%)	79 (10.9%)
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Table 10. Activity/procedure involved in medical and dental professionals with occupational exposure

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
Activity/procedure							
Blood-taking/IV catheter insertion	0 (0.0%)	3 (9.7%)	8 (15.1%)	5 (13.2%)	8 (20.0%)	0 (0.0%)	24 (11.3%)
Injection including recap	2 (28.6%)	8 (25.8%)	10 (18.9%)	8 (21.1%)	7 (17.5%)	10 (23.3%)	45 (21.2%)
Other bedside/treatment room procedures	2 (28.6%)	12 (38.7%)	23 (43.4%)	10 (26.3%)	9 (22.5%)	10 (23.3%)	66 (31.1%)
Cleansing/tidying up after procedures	3 (42.9%)	7 (22.6%)	9 (17.0%)	12 (31.6%)	7 (17.5%)	12 (27.9%)	50 (23.6%)
Surgery in operating theatre	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	3 (7.5%)	3 (7.0%)	6 (2.8%)
Sharps disposal	0 (0.0%)	1 (3.2%)	2 (3.8%)	2 (5.3%)	3 (7.5%)	7 (16.3%)	15 (7.1%)
Other	0 (0.0%)	0 (0.0%)	1 (1.9%)	1 (2.6%)	3 (7.5%)	1 (2.3%)	6 (2.8%)

Table 11. Activity/procedure involved in nursing professionals with occupational exposure

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
Activity/procedure							
Blood-taking/IV catheter insertion	4 (19.0%)	10 (19.6%)	16 (22.2%)	15 (23.4%)	14 (21.5%)	6 (23.1%)	65 (21.7%)
Injection including recap	4 (19.0%)	12 (23.5%)	18 (25.0%)	17 (26.6%)	11 (16.9%)	3 (11.5%)	65 (21.7%)
Other bedside/treatment room procedures	7 (33.3%)	16 (31.4%)	22 (30.6%)	13 (20.3%)	11 (16.9%)	2 (7.7%)	71 (23.7%)
Cleansing/tidying up after procedures	1 (4.8%)	3 (5.9%)	2 (2.8%)	3 (4.7%)	5 (7.7%)	2 (7.7%)	16 (5.4%)
Surgery in operating theatre	0 (0.0%)	2 (3.9%)	5 (6.9%)	5 (7.8%)	3 (4.6%)	6 (23.1%)	21 (7.0%)
Sharps disposal	3 (14.3%)	1 (2.0%)	4 (5.6%)	4 (6.3%)	11 (16.9%)	4 (15.4%)	27 (9.0%)
Other	2 (9.5%)	7 (13.7%)	5 (6.9%)	7 (10.9%)	9 (13.8%)	2 (7.7%)	32 (10.7%)

Table 12. Exposures with risk factors that may or may not be related to HIV transmission in health care workers with occupational exposure (n=498)

	*Higher risk	**Lower risk
1999 (Jul-Dec)	15 (50.0%)	7 (23.3%)
2000	54 (44.3%)	25 (20.5%)
2001	73 (43.7%)	50 (29.9%)
2002	98 (63.6%)	4 (2.6%)
2003	100 (63.7%)	7 (4.5%)
2004	62 (63.9%)	3 (3.1%)
Total	402 (55.3%)	96 (13.2%)

*risk factors of: deep percutaneous injury, involving procedures with device placed in a blood vessel, involving a hollow-bore needle, device which is visibly contaminated with blood, source person with AIDS

**risk factors of: moderate percutaneous injury, mucosal contact, contact with deep body fluids other than blood, source person is HIV infected but not or not sure to the stage of AIDS, other reasons contributing to increased risk

Table 13. Frequency of risk factors in health care workers with presence of risk factors from occupational exposure

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
Higher risk							
1 risk factor	13 (43.3%)	47 (38.5%)	61 (36.5%)	92 (59.7%)	99 (63.1%)	59 (60.8%)	371 (51.0%)
2 risk factors	1 (3.3%)	3 (2.5%)	3 (1.8%)	6 (3.9%)	1 (0.6%)	2 (2.1%)	16 (2.2%)
>=3 risk factors	1 (3.3%)	4 (3.3%)	9 (5.4%)	0 (0.0%)	0 (0.0%)	1 (1.0%)	15 (2.1%)
Lower risk							
1 risk factor	7 (23.3%)	24 (19.7%)	50 (29.9%)	4 (2.6%)	7 (4.5%)	3 (3.1%)	95 (13.1%)
2 risk factors	0 (0.0%)	1 (0.8%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.1%)
>=3 risk factors	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)

Table 14. Degree of presence of risk factors in health care workers who sustained higher risk occupational exposure during various activities/procedures

	No. of workers	No. of risk factors		
		Range	Mean	(95% CI)
Blood-taking/IV catheter insertion	98	1-3	1.39	1.25-1.53
Injection including recap	107	1-2	1.02	0.99-1.05
Other bedside/treatment room procedures	59	1-2	1.03	0.98-1.08
Cleansing/tidying up after procedures	52	1-1	1.00	-
Surgery in operating theatre	19	1-1	1.00	-
Sharps disposal	59	1-3	1.05	0.97-1.13
Other	51	1-2	1.04	0.99-1.10

Table 15. Precautions (can be more than one) taken during the exposure among health care workers with occupational exposure

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
Glove	8 (26.7%)	47 (38.5%)	79 (47.3%)	75 (48.7%)	97 (61.8%)	74 (76.3%)	380 (52.3%)
Goggle/glasses/mask	2 (6.7%)	2 (1.6%)	5 (3.0%)	5 (3.2%)	0 (0.0%)	2 (2.1%)	16 (2.2%)
Gown/apron	3 (10.0%)	1 (0.8%)	5 (3.0%)	4 (2.6%)	2 (1.3%)	5 (5.2%)	20 (2.8%)
Nil	2 (6.7%)	28 (23.0%)	20 (12.0%)	12 (7.8%)	8 (5.1%)	3 (3.1%)	73 (10.0%)

