

Hong Kong Poison Information Centre: Annual Report 2006

香港中毒諮詢中心年報：2006年

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Objective: To report the poisoning data of Hong Kong Poison Information Centre (HKPIC) in 2006. **Methods:** From 1st January 2006 to 31st December 2006, all poisoning cases received by HKPIC were retrieved from its database (DATOX) for analysis. **Results:** A total of 2555 poisoned cases were analysed. There were 1051 male and 1466 female patients and nearly 60% of the cases were between 20 and 49 years old. Common causes of exposure were suicidal attempts and accidents. Paracetamol, sedative-hypnotic and household products were common sources of poison exposure. The majority of the patients were managed conservatively, with 18.8% and 10.5% treated by decontamination and antidotes respectively. Most cases had uneventful recovery; less than 1% of the poison exposure resulted in death and about 5% of the exposure had major outcomes. Nearly half of the cases were managed in the accident & emergency department without hospitalisation. **Conclusions:** This annual report provides updated epidemiological information on poisoning in Hong Kong. Subsequent annual reports would provide important information on the trend of poisoning pattern and may guide further strategies in poison control and prevention in Hong Kong. (*Hong Kong j. emerg.med.* 2008;15:240-253)

目的：報告2006年香港中毒諮詢中心的中毒數據。**方法：**從數據庫找出及分析香港中毒諮詢中心從2006年1月1日至2006年12月31日期間接收的所有中毒個案。**結果：**共分析2555個中毒個案，1051名男病者及1466名女病者。接近60%個案年齡為20至49歲之間。常見的中毒原因為企圖自殺及意外。撲熱息痛、鎮靜-安眠藥及家居用品是常見的中毒源頭。大部份病者以保守方法處理，18.8%及10.5%分別以除污及解毒劑治理。大部份個案順利復原，少於1%的中毒引致死亡，而大約5%的中毒有重要後果。接近一半的個案是在急症室處理，沒有住院。**結論：**這年報提供更新的香港中毒流行病學資料。日後的年報會提供中毒模式趨勢的重要資訊，及可引導香港中毒控制及預防的進一步策略。

Keywords: Epidemiology, poisoning

關鍵詞：流行病學、中毒

Introduction

The Hong Kong Poison Information Centre (HKPIC) was established in July 2005.¹ Its main functions include the provision of poison information and

toxicology management advice to health care professionals in Hong Kong. Moreover, it also serves to collect poisoning data for toxico-vigilance, training and poisoning prevention. As the data collected over the initial run-in period in 2005 were not representative, the full 2006 year data were reported here as our inaugural annual report.

Although the HKPIC takes call from all doctors, nurses and pharmacists in Hong Kong, there may be consultations from outside Hong Kong including Macau, China or outside Asia in the form of emails or phone calls. For the purpose of our annual report, we

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would only include those poisoning cases originated from Hong Kong. As the annual report aimed to document all poisoning cases, we also excluded cases of enquiries for poisoning information without a real patient. Furthermore, we also captured all the poisoning cases reported from six major accident & emergency departments (AED), namely Queen Elizabeth Hospital, Queen Mary Hospital, Pamela Youde Nethersole Eastern Hospital, Prince of Wales Hospital, Tuen Mun Hospital and United Christian Hospital. As these AEDs receive about half of the total emergency attendance yearly, they represent a reliable source for monitoring the poisoning situation in Hong Kong.

Methods

From 1st January 2006 to 31st December 2006, all poisoning cases received by HKPIC were retrieved from its database (DATOX) for analysis.

The poison information officers of HKPIC, most being emergency medicine fellows or higher trainees, upon receiving a case would input data to the DATOX based on information from the consulting health care

professional, AED record, data obtained from the electronic patient record (ePR) of the Hospital Authority (HA) and other relevant sources. Data collected in DATOX (Appendix 1) included patient demographic data, poison data (poison type and dose, route, time, place and reason of exposure), clinical data (clinical features, investigation results), management data (use of decontamination, antidotes and other specific treatment), and outcome data (disposal for AED patient, final outcome and its relationship to the poison exposure).

Senior staffs from the HKPIC, all being emergency medicine specialists with post-graduation training in clinical toxicology, would classify the outcome of the cases into five categories: no effect, mild effect, moderate effect, major effect or death with reference to the American Association of Poison Control Centers' National Poison Data System.² (Table 1) Besides, the relationship between the poison exposure and clinical outcome are graded as definite, probable, possible, not related or undetermined according to the available information. All death or severe adverse effect cases are further reviewed by a second senior.

Table 1. Definition of clinical outcome

No effect	The patient did not develop any signs or symptoms
Mild effect	The patient developed some signs or symptoms that were minimally bothersome and generally resolved rapidly with no residual disability or disfigurement. Examples are self-limited gastrointestinal symptoms, drowsiness, skin irritation and sinus tachycardia without hypotension.
Moderate effect	The patient exhibited signs or symptoms that were more pronounced, more prolonged, or more systemic in nature than mild effect. Usually, some form of treatment was indicated. Symptoms were not life-threatening, and the patient had no residual disability or disfigurement. Examples are hypotension that is rapidly responsive to treatment, and isolated brief seizures that respond readily to treatment.
Major effect	The patient exhibited signs or symptoms that were life-threatening or resulted in significant residual disability or disfigurement. Examples are repeated seizures or status epilepticus, respiratory compromise requiring intubation, unstable arrhythmias and refractory hypotension.
Death	The patient died
Unknown effect	The clinical outcome was unknown from the available information

For this annual report, case is defined as a poisoning incident of a patient at one occasion. HKPIC may receive multiple consultations from different specialties for the same poisoning incident. For example, an emergency physician consults for the acute management advice on organophosphate poisoning and then the intensivist of the same patient seeks advice on the features of the immediate syndrome of organophosphate poisoning days afterwards. This would generate two DATOX records and for the analysis in this report or the subsequent annual reports, the two DATOX records would be counted as one case only.

