

## Original article

# Highly active antiretroviral therapy per se decreased mortality and morbidity of advanced human immunodeficiency virus disease in Hong Kong

CHAN Chi-wai , CHENG Lai-sim , CHAN Wai-kit and WONG Ka-hing

*Keywords* : highly active antiretroviral therapy · mortality · morbidity · acquired immunodeficiency syndrome

**Background** Morbidity and mortality of advanced human immunodeficiency virus infection ( HIV ) have declined in Western industrialized countries since the availability of highly active antiretroviral therapy ( HAART ). It is unclear if this has also happened in Hong Kong.

**Methods** We studied a retrospective cohort of patients with advanced HIV disease in Hong Kong , China. First , the mortality of advanced HIV disease per year was calculated for the decade 1993 to 2002 , both annually and according to patient observation before and after 1997. Second , the event rates were estimated for the clinical end points of acquired immune deficiency syndrome ( AIDS ) and death. Univariate and multivariate analyses were then performed to identify associated factors.

**Results** The crude mortality of advanced HIV disease declined from 10.8 – 30.4 per 100 patients during 1993 – 1996 , to 0.8 – 6.9 per 100 patients during 1997 – 2002. A rate ratio of 4.04 ( 95% CI , 2.52 – 6.47 ) was evident for those observed in 1993 – 1996 , compared to those in 1997 – 2002. In a multivariate analysis where calendar period was adjusted , use of highly active antiretroviral therapy was associated with rate ratios of 0.13 ( 95% CI , 0.05 – 0.33 ) for death after AIDS , 0.08 ( 95% CI , 0.04 – 0.19 ) for AIDS after a CD4 cell count < 200/μl , and 0.21 ( 95% CI , 0.07 – 0.67 ) for death after CD4 cell count < 200/μl. In the same analysis , calendar period ceased to be a significant factor after adjustment for use of HAART.

**Conclusions** The mortality and morbidity of advanced human immunodeficiency virus disease have declined in Hong Kong. This improved prognosis was attributable to the use of highly active antiretroviral therapy.

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General perception of human immunodeficiency virus infection ( HIV ) infection has changed from that of a deadly disease to one controllable with specific antiretroviral therapy. The change was concurrent with the availability of protease inhibitors in mid 1990s , which effectively expanded combination therapy from one comprising two drugs of the same class to one that was at least dual-class consisting of three or more drugs. The debut of non-nucleoside reverse transcriptase inhibitor closely followed that of protease inhibitors and served a similar role. The new combination therapy came to be known as highly active antiretroviral therapy ( HAART ) , and was quickly adopted in Hong Kong in 1997 , although it had been in use earlier in Western industrialized countries.

HAART has been efficacious , with the common occurrence of improved CD4 count and suppression of viral load to undetectable levels in clinical trials. However , these are surrogate markers<sup>1,2</sup> which may not fully capture important clinical end points. Furthermore , virologic success has been typically lower in population studies , and virologic efficacy did not invariably lead to immune restoration.<sup>3</sup> Regardless , population-based studies in Western

Integrated Treatment Centre , Department of Health , Hong Kong , China ( Chan CW , Cheng LS , Chan WK and Wong KH )

Correspondence to : Dr. CHAN Chi-wai , Integrated Treatment Centre , Department of Health , 9/F , 9 Kai Yan Street , Kowloon Bay , Hong Kong , China ( Tel : 852-21162930. Fax : 852-21170812. Email : kewchan@dhspp.net )

developed countries have shown improved survival and decrease in AIDS-defining illnesses since the advent of HAART.

Although Hong Kong has been using HAART since 1997, it is unclear if similar benefits have occurred here and, if they have, whether they would be attributable to the use of HAART. First, antiretroviral use, though important, is but one part of medical care. Second, with a low prevalence of HIV infection, Hong Kong has developed only a limited number of experienced health care providers in the care of HIV-infected patients.<sup>4</sup> Third, certain endemic diseases, such as mycobacterial and fungal infections, may differ between Western countries and Hong Kong.<sup>5</sup> Fourth, there may be unique socio-cultural contribution<sup>6</sup> to adherence which has been shown to be a major factor in the impact of HAART.<sup>7,8</sup> Even among developed countries, significant differences in survival have been shown.<sup>9</sup> As of now, HAART is expensive and will weigh heavily in the health care budget of most non-industrialized countries, including Hong Kong. A cost-effectiveness analysis using local data will be important to support public health policy on access to HAART.