Table 16. Time lag in hours between exposure and first medical consult

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure							
<2	15 (50.0%)	66 (54.1%)	91 (54.5%)	77 (50.0%)	100 (63.7%)	54 (55.7%)	403 (55.4%)
2-12	7 (23.3%)	25 (20.5%)	47 (28.1%)	57 (37.0%)	46 (29.3%)	30 (30.9%)	212 (29.2%)
12-24	1 (3.3%)	7 (5.7%)	9 (5.4%)	5 (3.2%)	5 (3.2%)	5 (5.2%)	32 (4.4%)
24-48	4 (13.3%)	6 (4.9%)	6 (3.6%)	8 (5.2%)	2 (1.3%)	2 (2.1%)	28 (3.9%)
48-72	3 (10.0%)	5 (4.1%)	4 (2.4%)	1 (0.6%)	0 (0.0%)	2 (2.1%)	15 (2.1%)
>72	0 (0.0%)	13 (10.7%)	10 (6.0%)	6 (3.9%)	4 (2.5%)	4 (4.1%)	37 (5.1%)
Median (inter-quartile range)	1.9 (0.8-21.6)	1.7 (0.7-13.4)	1.4 (0.5-5.3)	2.0 (1-3.2)	1 (0.5-3.0)	1.3 (0.7-4)	1.6 (0.7-4)
B. All other exposure cases							
<2	55 (55.6%)	127 (47.7%)	163 (42.9%)	116 (39.3%)	102 (44.2%)	92 (41.4%)	655 (43.9%)
2-12	21 (21.2%)	57 (21.4%)	96 (25.3%)	118 (40.0%)	81 (35.1%)	93 (41.9%)	466 (31.2%)
12-24	8 (8.1%)	24 (9.0%)	37 (9.7%)	21 (7.1%)	15 (6.5%)	16 (7.2%)	121 (8.1%)
24-48	8 (8.1%)	19 (7.1%)	34 (8.9%)	15 (5.1%)	12 (5.2%)	13 (5.9%)	101 (6.8%)
48-72	2 (2.0%)	9 (3.4%)	15 (3.9%)	5 (1.7%)	8 (3.5%)	1 (0.5%)	40 (2.7%)
>72	5 (5.1%)	30 (11.3%)	35 (9.2%)	20 (6.8%)	13 (5.6%)	7 (3.2%)	110 (7.4%)
Median (inter-quartile range)	1.2 (0.6-10.4)	2 (0.7-20.0)	2.4 (1-21.5)	2.7 (1.3-7.9)	2.3 (1-7)	2.3 (1.0-6)	2.3 (1-11.3)

Table 17. Baseline epidemiology of hepatitis B, hepatitis C and HIV in the *exposed persons

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure							
HBsAg+ve	2 (8.0%)	5 (6.3%)	5 (4.8%)	10 (9.9%)	6 (6.0%)	5 (7.8%)	33 (7.0%)
Anti-HBs+ve	10 (45.5%)	59 (77.6%)	59 (54.6%)	72 (71.3%)	71 (71.0%)	41 (65.1%)	312 (66.2%)
Anti-HCV+ve	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Anti-HIV+ve	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
B. All other exposure cases							
HBsAg+ve	13 (14.0%)	22 (9.4%)	24 (7.3%)	23 (8.6%)	25 (11.8%)	15 (7.7%)	122 (9.2%)
Anti-HBs+ve	43 (53.8%)	95 (44.8%)	149 (47.6%)	143 (55.9%)	84 (44.4%)	107 (56.6%)	621 (50.1%)
Anti-HCV+ve	0 (0.0%)	1 (6.3%)	1 (2.0%)	1 (2.0%)	0 (0.0%)	0 (0.0%)	3 (1.5%)
Anti-HIV+ve	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)

*Number of cases tested for each blood-borne pathogens varied

Table 18. Number and proportion of all exposed persons with prescription of HIV post-exposure prophylaxis at Accident & Emergency Department or Therapeutic Prevention Clinic

	Health care work workers with occupational exposure	All other exposure cases	Total
1999 (Jul-Dec)	3 (10.0%)	1 (1.0%)	4 (3.1%)
2000	2 (1.6%)	4 (1.5%)	6 (1.5%)
2001	6 (3.6%)	4 (1.1%)	10 (1.8%)
2002	4 (2.6%)	4 (1.4%)	8 (1.8%)
2003	8 (5.1%)	4 (1.7%)	12 (3.1%)
2004	1 (1.0%)	5 (2.3%)	6 (1.9%)
Total	24 (3.3%)	22 (1.5%)	46 (2.1%)

Table 19. Time lag between injury and start of HIV PEP

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure							
Mean (hours)	3.2	5.0	4.3	7.4	6.2	4.0	5.4
Median (hours)	3.9	5.0	2.4	3.1	3.7	4.0	3.1
No. <=2 hours	1	1	3	2	3	0	10
No. <=24 hours	3	2	6	4	8	1	24
B. All other exposure cases							
Mean (hours)	37.7	14.2	14.4	16.2	14.8	15.6	16.1
Median (hours)	37.7	13.4	15.8	18.3	15.2	16.7	16.7
No. <=2 hours	0	1	0	0	0	1	2
No. <=24 hours	0	3	4	5	4	4	20

Table 20. Clients with PEP initiated who were continued treatment at TPC

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure	1 (33.3%)	1 (50.0%)	5 (83.3%)	3 (75.0%)	6 (75.0%)	1 (100.0%)	17 (70.8%)
B. All other exposure cases	0 (0.0%)	0 (0.0%)	0 (0.0%)	2 (50.0%)	3 (75.0%)	2 (40.0%)	7 (31.8%)

Table 21. Outcome of exposed persons who were continued on PEP at TPC regarding their PEP intake and HIV status of source

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure							
Known PEP intake outcome	1 (100.0%)	1 (100.0%)	4 (80.0%)	3 (100.0%)	6 (100.0%)	1 (100.0%)	16 (94.1%)
Source confirmed HIV positive	0 (0.0%)	0 (0.0%)	1 (20.0%)	0 (0.0%)	3 (50.0%)	0 (0.0%)	4 (23.5%)
B. All other exposure cases							
Known PEP intake outcome	-	-	-	2 (100.0%)	2 (66.7%)	2 (100.0%)	6 (85.7%)
Source confirmed HIV positive	-	-	-	0 (0.0%)	1 (33.3%)	1 (50.0%)	2 (28.6%)

Table 22. PEP regimens prescribed to exposed persons with known PEP intake outcome

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure							
2 drugs	1 (100.0%)	-	-	1 (33.3%)	-	-	2 (12.5%)
3 drugs	-	1 (100.0%)	4 (100.0%)	2 (66.7%)	6 (100.0%)	1 (100.0%)	14 (87.5%)
B. All other exposure cases							
2 drugs	-	-	-	-	-	2 (100.0%)	2 (33.3%)
3 drugs	-	-	-	2 (100.0%)	2 (100.0%)	-	4 (66.7%)

Table 23. Experience of toxicity in exposed persons with known PEP intake outcome