Results

In 2006, there were 2671 DATOX records in which 79% were reports, 18% were consultations and 3% were information enquiries which were general poison information requests by health care professionals without patient being involved. From the reason mentioned above, the DATOX generated 2555 poisoned cases for the subsequent analysis.

The age and gender distribution of the cases is outlined in Figure 1. There were 1051 male (41.1%) and 1466 female (57.4%) patients included. A male

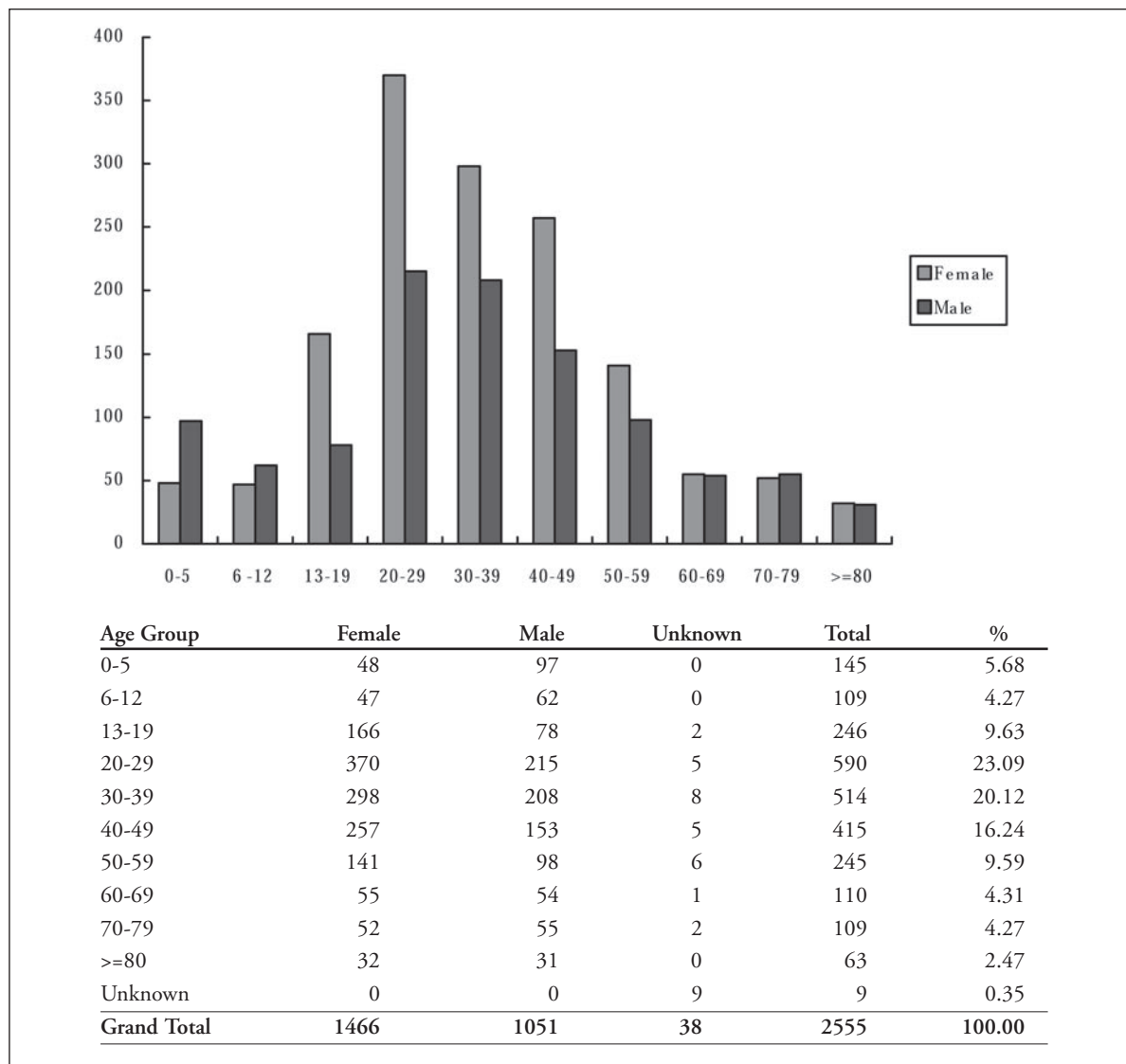


Figure 1. Age and gender distribution.

predominance was found among cases involving children younger than 13 years, but this gender distribution was reversed in teenagers and adults, with women comprising the majority of cases. With increasing age to 60 years onwards, similar numbers of men and women were involved. Concerning age distribution, the 20-29 years group was the commonest (23%) and nearly 60% of the cases were between 20 and 49 years old. Children younger than 6 years were involved in 6% of cases.

The reason of poisoning is shown in Figure 2 with the commonest cause (40.1%) being suicidal attempt. Another 13.7% of the cases were secondary to accidents and 8.5% of the cases were related to the use of abusive drugs. Adverse drug reaction and therapeutic errors contributed 6.9% and 3.6% of the cases respectively.

As shown in Figures 3 & 4, the commonest place of exposure was in the patient's home (45%). The commonest route of exposure was ingestion (87.1%), followed by inhalation (4.6%) and dermal exposure (2.1%).