To address the question whether HAART also decreased mortality and morbidity in Hong Kong, we analyzed a clinic cohort of patients with advanced HIV disease. The study began with a determination of the mortality rates of advanced HIV disease in the decade from 1993 to 2002. This showed a declining trend, coinciding with the availability of HAART. Thus we hypothesized that use of HAART improved survival. This hypothesis was further examined by analysis of the actual use of HAART and its relationship with clinical end points including death.

## METHODS

### Study population and design

In 1984 upon the first report of AIDS in Hong Kong, the Department of Health set up a designated HIV clinic for adult patients, later called the Integrated Treatment Centre. The cohort of patients who attended this clinic formed the study population. Another HIV clinic of Hong Kong was set up in 1991 and was affiliated with a tertiary care hospital. The referral bases for the two clinics were similar, except that the Integrated Treatment Centre received exclusive referrals from the tuberculosis and sexually transmitted disease clinics. These two

clinics take care of the vast majority of active patients in Hong Kong, as both offer antiretroviral treatment only for a fraction of its costs. Additional financial assistance is also given to those in need. In the Integrated Treatment Centre, a computerized clinical information system was put in place on July 14, 1999. Data were then collected prospectively of all patients with respect to demographics, diagnoses, treatment, investigation findings, and prescriptions. Prior corresponding data were obtained from retrospective chart review and entered into the database. These included early patients who failed to attend the clinic after July 14, 1999. Any patient who missed a follow up appointment was routinely contacted for disease status and rebooking. For those on HAART, adherence was monitored by self-report and pill count, and enforced by intensive counseling.<sup>6</sup> Adherence was generally good. Among 2000 assessments made in 2002, 1949 (97.5%) were of  $\geq 95\%$  adherence.

Antiretroviral treatment in the clinic began with zidovudine monotherapy in 1987, followed by dual nucleosides in 1994, and finally HAART in 1997. All antiretroviral drugs were dispensed on site. Routine use of viral load for monitoring started in 1998. The practice of prophylaxis and management of opportunistic infections essentially followed that of Western developed countries.<sup>10</sup> In addition, secondary prophylaxis against disseminated *Penicillium marneffei* was practiced. We have never participated in antiretroviral drug trials. As of end of 2002, approximately 600 patients, representing 30% of the cumulative reported HIV cases in Hong Kong, were regularly followed up in the clinic. Eighty-one percent of them were ethnic Chinese whereas about 14% and 4% were Caucasians and non-Chinese Asians respectively. All patients were aged 15 or above. Since 1997, annual default rate had been low at less than 4%.

### Data collection

For evaluation of crude mortality rate, the period from 1993 to 2002 was chosen because this decade spanned the transition to HAART. Only patients with advanced HIV disease were analyzed. Advanced HIV disease was defined by CD4 cell count  $< 200/\mu\text{l}$  without AIDS, or AIDS. For the latter, the AIDS surveillance case definition of the Hong Kong Advisory Council on AIDS was used.<sup>11</sup> This differed from that of the US Centers for Disease Control and Prevention<sup>12</sup> in that (1) pulmonary or cervical lymph node tuberculosis was counted as

AIDS-defining only with CD4 cell count < 200/ $\mu$ l ; ( 2 ) disseminated *Penicillium marneffei* infection was regarded as AIDS-defining ; and ( 3 ) CD4 cell count < 200/ $\mu$ l alone was not regarded as AIDS-defining. In our study , HAART was defined as treatment consisting of three or more antiretrovirals , at least one of which was a protease inhibitor or a non-nucleoside reverse transcriptase inhibitor. The crude mortality rate of patients with advanced disease was calculated according to calendar year of the decade. Differences in mortality rates were then tested by Chi-square test with Yates correction or Fisher 's exact test. The trend was tested by Chi-square test for trend. Patients with advanced HIV disease in the decade were also divided according to observation from 1993 to 1996 , and 1997 to 2002. The respective mortality rate was calculated , the denominator of which was follow-up duration measured in patient-months. The latter was the total number of months patients were followed , either in 1993 – 1996 or 1997 – 2002. Thus , patients who were seen only once would not contribute. A rate ratio of death and its 95% confidence interval ( CI ) were then obtained for the pre-HAART and HAART eras. Demographic and CD4 characteristics for patients in the pre-HAART and HAART eras were compared by Chi-square test.