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure							
Had toxicity							
2 drugs	1 (100.0%)	-	-	1 (100.0%)	-	-	2 (100.0%)
3 drugs	-	1 (100.0%)	4 (100.0%)	2 (100.0%)	6 (100.0%)	1 (100.0%)	14 (100.0%)
2/3 drugs	1 (100.0%)	1 (100.0%)	4 (100.0%)	3 (100.0%)	6 (100.0%)	1 (100.0%)	16 (100.0%)
Moderate to severe toxicity	1 (100.0%)	0 (0.0%)	1 (25.0%)	3 (100.0%)	4 (66.7%)	0 (0.0%)	9 (56.3%)
B. All other exposure cases							
Had toxicity							
2 drugs	-	-	-	-	-	2 (100.0%)	2 (100%)
3 drugs	-	-	-	1 (50.0%)	1 (50.0%)	-	2 (50%)
2/3 drugs	-	-	-	1 (50.0%)	1 (50.0%)	2 (100.0%)	4 (66.7%)
Moderate to severe toxicity	-	-	-	1 (50.0%)	0 (0.0%)	1 (50.0%)	2 (33.3%)

Table 24. Completion of PEP in exposed persons with known PEP intake outcome

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure							
2 drugs	1 (100.0%)	-	-	0 (0.0%)	-	-	1 (50%)
3 drugs	-	0 (0.0%)	2 (50.0%)	0 (0.0%)	2 (33.3%)	1 (100.0%)	5 (35.7%)
2/3 drugs	1 (100.0%)	0 (0.0%)	2 (50.0%)	0 (0.0%)	2 (33.3%)	1 (100.0%)	6 (37.5%)
B. All other exposure cases							
2 drugs	-	-	-	-	-	2 (100.0%)	2 (100%)
3 drugs	-	-	-	1 (50.0%)	1 (50.0%)	-	2 (50%)
2/3 drugs	-	-	-	1 (50.0%)	1 (50.0%)	2 (100.0%)	4 (66.7%)

Table 25. Duration of PEP in exposed persons with known PEP intake outcome

	1999 (Jul-Dec)		2000		2001		2002		2003		2004		Total	
A. Health care workers with occupational exposure	Mean	Median	Mean	Median	Mean	Median	Mean	Median	Mean	Median	Mean	Median	Mean	Median
2 drugs	28.00	28.00	-	-	-	-	5.00	5.00	-	-	-	-	16.5	16.5
3 drugs	-	-	7.00	7.00	19.75	20.50	14.00	14.00	12.50	6.50	28.00	28.00	15.5	11.5
2/3 drugs	28.00	28.00	7.00	7.00	19.75	20.50	11.00	7.00	12.50	6.50	28.00	28.00	15.6	11.5
B. All other exposure cases														
2 drugs	-	-	-	-	-	-	-	-	-	-	28.00	28.00	28.00	28.00
3 drugs	-	-	-	-	-	-	15.50	15.50	21.00	21.00	-	-	18.25	21.00
2/3 drugs	-	-	-	-	-	-	15.50	15.50	21.00	21.00	28.00	28.00	21.50	28.00

Table 26. Reasons (can be >1) not completing PEP in exposed persons with known PEP intake outcome who were prescribed 2 or 3 drugs

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure							
Adverse effects	0 (0.0%)	0 (0.0%)	1 (25.0%)	3 (100.0%)	3 (50.0%)	0 (0.0%)	7 (43.8%)
Source confirmed HIV negative	0 (0.0%)	1 (100.0%)	2 (50.0%)	1 (33.3%)	2 (33.3%)	0 (0.0%)	6 (37.5%)
B. All other exposure cases							
Adverse effects	-	-	-	1 (50.0%)	0 (0.0%)	0 (0.0%)	1 (16.7%)
Source confirmed HIV negative	-	-	-	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)

Table 27. Promptness and completion of HIV PEP among exposures to known HIV positive source

	1999 (Jul-Dec)	2000	2001	2002	2003	2004	Total
A. Health care workers with occupational exposure	n= 0	n= 0	n= 1	n= 0	n= 3	n= 0	n=4
Number with PEP prescribed	-	-	1 (100.0%)	-	3 (100%)	-	4 (100%)
Median time lag of PEP (hours)	-	-	2.7	-	1.9	-	2.3
PEP within 2 hours	-	-	0 (0.0%)	-	2 (66.7%)	-	2 (50%)
PEP within 24 hours	-	-	1 (100.0%)	-	1 (33.3%)	-	4 (100%)
Completion of PEP	-	-	1 (100.0%)	-	2 (66.7%)	-	3 (75%)
B. All other exposure cases	n= 0	n= 0	n= 0	n= 0	n= 1	n= 1	n=2
Number with PEP prescribed	-	-	-	-	1 (100%)	1 (100.0%)	2 (100%)
Median time lag of PEP (hours)	-	-	-	-	13.3	21	17.2
PEP within 2 hours	-	-	-	-	0 (0.0%)	0 (0.0%)	0 (0%)
PEP within 24 hours	-	-	-	-	1 (100%)	1 (100.0%)	2 (100%)
Completion of PEP	-	-	-	-	0 (0.0%)	1 (100.0%)	1 (50%)

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ABBREVIATIONS

AIDS	Acquired immunodeficiency syndrome
A&E Department	Accident & Emergency Department
BBV	Blood-borne viruses
CDC	Centers for Disease Control and Prevention, US
CHP	Centre for Health Protection
HAART	Highly active antiretroviral therapy
HBIg	Hepatitis B immunoglobulin
HBV	Hepatitis B virus
HCV	Hepatitis C virus
HCW	Health care worker
HIV	Human immunodeficiency virus
HPA	Health Protection Agency, UK
ITC	Integrated Treatment Centre
NRTI	Nucleoside reverse transcriptase inhibitor
PEP	Post exposure prophylaxis
PI	Protease inhibitor
TPC	Therapeutic Prevention Clinic

REFERENCES

1. Bell DM. Occupational risk of human immunodeficiency virus infection in healthcare workers: An overview. *Am J Med* 1997;102(suppl 5B):9-15.
2. Ippolito G, Puro V, De Carli G, the Italian Study Group on Occupational Risk of HIV Infection. The risk of occupational human immunodeficiency virus infection in health care workers. *Arch Intern Med* 1993;153:1451-8.
3. Anon. Needlestick transmission of HTLV-III from a patient infected in Africa. *Lancet* 1984;ii:1376-7.
4. Occupational Health Protection Agency Centre for Infections & Collaborators. Occupational transmission of HIV. Summary of published reports. March 2005.
5. Gerberding JL. Management of occupational exposures to blood-borne exposures. *Engl J Med* 1995;332:444-51.
6. US CDC. Recommendations for follow-up of health care workers after occupational exposure to hepatitis C virus. *MMWR*. 1997;46:603-6.
7. Health Protection Agency Centre for Infections, National Public Health Service for Wales, CDSC Northern Ireland. Eye of the needle. Surveillance of significant occupational exposure to blood-borne viruses in healthcare workers. England, Wales and Northern Ireland, Seven-year report: January 2005.
8. Cardo DM, Culver DH, Ciesielski CA, et al. A case-control study of HIV seroconversion in health-care workers after percutaneous exposure. *N Engl J Med* 1997;337:1485-90.
9. US CDC. Updated US Public Health Service guidelines for the management of occupational exposures to HBV, HCV, and HIV and recommendations for post exposure prophylaxis. *MMWR* 2001;50:RR-11.