Notably, 68% of the cases were exposed to a single poison while the remaining 32% of the cases were exposed to multiple poisons. The type of poison exposed is shown in Figure 5. The three main categories: pharmaceutical, non-pharmaceutical and Chinese & alternative medicine contributed to 62.9%, 30.7% and 6.4% of the poisons exposed respectively. Apart from food poisoning, the three commonest types of poison exposed were non-benzodiazepine sedative-hypnotics (7.3%), paracetamol (7.1%) and household products (7.0%).

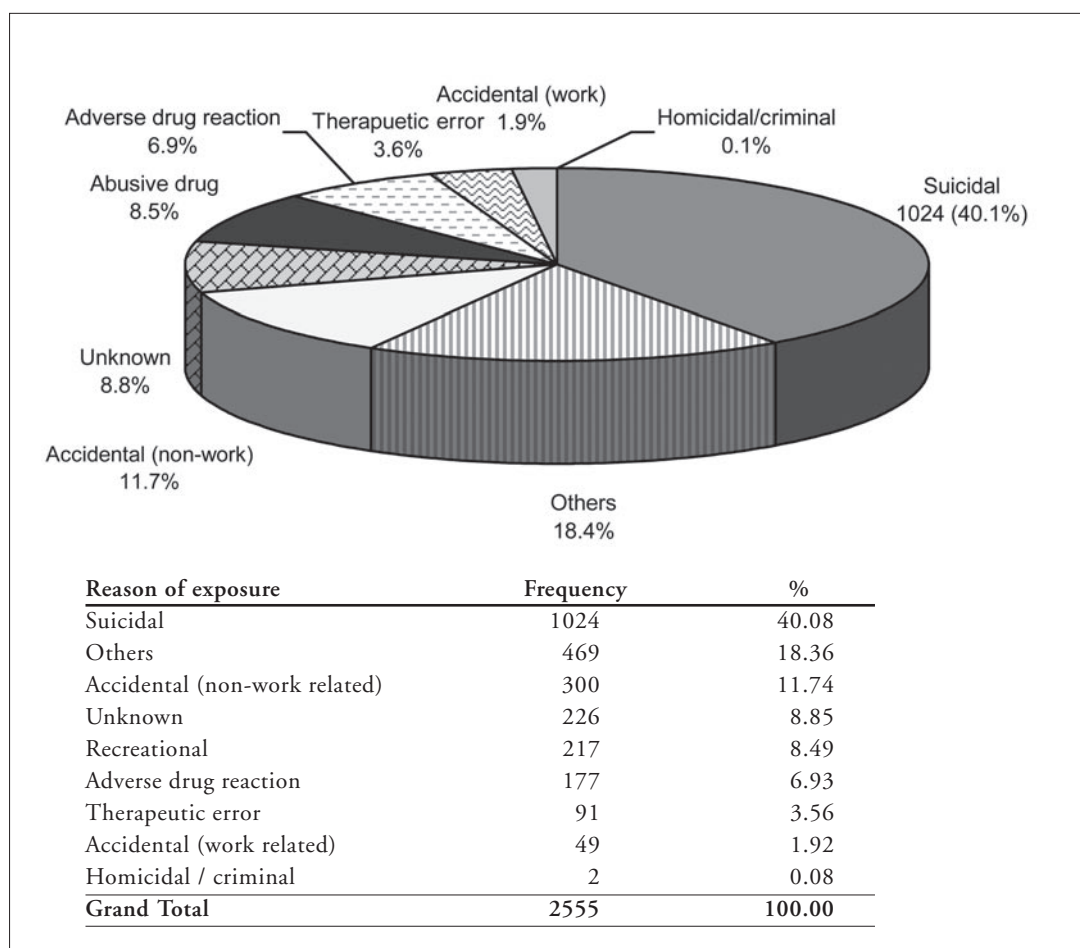


Figure 2. Reason of exposure.

Only 480 (18.8%) and 269 (10.5%) cases were treated by decontamination and antidotes respectively. For those 480 patients undergoing decontamination, the commonest method was oral administration of a single dose of activated charcoal (81.8%). Gastric lavage (12.8%) and other methods of decontamination including multiple doses activated charcoal, whole bowel irrigation and the use of cathartic without charcoal were performed infrequently, as shown in Figure 6. For those 269 patients who were given

antidotes, the five commonest ones were N-acetylcysteine (29.6%), naloxone (20.0%), calcium (13.0%), sodium bicarbonate (12.4%) and atropine (6.2%) respectively, as listed in Table 2.

The clinical outcome of the cases is shown in Table 3, 115 out of the total 2555 cases were graded as not related to the poison exposure and were not listed in these figures. For the remaining 2440 cases, there were 23 deaths (0.9%), 118 cases of major effects (4.8%),

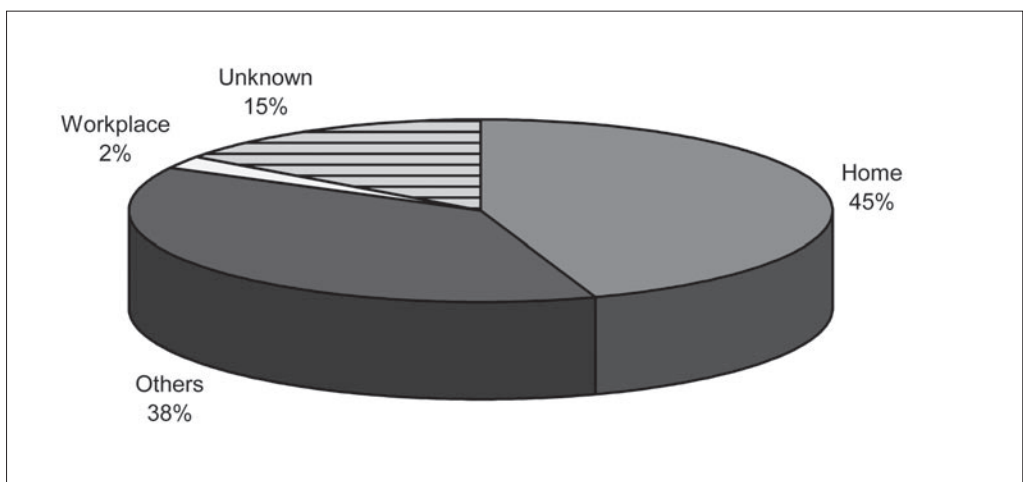


Figure 3. Place of exposure.