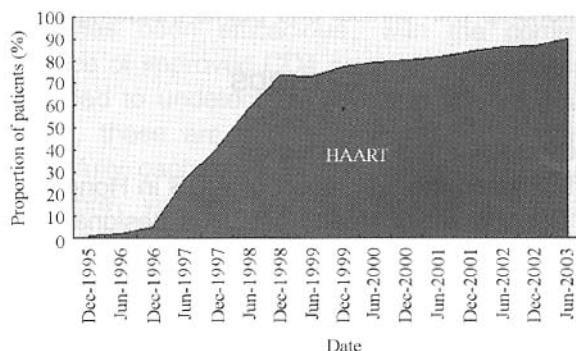
To directly measure the effects of HAART , we also assessed patients with advanced HIV disease from 1984 to mid-2003 for clinical end points. Advanced HIV disease was similarly defined as AIDS ( as defined above ) or CD4 cell count < 200/ $\mu$ l without AIDS. The clinical end points were ( 1 ) progression to death in those with AIDS , and progression to ( 2 ) AIDS and ( 3 ) death in those with CD4 cell count < 200/ $\mu$ l but without AIDS. Only patients seen more than once were included for analysis. Knowledge of the clinical end points was obtained during the course of follow-up and from contact with other health care providers or family members. Event rates were based on patient-years of observation , which were categorized into that which was on HAART and that which was not. The numerator was death or development of AIDS , depending on the clinical end point studied. The events were referred to their year of occurrence. Once a patient was started on HAART , his subsequent observation was classified as HAART even if it was discontinued or poorly adhered to. A patient might contribute observation time to one , two or three of the clinical end points.

**Statistical analysis**

The association between event rates and HAART , baseline CD4 cell count , baseline viral load and calendar period was expressed as a rate ratio , with confidence intervals and P values calculated from a Poisson regression model. Adjustment of covariates was then carried out to determine an adjusted rate ratio associated with use of HAART. Poisson regression was performed with the statistical software SAS Enterprise version 2 and the other analyses with SPSS version 12. A P value of 0. 05 was taken as statistically significant.

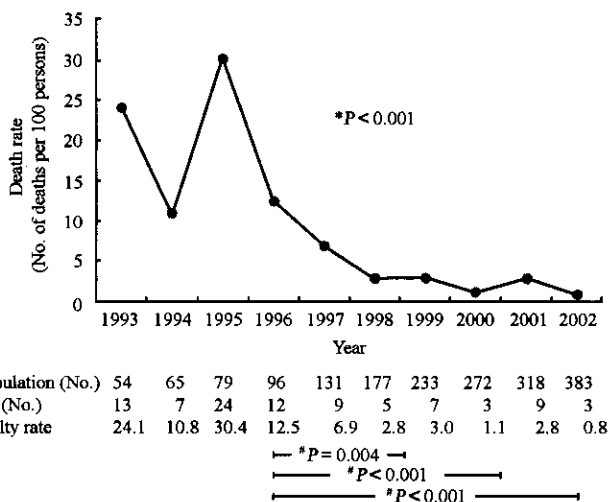
**RESULTS**

Among patients with advanced HIV infection , the use of HAART increased most rapidly in the year 1997 ( Fig. 1 ). In terms of crude mortality rate , it ranged from 10. 8 – 30. 4 per handred population in 1993 – 1996 , to 0. 8 – 6. 9 per handred population in 1997 – 2002. Using the mortality rate in 1996 as reference , statistically significant decreases were observed in 1998 , 2000 and 2002. An overall decreasing trend was also apparent for the decade from 1993 to 2002 ( Fig. 2 ). Subgroup analysis was not performed because the numbers of deaths in some subpopulations were small. Of 476 patients with advanced HIV disease observed in 1993 – 1996 and 1997 – 2002 , the baseline characteristics of sex , ethnicity , HIV exposure risk factor , and age were not statistically different , but those in the later period had a lower CD4 count ( P = 0. 018 ). The number of deaths per 1000 patient-months decreased from 9. 2 in the early period to 2. 4 in the later period. The rate ratio was 4. 04 ( 95% CI , 2. 52 – 6. 47 ) .