10. US CDC. Updated US Public Health Service guidelines for the management of occupational exposures to HIV and recommendations for post exposure prophylaxis. MMWR 2005;54:RR-9.
11. UK Health Department. HIV post-exposure prophylaxis: guidance from the UK Chief Medical Officers Expert Advisory Group on AIDS. July 2000.
12. Euro-NONPEP Project Group. Proposed recommendations for the management of HIV post-exposure prophylaxis after sexual, injecting drug or other exposures in Europe. Eurosurveillance 2004 Vol 9(2): 35-43.
13. US Department of Health and Human Services. Antiretroviral postexposure prophylaxis after sexual, injection-drug use, or other nonoccupational exposure to HIV in the United States. MMWR 2005;54:RR-2.
14. Scientific Committee on AIDS & Scientific Working Group on Viral Hepatitis Prevention. Recommendations on the management and postexposure prophylaxis of needlestick injury or mucosal contact to HBV, HCV and HIV. 2003
15. Hospital Authority. A&E clinical guideline No. 6. Management of needlestick injury or mucosal contact with blood or body fluid. February 2005.
16. Ippolito G, Puro V, Petrosillo N, De Carli G, and the Italian Study Group on Occupational Risk of HIV Infection. Surveillance of occupational exposure to bloodborne pathogens in health care workers: the Italian national programme. Eurosurveillance 1999;4:33-6.

APPENDIX. Therapeutic prevention of HIV infection. HIV Manual 2001. Chapter 3

3.1 Management of accidental percutaneous to HIV

Percutaneous or mucosal exposure to HIV-contaminated blood or body fluids in health care setting is a rare but recognized mode of HIV transmission. It has been estimated that the average risk of infection after percutaneous exposure is 0.3%¹ whereas that of mucosal membrane exposure is about 0.09%.² As of end of December 1999, 102 definite and 217 possible occupationally acquired HIV (OAI) infections had been reported globally.³ Amongst these OAI, nurses and doctors were the most frequently implicated health care workers (HCW). Nevertheless, although OAI occurs, it accounts for just a fraction of the known HIV infections in HCW and therefore plays an infinitesimal role in the global HIV epidemic.

Attempts have been made to prevent HIV infection by the percutaneous route through administration of antiretroviral chemoprophylaxis, before or after exposure. Evidence of its effectiveness is available in both animal and human studies. In chimpanzees receiving nevirapine, HIV-1 challenge did not result in infection.⁴ In another animal model, antiretroviral drug given after simian immunodeficiency virus (SIV) exposure prevented virus transmission.⁵ In humans, a case-control study of health care workers who sustained mainly percutaneous injury also found efficacy of zidovudine in protecting against seroconversion.⁶

Despite the small absolute risk of infection, occupational exposure to HIV in health care setting could pose a significant psychological burden to the injured. Post-exposure management offers a unique chance for therapeutic prevention of HIV infection. Humane counseling, professional advice, together with appropriate treatment shall aim at minimizing psychosocial as well as physical morbidity consequent to the exposure. Currently, management of potential exposure to HIV has been one major focus of the scope of Accident & Emergency Departments and HIV care units in Hong Kong. Exposure to known HIV-infected patients is rare and a handful of post-exposure prophylaxis has been prescribed over the last years. There was no known incident of seroconversion upon accidental exposure in health care setting locally.

Primary prevention of occupational exposure to blood or body fluids is crucial. Adherence to standard infection control practice minimizes the occurrence of injury. Some 'accidents' are avoidable. Universal precautions protect HCW from blood-borne diseases beyond HIV, irrespective of the HIV status (whether known or unknown) of those involved.

The management of an incident of occupational exposure involves proper risk assessment, counselling tailored to the need of individual client, and the prescription of antiretroviral drugs to prevent HIV transmission if the risk is substantial. These are discussed in the following sections. An algorithm is provided at the end of the Chapter.

I. Risk assessment of exposure

HIV transmission can potentially occur in the event of a significant exposure to body fluids/tissues (e.g. blood, cerebrospinal fluid) which may harbor infectious blood-borne viruses from an HIV/AIDS patient. Risk assessment is the most important component of post-exposure management and should be commenced as soon as possible after exposure. The risk of transmission depends on (i) type and extent of exposure, and (ii) HIV status and stage of the source. In assessing the exposure, multiple factors need to be considered:

- a. time lag between the incident of exposure and clinical consultation;
- b. nature of exposure, i.e. (breached) skin, mucosal or percutaneous;
- c. type of contact specimen;
- d. severity of injury;

- e. amount of blood/body fluids transferred to the injured;
- f. device involved in injury; and
- g. protective measures during the exposure.

Some factors of the accident itself are associated with a higher potential of seroconversion after percutaneous exposure to HIV-infected blood:⁶

- a. injury with a device visibly contaminated with the patient's blood;
- b. a procedure that involved a needle directly placed in a vein or artery;
- c. deep injury; and
- d. exposure to source patients with AIDS or high plasma viral burden.

No matter how extensive the exposure is, HIV transmission may only happen if the source is HIV infected. For known infected source, his/her stage of disease has bearing on the risk of transmission. It is, however, not uncommon that HIV status of the source is unknown or cannot be ascertained. In this case, the likelihood of HIV infection in the source shall be assessed by clues such as (a) HIV-related illnesses, e.g. *Pneumocystis carinii* pneumonia, oral thrush, (b) HIV-related risk behaviors, e.g. unprotected sex, multiple sex partners, needle-sharing for drug injection, and (c) HIV prevalence of the community group which the source belongs to. Re-evaluation of the exposed person should be considered if there is additional information after the first assessment, e.g. HIV status of source.⁷

II. Counseling and health advice

Counseling and psychological support is another crucial component of post-exposure management. This is often provided by the nursing as well as medical staff. The assessment and the determination of the risk of HIV transmission would provide a good case-by-case base for further counseling. A variety of areas need to be covered, including: general risk of infection after percutaneous or mucosal exposure, assessment of the specific exposure, usefulness and limitations of PEP, necessary investigations, necessary precautions, arrangement of follow-up care and others.