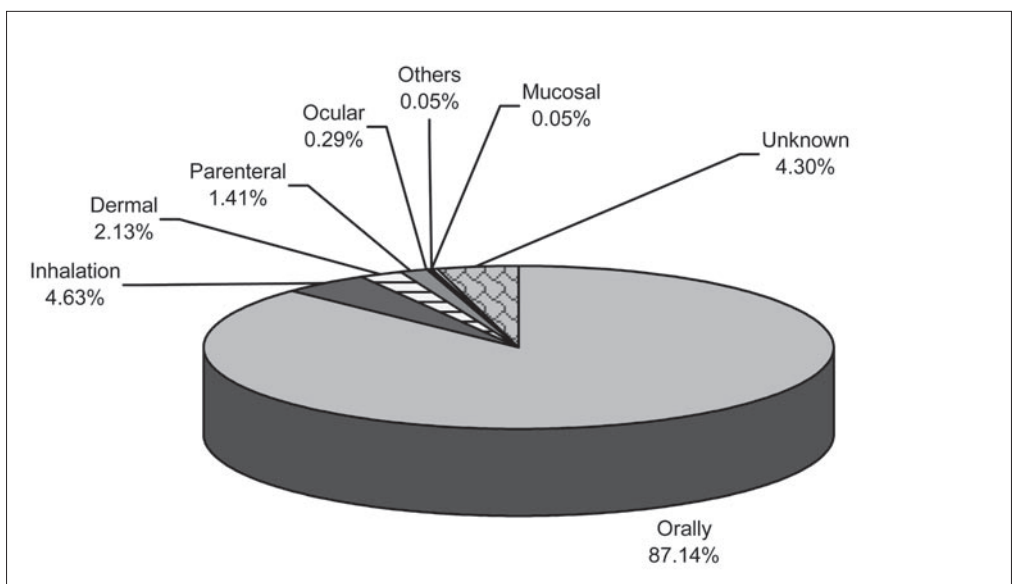


Figure 4. Route of exposure.