**Fig. 1.** Prevalence of HAART use in patients with advanced HIV infection.

Table 1 lists the characteristics of 477 patients with advanced HIV disease evaluated for the use of



**Fig. 2.** Crude mortality rate of patients with advanced HIV disease from 1993 to 2002. \* *P* value by Chi-square test for trend. # *P* value by Chi-square test or Fisher's exact test, using 1996 as reference.

	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002
Mil-year population (No.)	54	65	79	96	131	177	233	272	318	383
Total deaths (No.)	13	7	24	12	9	5	7	3	9	3
Crude mortality rate	24.1	10.8	30.4	12.5	6.9	2.8	3.0	1.1	2.8	0.8

— \**P* = 0.004 —  
 ————— \**P* < 0.001 —————  
 ————— \**P* < 0.001 —————

HAART. These were patients who were followed from 1984 to mid-2003 and seen in the clinic more than once. Most of them were heterosexual, Chinese and aged between 30 and 49. Table 2 shows the event rates and unadjusted rate ratios of ( 1 ) death after AIDS, and ( 2 ) AIDS and ( 3 ) death after CD4 cell count < 200/ $\mu$ l ( but without AIDS ), that were stratified by baseline CD4 cell count, viral load and calendar period. Both the CD4 count and calendar period, but not viral load, were statistically associated with the outcome measures. As CD4 count lowered, rates of death and AIDS after a CD4 count < 200/ $\mu$ l increased. However, the rate of death after AIDS increased significantly only if the CD4 count was less than 10/ $\mu$ l. For all 3 end points, the rates decreased significantly in the calendar periods, 1998 – 2000 and 2001–mid 2003, relative to 1994 – 1995. There was a consistent but non-significant decrease in the period 1996 to 1997.

The three end points were also classified according to their occurrence in pre-HAART or HAART patient-years of observation, and a significant decrease was noted for HAART patient-years for all of them ( Table 3 ). The unadjusted rate ratio was smallest for death after AIDS at 0.09 ( 95% CI, 0.05 – 0.16 ). The rate ratios for AIDS and death after a CD4 cell count < 200/ $\mu$ l were 0.13 ( 95% CI, 0.08 – 0.23 ) and 0.20 ( 95% CI, 0.10 – 0.43 ) respectively. Remarkably, in the multivariate analysis where use of HAART, CD4 count, and calendar period were adjusted ( Table 4 ), calendar period ceased to be a significant factor. The effect of HAART remained highly significant as well as that of a low baseline CD4 count. A risk reduction of 79% ( for death after CD4 cell count < 200/ $\mu$ l ) to 92% ( for AIDS after CD4 cell count < 200/ $\mu$ l ) was

**Table 1.** Baseline characteristics of 477 patients evaluated for use of HAART ( 1984 to mid-2003 )

Characteristic ( <i>n</i> = 477 )	Patients, <i>n</i> ( % )
Sex	
Male	400 ( 83.9 )
Female	77 ( 16.1 )
Ethnicity	
Chinese	389 ( 81.6 )
Asian, non-Chinese	56 ( 11.7 )
Caucasian	28 ( 5.9 )
Others	4 ( 0.8 )
Risk factor	
Heterosexual	334 ( 70.0 )
MSM	116 ( 24.3 )
Injecting drug use	8 ( 1.7 )
Others	19 ( 4.0 )
Age at first ADI or CD4 < 200/ $\mu$ l	
≤ 29	96 ( 20.1 )
30-39	208 ( 43.6 )
40-49	115 ( 24.1 )
≥ 50	58 ( 12.2 )

ADI, AIDS-defining illness.

evident with the use of HAART.

## DISCUSSION

Most published studies on the benefits of HAART on AIDS mortality and morbidity in a population setting compared mortality and morbidity figures before and after HAART was available.<sup>13-19</sup> Furthermore, some of these studies were of highly selected populations, making it unclear how generalizable their findings were. Therefore, it is not surprising that the range of reduction in mortality and morbidity in these studies was wide. Whereas there was only 20.7% mortality reduction in a hospital in Brazil,<sup>18</sup> patients in Australia<sup>19</sup> showed a 79% reduction.