In counseling for HIV antibody testing, one might need to explore if the injured is already at risk of HIV infection before injury. Any previous HIV antibody testing and its results should be inquired. This is particularly relevant if PEP has to be prescribed. For infected individuals, the antiretroviral prophylaxis (regimen similar to antiretroviral therapy) will actually be treatment of the disease. A thorough assessment and plan would be a must for optimizing antiretroviral therapy for any infected patient.

All cases with potential risk of infection have to be warned about HIV seroconversion illness, for which they should seek medical consultations. This typically occurs 2-6 weeks after exposure. (see Chapter 4.5) Classical symptoms are fever, skin rash, sore throat, swollen lymph glands especially on neck, and ulcers in mouth/genitalia. Sometimes, it can just be non-specific symptoms such as fever, headache or malaise. Also, there was report of delayed HIV seroconversion (8 to 9.5 months after exposure) in a health care worker who contracted both HIV and HCV after a needle-stick injury.⁸ Thus, symptoms suggestive of acute HIV infection should not be discarded lightly especially in the case of delayed presentation.

Similarly, the injured should be advised to take precautions while pending outcome of the exposure, if risk of HIV infection exists. For example, he/she should practice safer sex and not donate organ, blood and semen. Female should avoid pregnancy.

III. Blood investigations

A baseline HIV antibody test is needed for most of the injured. Its result serves as a reference for interpreting subsequent testing results, in case there is seroconversion after the exposure. Also, it can exclude an underlying HIV infection. One option is to test for HIV antibody only when subsequent follow-up blood samples are tested antibody positive. However, a baseline antibody test must be performed immediately if underlying HIV infection is suspected when an

injured is put on PEP. In such circumstance, baseline blood investigations of complete blood picture (CBP), renal/liver function tests (R/LFT) and sugar (if protease inhibitor given) should be performed concurrently. Creatinine kinase and amylase are optional. Baseline investigations for viral hepatitis B and C shall also be pursued as appropriate for the injured.

In some cases, testing of source patient for HIV antibody might assist in the management of the injured. This should however be done after clear explanation of the rationale to the source and consent obtained. Confidentiality should be upheld throughout.

Follow-up investigations depend on whether the injured has been put on PEP. For those who are not on HIV PEP, they can be followed up 3-6 months after the initial assessment and consultation. For those on PEP, clinical and biochemical (CBP, R/LFT, amylase, CPK, sugar (if protease inhibitor is prescribed)) monitoring for drug tolerance at week 0,2,4, and third month should be considered.

Follow-up HIV antibody test shall be performed at month 6. A test earlier at month 3 is optional. An additional month 12 testing may be considered case-by-case for high infection risk cases who have taken PEP, for fear of the possibility of delayed seroconversion. HIV antibody testing and HIV RNA are performed for cases presenting with suspected seroconversion illness. Plasma shall be stored for resistance testing in such case.

IV. Post-exposure prophylaxis

IV.A The scientific evidence

Pathogenically, there is time lag between HIV exposure, viral seeding, replication and systemic infection. Antiretrovirals may theoretically be able to intervene by inhibiting viral replication after exposure. Both animal and human studies have provided evidence for the efficacy of PEP. From animal studies, it has been demonstrated that size of the inoculum, timing and duration of PEP affects chance of infection. Success of post-exposure prophylaxis (PEP) depends on its early initiation. PEP initiated at 72 hours after exposure in animal models was often ineffective. A retrospective case-control study on health care workers revealed that after controlling for other factors, there was a 81% (95% confidence interval, 48-94%) reduction in risk of infection with the use of zidovudine (AZT) after percutaneous exposure.⁶ The efficacy of abbreviated postpartum regimens initiated after childbirth in reducing mother-to-child HIV transmission also provides support to the principle of PEP. Nevertheless, PEP is definitely not fool-proof, and failure of antiretroviral prophylaxis has been reported in both animal and human studies.

Given the small absolute risk of HIV transmission even with significant exposure to HIV-contaminated blood or body fluids, the prescription of PEP depends on a balance of risks and benefits, which should be explained clearly to the injured. Potential drawbacks of PEP include: toxicity of antiretrovirals, unknown long term effects in HIV positive and negative people, uncertain level of effectiveness, development of resistance, and unknown impacts to the course of disease if seroconversion occurs. Potential benefits of PEP are: reduction of the chance of infection for injury with significant risk.

IV.B The recommended regimens

Though only zidovudine has been demonstrated to be effective in clinical studies, combination antiretroviral PEP is now recommended (based on data from treatment of HIV-infected patients) for its greater suppression of viral replication, broader coverage of resistance strains, and thus the theoretical advantage of more effective prevention of HIV transmission. When indicated, PEP should be initiated as soon as possible.

The US CDC recommended a basic 2-drug regimen of AZT (200 mg tid or 300 mg bid) and 3TC (150 mg bid) for cases with risk of infection.⁹ The tablet of Combivir (AZT 300 mg +3TC 150 mg) can be considered and given bid. A protease inhibitor (PI) such as indinavir (800 mg Q8H) or nelfinavir (750 mg tid) is added in an expanded 3-drug regimen if there is higher risk for transmission.⁹ However, UK authority generally favored a 3-drug PI-containing (indinavir, followed by nelfinavir) regimen for all exposures where PEP is indicated.¹⁰ There is insufficient evidence to support whether the US differentiated 2-drug/3-drug approach or the UK blanket 3-drug approach is superior. Decision should be made on a case-by-case basis,

balancing against such factors as assessed risk, anticipated PEP efficacy and potential toxicity and tolerance for the injured.

The PEP regimen might need to be modified accordingly if resistance is known or suspected in source HIV strains. Suspicion is usually based on (a) failure of treatment in source, (b) prevalence of primary resistance in the locality, or (c) documented resistance by resistance assays. Evaluation of the treatment history and efficacy of the current regimen in the source patient is a must.