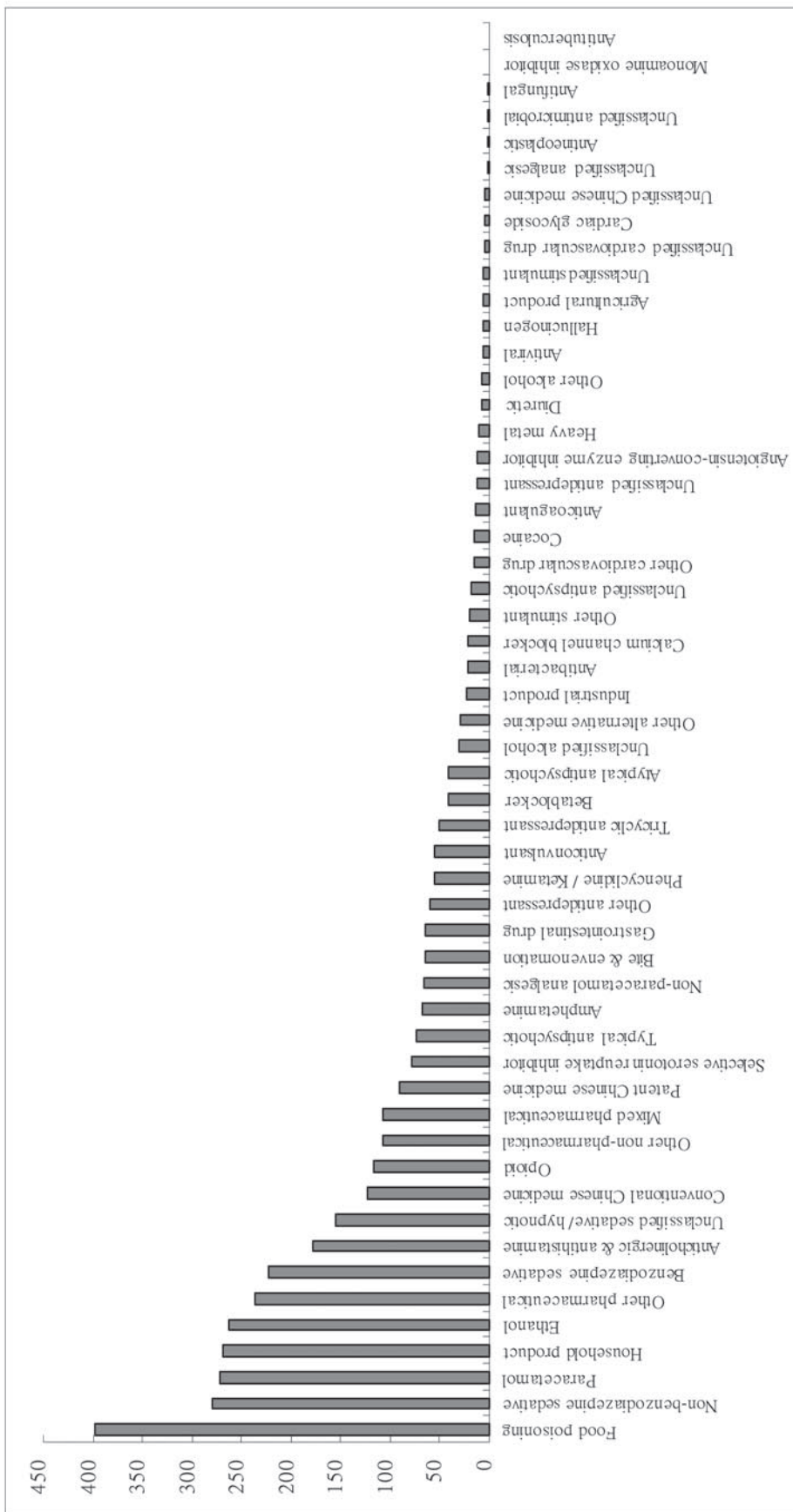


Figure 5. Type of poison exposed.

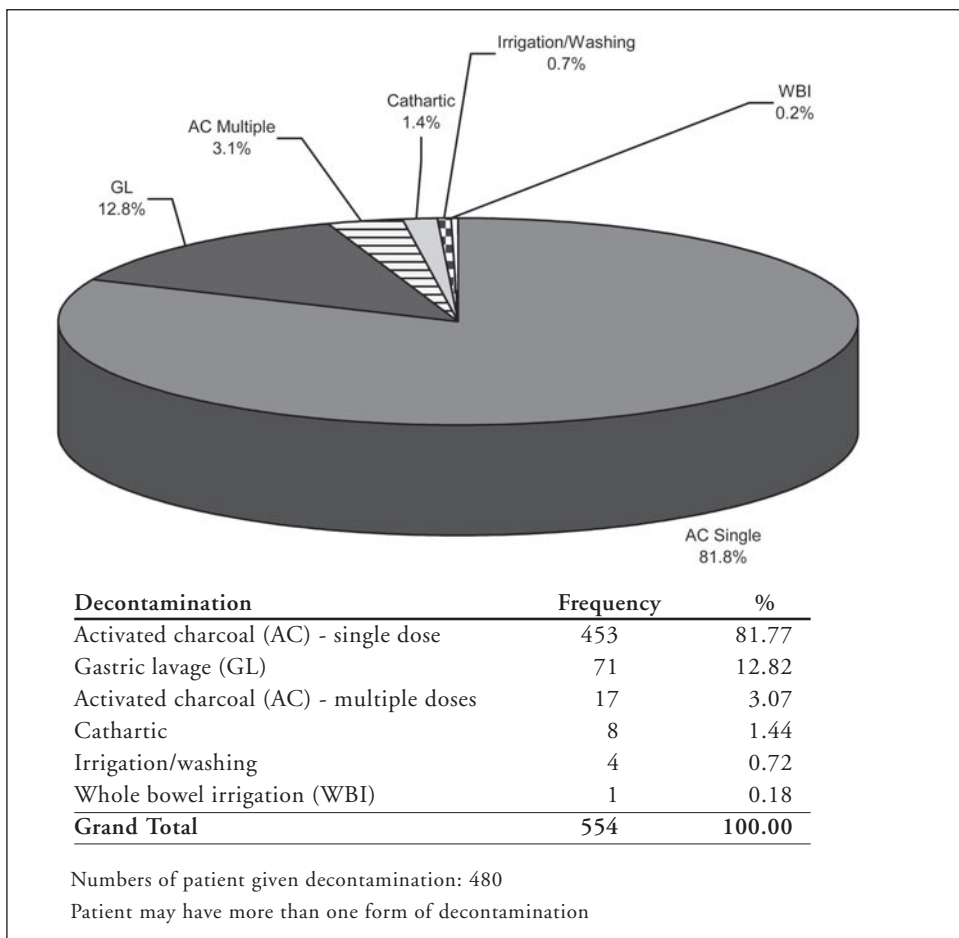


Figure 6. Decontamination.

333 cases of moderate effects (13.6%), 1508 cases of mild effects (61.8%), 344 cases of no effect (14.1%) and 114 cases of unknown effect (4.7%). Similarly, 64.8%, 17.5% and 6.8% of the outcomes were graded as definitely, probably and possibly related to the poison exposure respectively while the remaining 10.9% were undetermined from the available information. For the seven deaths which were definitely related to the poison exposed, there were two cases of carbon monoxide poisoning from burning charcoal, one case of cardiac active steroid poisoning from Chinese herbal medicine, one case of caustic injury from drain opener, and the remaining three cases were insecticide, warfarin, and dologesic poisoning.

Totally 2316 cases (90.6% of the cases) were patients from the AED, the disposal of those patients is listed in Table 4. Nearly half of the AED patients (48.8%)

Table 2. Antidotes use

Antidote	Frequency	%
N-acetylcysteine	105	29.58
Naloxone	71	20.00
Calcium	46	12.96
Sodium bicarbonate	44	12.39
Atropine	22	6.20
Benzodiazepines	12	3.38
Thiamine	9	2.54
Dextrose	9	2.54
Flumazenil	8	2.25
Antivenin/antitoxin	7	1.97
Vitamin K	5	1.41
Physostigmine	4	1.13
Glucagon	4	1.13
Pralidoxime	4	1.13
Methylene blue	2	0.56
Insulin	2	0.56
Digoxin Fab	1	0.28
Total	355	100.00