**Table 2.** Event rates and rate ratios for death after AIDS , AIDS after a CD4 cell count <200/ $\mu$ l , and death after a CD4 cell count <200/ $\mu$ l and without ADI by baseline CD4 count , baseline HIV RNA level , and calendar period

Stratifying variable	Death after AIDS			AIDS after a CD4 cell count <200/ $\mu$ l and without ADI			Death after a CD4 cell count <200/ $\mu$ l and without ADI		
	Rate ( n/patient-years )	Rate ratio ( * 95% CI )	* P value	Rate ( n/patient-years )	Rate ratio ( * 95% CI )	* P value	Rate ( n/patient-years )	Rate ratio ( * 95% CI )	* P value
Baseline CD4 count			0.091			<0.001			<0.001
100 –199/ $\mu$ l	0.06 ( 4/64.6 )	1.00		0.03 ( 10/311 )	1.00		0.01 ( 4/337.2 )	1.00	
50 –99/ $\mu$ l	0.13 ( 14/109 )	2.07 ( 0.68 –6.30 )		0.18 ( 12/74.1 )	5.04 ( 2.18 –11.65 )		0.08 ( 7/92.8 )	6.36 ( 1.86 –21.71 )	
10 –49/ $\mu$ l	0.13 ( 20/158 )	2.04 ( 0.70 –5.98 )		0.17 ( 25/146.7 )	5.30 ( 2.55 –11.04 )		0.05 ( 10/197.1 )	4.28 ( 1.34 –13.63 )	
<10/ $\mu$ l	0.26 ( 9/34.4 )	4.22 ( 1.30 –13.72 )		0.31 ( 6/19.6 )	9.53 ( 3.47 –26.22 )		0.25 ( 6/24.3 )	20.82 ( 5.88 –73.73 )	
Baseline viral load , copies/ml			0.414			0.084			0.391
<100 000	0.03 ( 5/148 )	1.00		0.05 ( 17/357.1 )	1.00		0.01 ( 5/405.8 )	1.00	
100 000 –499 999	0.07 ( 8/111.3 )	2.13 ( 0.70 –6.50 )		0.09 ( 12/129.1 )	1.95 ( 0.93 –4.09 )		0.02 ( 4/160.1 )	2.03 ( 0.54 –7.55 )	
$\geq$ 500 000	0.05 ( 2/40 )	1.48 ( 0.29 –7.63 )		0.14 ( 3/21.2 )	2.97 ( 0.87 –10.14 )		0.04 ( 1/23.6 )	3.44 ( 0.40 –29.43 )	
Calendar period			<0.001			<0.001			<0.001
1994 –1995	0.49 ( 11/22.4 )	1.00		0.24 ( 9/40.8 )	1.00		0.12 ( 5/43.1 )	1.00	
1996 –1997	0.35 ( 20/57.5 )	0.71 ( 0.34 –1.48 )		0.17 ( 19/110 )	0.78 ( 0.35 –1.73 )		0.09 ( 12/126.3 )	0.82 ( 0.29 –2.32 )	
1998 –2000	0.05 ( 10/194.1 )	0.10 ( 0.04 –0.25 )		0.08 ( 25/328.9 )	0.34 ( 0.16 –0.74 )		0.02 ( 6/386.5 )	0.13 ( 0.04 –0.44 )	
2001 –Jun 2003	0.03 ( 10/303.6 )	0.07 ( 0.03 –0.16 )		0.06 ( 25/416.5 )	0.27 ( 0.13 –0.58 )		0.02 ( 8/507.4 )	0.14 ( 0.04 –0.42 )	

ADI , AIDS-defining illness. \* P value and 95% CI derived with Poisson regression model.

**Table 3.** Event rates and rate ratios for death and AIDS by HAART status

Event and use of HAART	Cases ( n )	Follow-up ( patient-years )	Rate ( cases/ patient-years )	Rate ratio ( * 95% CI )	* P value
Death after AIDS					
HAART	15	478.4	0.03	0.09 ( 0.05 –0.16 )	<0.001
Pre-HAART	36	99.1	0.36	1.00	
AIDS after a CD4 cell count <200/ $\mu$ l and without ADI					
HAART	16	601.8	0.03	0.13 ( 0.07 –0.22 )	<0.001
Pre-HAART	62	294.5	0.21	1.00	
Death after a CD4 cell count <200/ $\mu$ l and without ADI					
HAART	10	744.9	0.01	0.20 ( 0.10 –0.43 )	<0.001
Pre-HAART	21	318.4	0.07	1.00	

ADI , AIDS-defining illness ; \* P value and 95% CI derived with Poisson regression model.