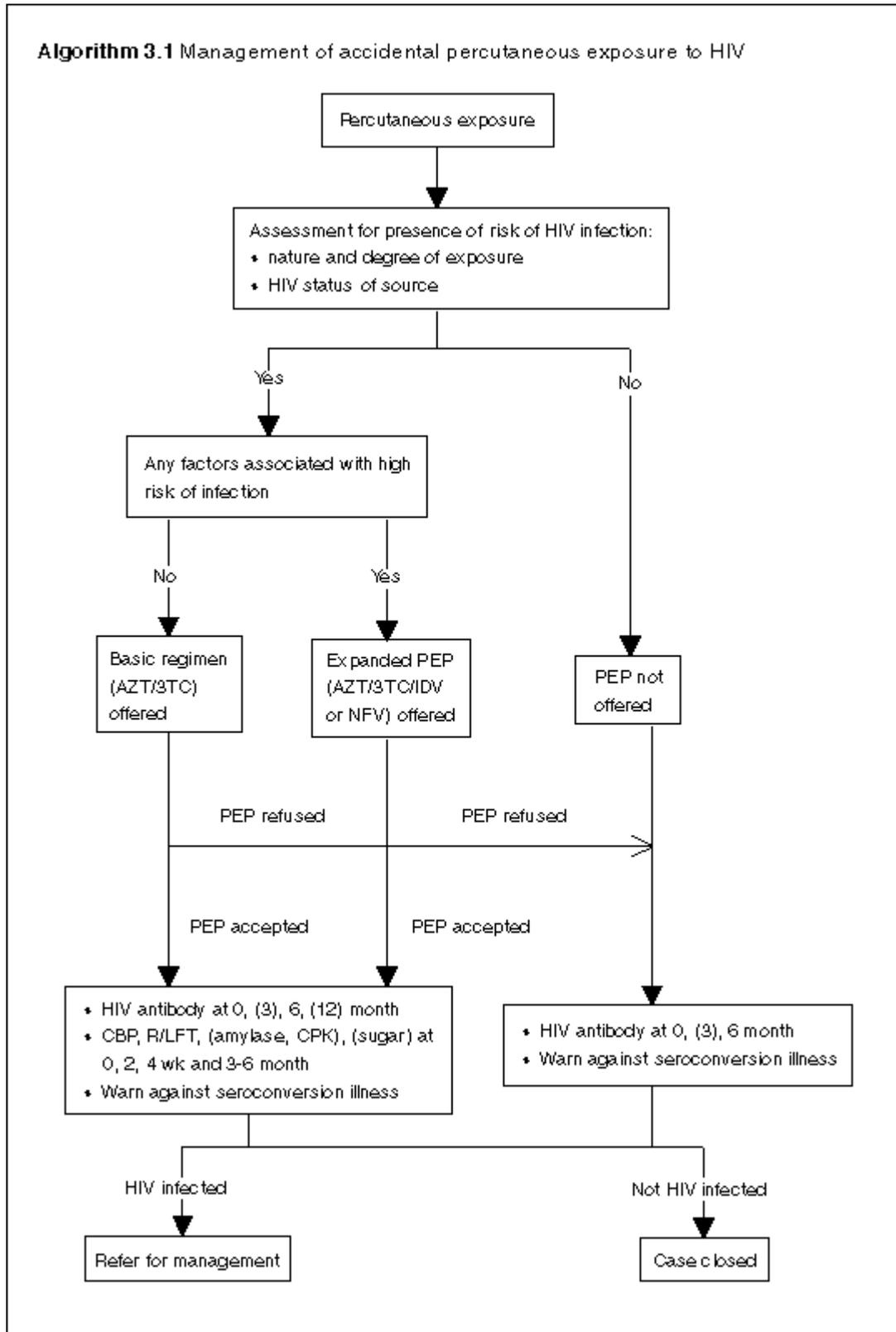
Lately, the US CDC suggested that nucleoside reverse transcriptase inhibitor other than AZT and 3TC could be considered, e.g. stavudine (d4T), didanosine (ddI).⁷ Other drugs such as efavirenz or abacavir can be the alternative for the third drug in the expanded regimen. However, the limited experience of using these alternative drugs for PEP shall be taken into account when choosing the regimen. The use of nevirapine to spare PI has caused severe morbidity and even deaths, which is thus contraindicated in PEP.¹¹

The dosage of common antiretrovirals and the potential side effects are in Box 3.1. Potential interactions of antiretrovirals with drugs that the injured is taking should be borne in mind. Special considerations are needed for those who are pregnant. For example, efavirenz is contraindicated because of its teratogenicity and indinavir should be avoided in late pregnancy for hyperbilirubinemia in newborn.

Box 3.1 Common antiretroviral preparations for post-exposure prophylaxis		
Drug	Dosage	Potential side effects
Zidovudine (ZDV, AZT)	200 mg tid/300 mg bid	Anemia, neutropenia, gastrointestinal upset, myalgia/ myositis
Lamivudine (3TC)	150 mg bid	GI upset, pancreatitis
Zidovudine + lamivudine (Combivir)	1 tablet bid	Anemia, neutropenia, gastrointestinal upset, myalgia/ myositis, pancreatitis
Indinavir (IDV, Crixivan)	800 mg q8h, empty stomach	nausea, vomiting, renal stone, unconjugated hyperbilirubinemia
Nelfinavir (NFV, Viracept)	750 mg tid/1250 bid with food	Diarrhea, rash

The optimal duration of PEP is unknown but a complete course is normally 4 weeks. This is prescribed in no less than 2 separate visits. Many HCWs who took PEP experienced one or more symptoms and a substantial proportion could not complete the course.^{12,13} Pretreatment counseling of all potential side effects might hopefully improve compliance of PEP. Besides blood investigations, toxicity should be evaluated clinically and managed accordingly. The best efforts should be made to have the exposed person complete the 4-week course if PEP is indicated. For PEP prescribed for unknown source who is subsequently found HIV negative, the PEP should be stopped.

Algorithm 3.1 Management of accidental percutaneous exposure to HIV



References

1. Bell DM. Occupational risk of human immunodeficiency virus infection in healthcare workers: An overview. *Am J Med* 1997;102(suppl 5B):9-15.
2. Ippolito G, Puro V, De Carli G, the Italian Study Group on Occupational Risk of HIV Infection. The risk of occupational human immunodeficiency virus infection in health care workers. *Arch Intern Med* 1993;153:1451-8.

3. PHLS AIDS & STD Centre at the Communicable Disease Surveillance Centre & Collaborators. *Occupational transmission of HIV*. December 1999.
4. Grob PM, Cao Y, Muchmore E, et al. Prophylaxis against HIV-1 infection in chimpanzees by nevirapine, a nonnucleoside inhibitor of reverse transcriptase. *Nature Med* 1997;3:665-70.
5. Tsai CC, Follis KE, Sabo A, et al. Prevention of SIV infection in macaques by (R)-9-(2-phosphononylmethoxypropyl)adenine. *Science* 1995;270:1197-9.
6. Cardo DM, Culver DH, Ciesielski CA, et al. A case-control study of HIV seroconversion in health-care workers after percutaneous exposure. *N Engl J Med* 1997;337:1485-90.
7. US CDC. Updated US Public health service guidelines for the management of occupational exposures to HBV, HCV, and HIV and recommendations for post exposure prophylaxis. *MMWR* 2001;50(RR-11):1-52.
8. Uridzon R, Gallagher K, Ciesielski C, et al. Simultaneous transmission of human immunodeficiency virus and hepatitis C virus from a needle-stick injury. *N Engl J Med* 1997;336:919-22.
9. US CDC. Public health service guidelines for the management of health care worker exposures to HIV and recommendations for post exposure prophylaxis. *MMWR* 1998;47(RR-7):1-33.
10. UK Health Department. *HIV post-exposure prophylaxis: guidance from the UK Chief Medical Officers Expert Advisory Group on AIDS*. July 2000.
11. US CDC. Serious adverse events attributed to nevirapine regimens for postexposure prophylaxis after HIV exposures – Worldwide, 1997-2000. *MMWR* 2001;49:1153-6.
12. NaSH Surveillance Group. Experience of health care workers taking antiretroviral agents as postexposure prophylaxis for occupational exposure to HIV. [Abstract 489] *National HIV Prevention Conference*, 1999.
13. Parin JM, Murphy M, Anderson J, et al. Tolerability and side-effects of post-exposure prophylaxis for HIV infection. *Lancet* 2000;355:722-3.