Number of patient given antidote: 269
 Patient may have more than one antidote

Table 3. Clinical outcome

Outcome	Definite	Probable	Possible Frequency (%)	Undetermined	Total
Death	7 (0.3)	6 (0.2)	10 (0.4)	0 (0)	23 (0.9)
Major effect	97 (4.0)	14 (0.6)	7 (0.3)	0 (0)	118 (4.8)
Moderate effect	205 (8.4)	64 (2.6)	37 (1.5)	27 (1.1)	333 (13.6)
Mild effect	995 (40.8)	310 (12.7)	97 (4.0)	106 (4.3)	1508 (61.8)
No effect	275 (11.3)	26 (1.1)	14 (0.6)	29 (1.2)	344 (14.1)
Unknown	2 (0.1)	7 (0.3)	2 (0.1)	103 (4.2)	114 (4.7)
Total	1581 (64.8)	427 (17.5)	167 (6.8)	265 (10.9)	2440 (100)

Table 4. Disposal of cases from accident & emergency departments (AED)

Disposal (Limited to AED cases only)	Frequency	%
Admission to general ward	909	39.25
Discharge after a period of observation	594	25.65
Discharge directly after AED consultation	408	17.62
Discharge against medical advice	128	5.53
Admission to intensive/critical care unit	127	5.48
Admission to psychiatric ward	106	4.58
Unknown	35	1.51
Disappeared	9	0.39
Total	2316	100.00

were managed without hospitalisation and another 4.6% of the patients were admitted to psychiatric units either directly or after a period of management in the AED; 39.3% and 5.5% of the cases required general medical and intensive care unit in-hospital management respectively.

Discussion

There were scanty epidemiological studies of poisoning in the past which provided limited data on the overall poisoning pattern in Hong Kong.³⁻⁵ With the establishment of the HKPIC in July 2005, one of its functions is to facilitate the collection and analysis of poisoning data. This first annual report of the HKPIC presented the data of more than 2500 poisoned cases

in 2006 which is the largest epidemiological study so far in Hong Kong.

The age and gender distribution, the reasons, as well as routes of exposure are expected and compatible with previous local³ and overseas data.^{2,6} Apart from food poisoning, of which most of them are presumed to be infective in origin, the three commonest types of poison exposed are non-benzodiazepine sedative-hypnotics (7.3%), paracetamol (7.1%) and household products (7.0%). The non-benzodiazepine sedative-hypnotics include mainly the Z-hypnotics (zopiclone and zolpidem), melatonin and other over-the-counter sleeping pills with herbal ingredients. One of the reasons for sleeping pill exposure was the easy availability of this group of drugs with or without prescription and the belief that it was an effective

method of committing suicide. Paracetamol was the next commonest poison involved, as expected from its extremely common and ready availability. Household products, again was readily available for poisoning by intentional exposure from suicidal attempt as well as accidental exposure.

Notably, 18.8% of our patients were treated with decontamination which was less than the reported figure of 40% in an epidemiologic study of AED patients in 2001.⁴ This reduction in decontamination is compatible with the situation in the United States as reflected by the decrease in activated charcoal administration from 7.1% in 1997 to 4.6% in 2006.³

Concerning the clinical outcome, the majority of cases (>75%) are classified as no effect or mild effect. On the other hand, 23 (0.9%) patients died at least possibly related to the poison exposure. The death rate is compatible with the previous figure of 1.4%.² More than 90% of the poisoned cases required hospital admission in 2001⁴ as compared with nearly half of the AED patients (48.8%) were managed without hospitalisation in this current study. Multiple reasons might contribute to this dramatic difference including improved toxicology knowledge among emergency physicians, development of toxicology team in several AEDs, optimal use of observational facilities in AEDs and the cooperation of other clinical units such as psychiatric support.

There are several limitations of this report. First of all, the majority of our cases were from voluntary reporting and were limited to six major AEDs only. This might not totally reflect the whole poisoning pattern in Hong Kong although significant inter-district difference in poisoning pattern was not expected in such a small city and the six AEDs had already been selected from different territories. In order to have better surveillance, we shall extend the reporting to all HA AEDs in the near future. On the other hand, we are expecting an increasing number of consultations from health care professionals in both the private and public health systems. The consultation cases shall contribute an

increasing proportion of our DATOX and more comprehensive poisoning data are expected in the subsequent HKPIC annual report. Besides, the classification of poison type in DATOX was less than perfect: not uncommonly we found difficulty and discrepancy in classifying the type of poison. Moreover, the judgement on clinical outcome and its relationship to the poison exposure may be suboptimal in some cases especially if the available information provided is limited or unreliable. However, we have tried our best to be objective and consistent in the analysis.

Hopefully, this annual report serves as a reference for subsequent comparison of poisoning data in Hong Kong which may provide guidance and direction for future strategies in better poison control and prevention in Hong Kong.

Conclusion

This annual report provides updated epidemiological information on poisoning in Hong Kong. Readily available pharmaceuticals such as paracetamol and sedative-hypnotics and household products were common sources of poison exposure. The majority of the patients were managed conservatively, with 18.8% and 10.5% of them treated specifically by decontamination and antidotes respectively. Most cases had uneventful recovery; less than 1% of the poison exposure resulted in death and about 5% of the exposure had major outcomes. Subsequent annual reports would provide important information on the trend of poisoning pattern in Hong Kong and may guide further strategies in poison control and prevention.

Interesting cases

Case 1

A 9-month-old Pakistani baby was investigated in a paediatric unit for microcephaly, and small anterior fontanelle. Her blood lead level at five months old was 1.29 $\mu\text{mol/L}$ (reference range: 0.2-0.47 $\mu\text{mol/L}$).