**Table 4.** Multivariate analysis of AIDS and death after adjustment of use of HAART , baseline CD4 cell count and calendar period

	Death after AIDS		AIDS after a CD4 cell count <200/ $\mu$ l		Death after a CD4 cell count <200/ $\mu$ l	
	Adjusted rate ratio ( * 95% CI )	* P value	Adjusted rate ratio ( * 95% CI )	* P value	Adjusted rate ratio ( * 95% CI )	* P value
HAART		<0.001		<0.001		0.009
Pre-HAART	1.00		1.00		1.00	
Post-HAART	0.13 ( 0.05 –0.33 )		0.08 ( 0.04 –0.19 )		0.21 ( 0.07 –0.67 )	
Baseline CD4 count		0.028		<0.001		<0.001
$\geq$ 100/ $\mu$ l	1.00		1.00		1.00	
50 –99/ $\mu$ l	2.12 ( 0.69 –6.45 )		8.09 ( 3.48 –18.86 )		7.74 ( 2.24 –26.71 )	
10 –49/ $\mu$ l	3.02 ( 1.02 –8.89 )		4.89 ( 2.33 –10.23 )		4.18 ( 1.31 –13.39 )	
<10/ $\mu$ l	5.39 ( 1.64 –17.65 )		19.55 ( 6.97 –54.85 )		30.49 ( 8.46 –109.9 )	
Calendar period		0.188		0.499		0.068
1994 –1995	1.00		1.00		1.00	
1996 –1997	1.28 ( 0.61 –2.68 )		0.75 ( 0.33 –1.70 )		0.77 ( 0.26 –2.25 )	
1998 –2000	0.61 ( 0.21 –1.78 )		0.54 ( 0.23 –1.26 )		0.24 ( 0.06 –0.92 )	
2001 –Jun 2003	0.43 ( 0.14 –1.30 )		0.56 ( 0.23 –1.40 )		0.21 ( 0.05 –0.88 )	

\* P value and 95% CI derived with Poisson regression model.

These differences may be explained by variation in study design , recruitment criteria , years analyzed , extent of HAART use and adjunctive medical treatments. A low CD4 cell count was often used as a proxy of advanced disease but it did not fully adjust for the tendency for slow progressors to be more prevalent in later cohorts. More importantly , confounding factors that varied with time were not controlled for. These factors could be related to the change of care delivery , case mix or unmeasured local factors. Methodologic inadequacies of these studies notwithstanding , the remarkable consistency of their findings supported the reasonable hypothesis that use of HAART decreased mortality and morbidity in AIDS at the population level.

Other studies directly measured the effects of HAART. Pallela et al<sup>20</sup> reported that in a group of outpatients with advanced HIV disease , the death rate varied inversely with the intensity of antiretroviral therapy. Compared with PI-containing combination therapy , the relative risk of death was 4.5 ( 95% CI , 3.6 – 6.2 ) in those on no antiretroviral therapy. Messeri et al<sup>21</sup> reported mortality reduction of 50% with the use of HAART in a group of outpatients in New York City. In Taiwan , there was also substantial decline in both mortality and morbidity. The overestimated mortality rate ( lost to follow up counted as death ) decreased from 148.4 per 100 patient-years in 1995 to 7.4 per 100 patient-years in 1999. After adjusting for CD4 cell count , age , gender , and risk behavior , HAART use was associated with 75% risk reduction of death in those with a CD4 cell count <100/ $\mu$ l.<sup>22</sup>

Besides the actual use of HAART , other factors associated with calendar period may be important and need to be controlled for. Detels et al<sup>23</sup> compared the hazards of AIDS and death in those reaching the same duration of infection at different calendar periods. It showed a 60% increase in time to AIDS in the period when HAART was used. A pooled analysis of different cohorts in Europe , whose seroconversion dates were known , showed that the risk of death was reduced by 84% in those seroconverted when HAART was available.<sup>24</sup> However , this was only one factor related to calendar year that was adjusted in this study. To address this , Mocroft et al<sup>25</sup> compared two groups of patients immediately before and after HAART was available and showed that the improved survival became non-significant if use of HAART was

adjusted for. Murphy et al<sup>26</sup> adjusted for calendar year in a clinical trial and showed that HAART reduced mortality by 62%.