3.2 Non-occupational exposure – sex and intravenous drug use

The average **risk of HIV transmission** associated with receptive anal intercourse (0.8% to 3.2%) or receptive vaginal intercourse (0.05% to 0.15%) with an HIV-infected partner is similar to that with puncture by an HIV-contaminated needle. The risk of insertive vaginal intercourse is lower (0.03% to 0.09%). The risks associated with insertive anal intercourse and oral sex are unknown.¹ In addition to the mode of exposure, the per-contact risk of HIV transmission also varies significantly according to the stage of disease, viral load, mode of contact, virulence of HIV strain, and co-morbid factors (e.g. genital ulcerative disease, bleeding, trauma).

While the average risk in shared injecting drug use is estimated to be between 0.67% and 1%, this can vary widely, depending, among other factors, on the size of the needle and syringe, and the sequence of sharing.²

I. Rationale of postexposure prophylaxis other than percutaneous contact

It has been shown in a case-controlled study that ZDV prophylaxis alone reduced the risk of HIV transmission by 81%.³ In this study, exposure occurred mainly by the percutaneous route. As a result, the use of ZDV as postexposure prophylaxis (PEP) is now routinely recommended in percutaneous exposure to HIV, especially in health care workers.⁴ Nevertheless, the utility of PEP in this context is still subject to debate. Isolated case reports have documented both success (after transfusion)⁵ and failure (after needlestick).⁶

The recommendation on ZDV PEP is not extended to routinely cover sexual contact and shared injecting drug use because of the following concerns on efficacy and public health implications:

I.A Efficacy of PEP for non-occupational injuries

The mechanism of infection via a percutaneous needle stick is not identical to that via sexual contact or shared injecting drug use (IDU). In sexual contact, HIV is transmitted through mucosa; in shared IDU, local defenses are bypassed as the injection is intended to be intravenous.

The time limit of prophylactic treatment should be noted, as this is often less well-defined than a case of occupational exposure. In animal studies where HIV was inoculated intravenously, the efficacy of PEP decreases rapidly with time after exposure. Treatment initiated after 8 hours is much less likely to be effective, and after 72 hours useless.^{7,8} These studies essentially impose a **time limit** of 72 hours after which PEP should not be considered at all.

The use of a potent and usually more toxic drug combination does not automatically translate into efficacy since the viral load in the initial stage of HIV infection is small. In fact, up to one third of health care workers could not tolerate postexposure treatment with ZDV alone.⁹ In another trial of PEP after mostly sexual contact, only 78% completed a 4-week regimen of double NRTI therapy.¹⁰ Long term toxicity of antiretrovirals in HIV-negative individuals is still unknown.

There are hitherto limited data to validate that routine PEP after IDU or sex is useful. In particular, the assessment of HIV risk is currently not standardized. To date there is no consensus as to the optimal regimen and criteria for PEP after sex and shared IDU.

I.B Public health implications

The application of PEP cannot be undertaken lightly without addressing the possible impacts on public health in terms of alteration of sexual behavior, prevalence of drug resistant virus, cost-effectiveness, and service delivery.

All in all, the use of PEP on exposure through sexual and shared injecting drug use is based on theoretical considerations only, there having been no systematic study to address its efficacy. At the moment, decision on PEP will have to balance drug toxicity in an HIV-negative individual against a variable risk of infection and the unknown efficacy of PEP. Until this balance is properly addressed, routine PEP after sexual and IDU contact is hardly convincing.

Nevertheless, the assessment for PEP after at-risk contact does provide a good opportunity for risk-reduction counseling.

II. Management on potential exposure to HIV via sex or IDU

The steps of assessment, behavioral modification, consideration for post-exposure chemoprophylaxis and followup are considered in the management of a case of potential exposure through sex or drug injection. (algorithm at the end of the Chapter) There is a difference from the management of primary HIV infection when infection has presumably occurred (Chapter 4.5).

II.A Assessment

The following assessment shall be made on presentation:

- a. The type and time of contact
- b. Risk factors for HIV in the source - examination for concurrent STD is indicated
- c. Practice of High risk behavior
- d. Likelihood of repeat exposure

All clients shall receive baseline testing for HIV. Syphilis screening and hepatitis B and C serology may also be indicated.

II.B Behavior modification

It is almost certain that as a public health tool, PEP as we know it today has a cost-effectiveness ratio inferior to that of intensive risk-reduction counseling. Counseling is given on safer sex and the danger of needle-sharing, with a view to modifying high risk behaviors.

II.C Postexposure chemoprophylaxis

Due to the theoretical basis of chemoprophylaxis in this situation, this should be considered in the exceptional case where there is a high risk of transmission, no contraindication to the use of antiretrovirals and only if the client understands and accepts the risks involved.