A brief course of chelation therapy had been given but no exposure source could be identified initially by repeated history taking. Home visit by staffs from the Department of Health identified a powder-form paint that had been applied around the patient's eyes for cosmetic reason since infancy as a possible source. It is a common practice in South Asia and this kind of products are called Surma or Kohl. That specific Surma was bought from a grocery store selling South Asian native goods and was confirmed to contain 84,000 ppm of lead, well exceeding the legal standard of 40 ppm for cosmetics. The child's blood lead level returned to normal after stop using the Surma.

Case 2

A 44-year-old woman developed convulsion, coma, and hypotension within two hours after overdosing herself with amitriptyline – a tricyclic antidepressant. Electrocardiogram showed widened QRS interval of at least 200 msec which rapidly degenerated into ventricular tachycardia with weak pulse. The arrhythmia responded to single electro-cardioversion. Five intravenous boluses of 50 ml 8.4% sodium bicarbonate given immediately after cardioversion narrowed the QRS interval down to 148 msec with no further recurrence of ventricular arrhythmia or convulsion. Other treatment given included intubation and mechanical hyperventilation, intravenous (IV) diazepam and amiodarone, and gastric lavage followed by 50 gm activated charcoal via a large-bore orogastric tube. Five additional doses of 50 ml 8.4% sodium bicarbonate were given by rapid IV infusion after the initial stabilisation due to unsatisfactory QRS normalisation and hypotension. A total of 500 ml 8.4% sodium bicarbonate was given to this patient in the first six hours of her presentation as the antidote for tricyclic antidepressant or sodium-channel blocker poisoning. The patient recovered rapidly and could be extubated and discharged from the intensive care unit in 24 hours. No residue disability was observed when the patient was discharged after four days of hospitalisation.

Case 3

A 52-year-old man who was labelled with ampicillin allergy underwent general anaesthesia for elective

thyroidectomy. His allergy history dated back to half-year ago when he developed hypotension and wheezing during general anaesthesia for a hand injury when ampicillin was given in addition to anaesthetic agents. Shortly after induction in this second operation, he developed skin rash and wheezing followed by sudden cardiac arrest with ventricular tachycardia and ventricular fibrillation. He was successfully resuscitated. The HKPIC was consulted in the subsequent investigation of the case. The anaesthetic agents involved were propofol, fentanyl and atracurium and the same three agents had been administered in the operation half-year ago. Anaesthetic agent was identified as the commonest pharmaceutical class causing death by anaphylaxis in a death certificate study in the United Kingdom. It accounted for 42% of all iatrogenic fatal anaphylaxis comparing with the 25% by antibiotics that came second.⁷ General anaesthesia carried a quoted 1 in 4500 to 25,000 rate of anaphylaxis with a mortality of 3.4%, and the commonest causes in descending order were muscle relaxants, rubber latex, antibiotics, volume expanders and other drugs.⁸ Intradermal test performed six weeks later confirmed atracurium allergy in the patient and showed negative result on other drugs involved including ampicillin.

Case 4

A 72-year-old man living on Lantau Island was bitten on his hand by a snake near his home at around midnight. He complained of abdominal pain and nausea, followed by brief syncope in minutes. Breathing difficulty developed in about an hour followed by progressive generalised paralysis. At about 24 hours, he became apparently "brain-dead" with loss of all brain stem reflexes. Anti-venom was not given because of the wrong identification of the offending snake to a non-venomous species. He showed signs of recovery after one week of mechanical ventilation and regained consciousness at day 10. He suffered from generalised muscle wasting and weakness requiring tracheostomy, assisted ventilation and prolonged rehabilitation. Biologists from the Agriculture, Fishery and Conservation Department visited the patient at four weeks and the patient identified from colour pictures shown to him the many-banded krait

(*Bungarus multicinctus*) as the offending snake. Many-banded krait is native to Hong Kong and its venom is highly neurotoxic. It rarely bites in daytime but becomes offensive at night. Unprovoked bites from kraits at night time have been well reported in South East Asia causing significant mortalities.^{9,10}

Case 5

A 12-month-old baby boy was rushed to hospital for turning blue. He was found to have obvious central and peripheral cyanosis together with mild tachypnoea, otherwise he was stable despite a pulse oximetry reading of SpO₂ 82 to 91%. He had been playing with a comic figure toy-stamp and had licked on it and stamped himself patches of blue over the face and upper limbs. His methaemoglobin level was 38.6%. Intravenous methylene blue 20 mg was given with rapid resolution of cyanosis and dropping of the methaemoglobin level to 1%. He was observed for three days without recurrence of methaemoglobinaemia. Subsequent analysis of toy-stamps from the same source confirmed the presence of aniline dye that has been a well known cause of methaemoglobinaemia and children are more susceptible.

Case 6

A 34-year-old woman presented with the nephritic syndrome and found to have membranous glomerulonephritis with some components of IgA nephropathy on renal biopsy. Her blood mercury was more than three times the upper limit of normal (ULN) and her 24 hour urine mercury was about 17 times of the ULN. Inorganic mercury poisoning was diagnosed and she was treated with a course of succimer, an oral heavy metal chelator. Further history revealed her use of three types of beauty facial cream given to her by a friend in Shenzhen for a few months that all were subsequently found to contain mercury, one of them up to a sky-high 74,810 ppm. Public warning was released by the Department of Health concerning the beauty cream.¹¹

Cases 7 - 10

Four barge sailors shared a self-prepared soup with a puffer fish caught in Hong Kong waters. They developed vomiting, numbness of tongue and face,

dizziness and throat discomfort in 15 minutes. They presented seven hours later for increasing dizziness and weakness. Two of them with obvious bulbar, respiratory weakness and limb weakness were intubated and mechanically ventilated for about 24 hours. One was discharged well on day 3 while the other suffered some residue lower limb weakness that required assistance in walking on his discharge on day 4. The other two less serious patients suffered only mild limb weakness and were observed with serial bed-side spirometry. They recovered rapidly and were discharged after 24 hours. The patients gave a history of consuming puffer-fish caught in their hometown of Southern China without sequelae. It may be explained by the variations in tetrodotoxin concentration among puffer fishes caught in different locations¹² and in different months of the year even in the same location.¹³