In our study we described the overall decreasing trend of the crude mortality rate among those with advanced HIV disease in the decade 1993 to 2002. From 1997 to 2002 , the mortality rate stabilized at very low levels , concomitant with the extensive use of HAART. When compared to 1993 – 1996 , mortality decreased by 75%. Although the introduction of HAART in 1997 represented a major change of HIV disease management in the last decade , many changes other than HAART use could have explained the phenomenon. Therefore , this was only weak evidence that HAART reduced mortality. To strengthen the evidence , we analyzed the actual use of HAART in patients and its relationship to the occurrence of clinical end points including death and new development of AIDS. Although calendar period was associated with clinical end points in univariate analysis , it ceased to be a factor in multivariate analysis , indicating that HAART but not calendar period contributed to the improved prognosis.

Limitations of an observational study will apply to our study. In addition , data prior to July 14 , 1999 were obtained from retrospective chart review in contrast to those after that date which were collected prospectively. This could have biased against HAART by under-recognition of events in the pre-HAART era. Failure to capture adherence and grouping dual nucleoside with nil and monotherapy would likely have resulted in similar bias. All-cause , rather than AIDS-specific , mortality was used. This was because HIV/AIDS as a cause of death was often omitted from the death certificate in Hong Kong , making this an unreliable source of information. Overall , 26 out of the 92 ( 28.3% ) deaths from 1993 to 2002 were classified as unspecified ( unpublished data ). It is also possible that by receiving exclusive referral from the sexually transmitted disease ( STD ) clinic , the Integrated Treatment Centre may have a patient pool with earlier disease than the other clinic. However , this potential bias should have been minimized by studying only those with advanced disease.

Although clinical trials would provide the strongest support of the efficacy of antiretroviral therapy , they are also far removed from the normal clinical settings in which people receive their care.

Furthermore , randomized trials of HAART in patients would be unethical to conduct. The Integrated Treatment Centre is one of only two designated adult HIV clinics in Hong Kong. No drug trial has been performed. Thus , findings of this study could be extrapolated to the outpatient population of advanced HIV disease in Hong Kong. Some characteristics of this clinic-based cohort are also its strengths. Drugs were dispensed in the clinic itself , making it possible to confirm that prescriptions were filled. The overall default rate was low and patients were followed in accordance with uniform medical protocols.<sup>27 28</sup> All viral load and lymphocyte counts were performed by one reference laboratory. The database was also managed by doctors who personally attended most of the patients.

In 2001 , the World Health Organization launched the three-by-five initiative , aiming at providing three million people in developing countries with HAART by 2005.<sup>29</sup> In this respect , a recently completed pilot trial of HAART in Mainland China yielded satisfactory virologic and immunologic outcomes.<sup>30</sup> Our favorable experience of HAART in Hong Kong provided further evidence that the benefits of HAART were not restricted to industrialized countries. In conclusion , the mortality and morbidity of advanced HIV disease have declined in Hong Kong. This improved prognosis was attributable to the use of HAART.

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## Medical news

### Traditional Chinese medicinal capsule for curing HIV/AIDS undergoes clinical test

A traditional Chinese medicinal capsule capable of curing HIV/AIDS has been put under clinical test with the approval of the State Food and Drug Administration.

Chen Dagang , inventor of the capsule and leader in charge of the clinical test , said the clinical test had been carried out in a number of medical establishments in Beijing , including You 'an Hospital , the No. 302 Hospital of the Chinese People 's Liberation Army , Peking Union Medical College Hospital and Ditan Hospital since July 7 , and the test would be completed in nine months.

Pre-stage research in the past five years proves that the capsule , made from 16 kinds of medicinal herbs , is capable of restraining HIV integration enzyme from outside the human body and curbing the virus from duplication inside the infected cell , according to Chen.

Integration enzyme is the third essential element for HIV duplication , or in other words , HIV without integration enzyme is not infectious , said Chen , adding that his capsule , like any other synthetic drugs , had to overcome drug-resistant substance occurring in the process of usage as of the first-generation anti-retrovirus medicine , which might gradually lead to a weakened curing effect.

( Source : xinhuanet )