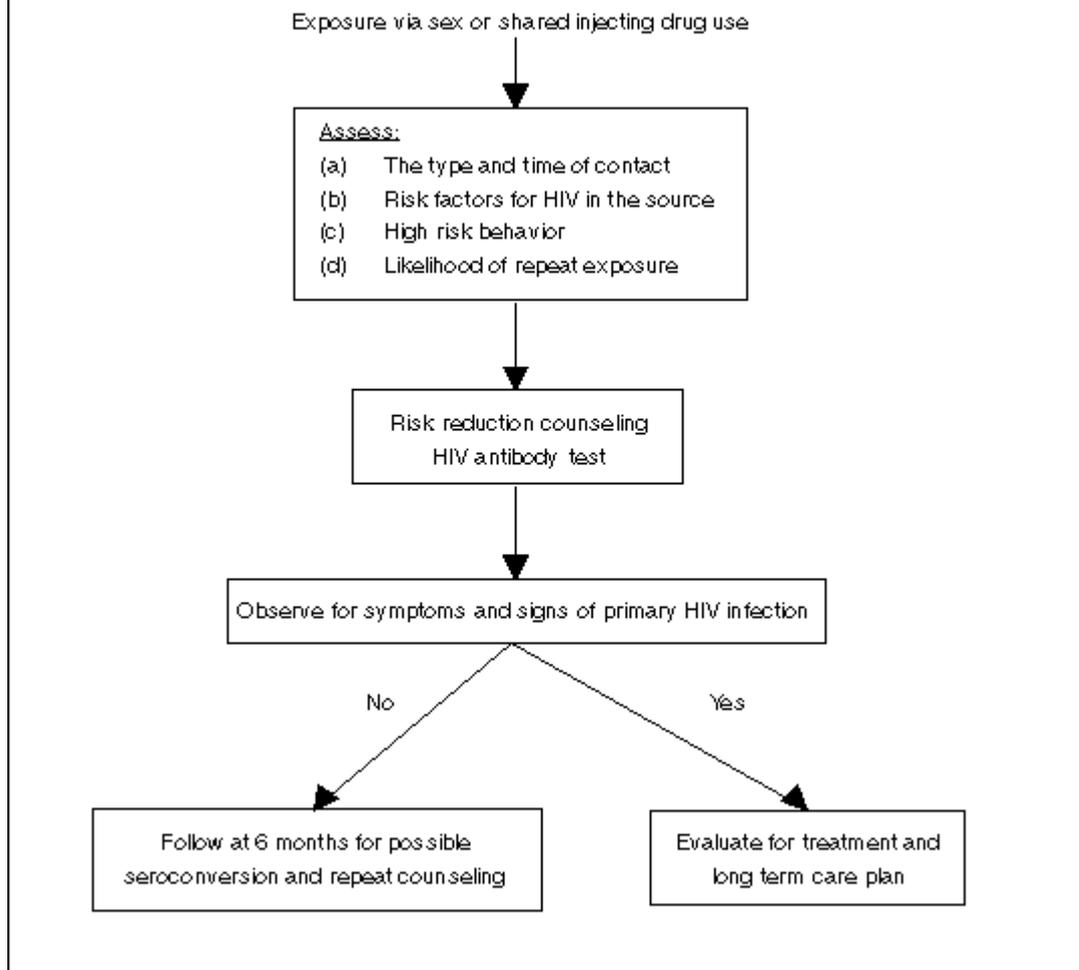
Were PEP to be used after exposure via sex and injecting drug use, the regimens would logically follow those in the percutaneous setting.¹¹ (refer to Chapter 3.1) Four weeks of zidovudine (ZDV), and lamivudine (3TC), with or without indinavir (IDV) would be the preferred choice. Alternatives are stavudine (d4T), didanosine (ddI) and nelfinavir (NFV). There is no consensus as to the number and kind of drugs that should be used.¹² However, it is noted that AZT is the only drug shown to be effective after percutaneous exposure in the health care setting. Non-nucleoside reverse transcriptase inhibitors (NNRTI) should normally not be used for this indication because of its low genetic barrier and its toxicity.¹³

It is emphasized that chemoprophylaxis in situations other than percutaneous contact should not be the rule. It should not be prescribed lightly without a thorough assessment and discussion with the patient who is likely to be in great anxiety and distress. As far as possible, treatment should form part of a clinical trial to derive the best information for the benefit of the index and other clients.

II.D Followup

Explanation should be given to clients of the symptoms and signs of primary HIV infection.¹⁴ Occurrence of such will prompt an evaluation, on a case-by-case basis, of the possibility of HIV antigen or viral load testing. Treatment of primary HIV infection itself is also fraught with controversy. Repeat testing for HIV antibody will be done for all the others to test for seroconversion. This provides a second opportunity of risk-reduction counseling.

Algorithm 3.2 Management of non-occupational exposure to HIV



References

1. Mastro TD, de Vincenzi I. Probabilities of sexual HIV-1 transmission. *AIDS* 1996;10(suppl A):S75-82.
2. Gaughwin MD, Gowans E, Ali R, et al. Bloody needles: the volumes of blood transferred in simulations of needlestick injuries and shared use of syringes for injection of intravenous drugs. *AIDS* 1991;5:1025-7.
3. Cardo DM, Culver DH, Ciesielski CA, et al and the CDC needle surveillance group. A case-control study of HIV seroconversion in health care workers after percutaneous exposure. *N Engl J Med* 1997;337(21):1485-90.
4. US CDC. Management of possible sexual, injecting-drug-use, or other nonoccupational exposure to HIV, including considerations related to antiretroviral therapy. A public health service statement. *MMWR* 1998;47:RR-17.
5. Katzenstein TL, Dickmeiss E, Aladdin H, et al. Failure To develop HIV Infection after receipt of HIV-Contaminated blood and postexposure prophylaxis. *Ann Intern Med* 2000;133:31-4.
6. Perdue B, Wolde Rufael D, Mellors J, et al. HIV-1 transmission by a needle-stick despite rapid initiation of four-drug postexposure prophylaxis. *6th Conference on Retroviruses and Opportunistic Infections*. Chicago 1999.
7. Tsai CC, Follis KE, Sabo A, et al. Prevention of SIV infection in Macaques by (R)-9-(2-Phosphonylmethoxypropyl)adenine. *Science* 1995;270:1197-9.

8. Martin LN, Murphey-Corb M, Soike KF, et al. Effects of initiation of 3'-azido, 3'-deoxythymidine (zidovudine) treatment at different times after infection of Rhesus monkeys with simian immunodeficiency virus. *J Infect Dis* 1993;168:825-35.
9. Ippolito G, Puro Vincenzo. Italian Registry of Antiretroviral Prophylaxis. *Am J Med* 1997;102(5B):58-62.
10. Kahn JO, Martin JN, Roland ME, et al. Feasibility of postexposure prophylaxis (PEP) against human immunodeficiency virus infection after sexual or injection drug use exposure: the San Francisco PEP Study. *J Infect Dis* 2001;183:707-14.
11. US Department of Health and Human Services. Public health service guidelines for the management of health care worker exposures to HIV and recommendations for post exposure prophylaxis. *MMWR* 1998;47:RR-7.
12. Saag M. Candidate antiretroviral agents for use in postexposure prophylaxis. *Am J Med* 1997;102(5B):25-31.
13. Gottlieb S. Nevirapine should not be prescribed for needlestick injuries. [news] *BMJ* 2001;322:126.
14. Schacker T, Collier AC, Hughes J, et al. Clinical and epidemiologic features of primary HIV infection. *Ann Intern Med* 1996;125:257-64.