Case 11

A 46-year-old man fell acutely sick with severe dizziness and abdominal pain with nausea within half an hour after consuming Chinese herbal medicine for the carcinoma of his oesophagus. He rapidly deteriorated and had cardiac arrest not responding to resuscitation in the accident and emergency department. Subsequent investigation found that Chan-su (toad venom) was inadvertently dispensed to him instead of charred toad skin written on the Chinese medicine formula prescribed in a hospital in Mainland China. The recommended dose of Chan-su is less than one-tenth of the latter. It resulted in a huge overdose of cardioactive steroids and hence the rapid fatality of the patient.

Case 12

A 20-year-old Columbian who was under police custody was admitted into hospital for the treatment of cocaine body packing. Whole bowel irrigation was administered that resulted in the passage of 45 packs of cocaine. After that he was discharged back to police custody. He was readmitted 10 hours later for anxiety state, dilated pupils, mild systolic hypertension and the passing of a broken pack. Ethylene glycol solution was given orally again with the passage of 3 more packs of cocaine in the next three days. Medical clearance after treatment for body packing by drug-trafficker is

difficult. Contrast enhanced abdominal radiology and computed tomography may be useful but is not 100% sensitive.¹⁴

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Appendix 1.

**Hong Kong Poison Information Centre
DATOX Entry Form**

Serial no: 0_ - _ - _ - _ -
Date/Time : _/_ _ : _: _

Consultation Reporting Information Enquiry
Patient Information (or use HA patient label) AED Non-AED

Name _____ (M/F, Age ____) ID no _____

Hospital/Ward/Bed _____ Body wt _____ (Kg/lb)

Phone no 1. _____ Occupation _____

Patient Label
(If appropriate)

Caller Information

Name _____ Rank (Doctor/Other Medical Profession/Non-medical personnel)

Hospital/Dept/Ward _____ Phone no 1. _____ Phone no 2. _____

Poison Information (Please in appropriate box and fill in the table)

Place of exposure : Home Workplaces Others
Reason of exposure : Accidental (work related) Accidental (non-work related) Suicidal Homicidal
 Recreational Therapeutic Error Adverse drug reaction Others

Poison	Name	Category Code (Appendix 1)	Dose	Route	Time of Exposure
1					
2					
3					
4					
5					

(Use extra paper if more than 5 entries)

Clinical Information (Please in appropriate box)

Vitals Signs : GCS/AVPU _____ BP _____ P _____ RR _____ SpO2 _____

CARDIOVASCULAR	GI	Neurological	Respiratory
<input type="checkbox"/> Arrest	<input type="checkbox"/> Abdominal Pain	<input type="checkbox"/> Agitated	<input type="checkbox"/> Bronchospasm
<input type="checkbox"/> Bradycardia	<input type="checkbox"/> Bowel Sound Decreased	<input type="checkbox"/> Ataxia	<input type="checkbox"/> Cyanosis
<input type="checkbox"/> Chest Pain	<input type="checkbox"/> Bowel Sound Increased	<input type="checkbox"/> Coma (GCS <8/15)	<input type="checkbox"/> Hyperventilation
<input type="checkbox"/> Hypertension	<input type="checkbox"/> Burn in esophagus or downwards	<input type="checkbox"/> Confusion	<input type="checkbox"/> Pulmonary edema
<input type="checkbox"/> Hypotension	<input type="checkbox"/> Constipation	<input type="checkbox"/> Dizziness	<input type="checkbox"/> Pneumonitis
<input type="checkbox"/> Palpitation	<input type="checkbox"/> Diarrhea	<input type="checkbox"/> Drowsiness (GCS >= 8/15)	<input type="checkbox"/> Respiratory depression
<input type="checkbox"/> Tachycardia	<input type="checkbox"/> Haematemesis /Melenna	<input type="checkbox"/> Dystonia	<input type="checkbox"/> Respiratory arrest
<input type="checkbox"/> Syncope	<input type="checkbox"/> Nausea	<input type="checkbox"/> Fasciculation	<input type="checkbox"/> SOB
<input type="checkbox"/> Other _____	<input type="checkbox"/> Oral burn	<input type="checkbox"/> Hallucination	<input type="checkbox"/> Other _____
	<input type="checkbox"/> Vomiting =< 4 times	<input type="checkbox"/> Headache	
	<input type="checkbox"/> Vomiting > 4 times	<input type="checkbox"/> Muscle rigidity	
	<input type="checkbox"/> Other _____	<input type="checkbox"/> Numbness	
		<input type="checkbox"/> Weakness	
		<input type="checkbox"/> Paralysis	
		<input type="checkbox"/> Seizure	
		<input type="checkbox"/> Seizure (Status)	
		<input type="checkbox"/> Tinnitus	
		<input type="checkbox"/> Tremor	
		<input type="checkbox"/> Other _____	
SKIN	EYE	RENAL	MISCELLANEOUS
<input type="checkbox"/> Sweating	<input type="checkbox"/> Miosis	<input type="checkbox"/> Acute renal failure	<input type="checkbox"/> Hyperthermia
<input type="checkbox"/> Dry	<input type="checkbox"/> Mydriasis	<input type="checkbox"/> Oxalate in urine	<input type="checkbox"/> Hypothermia
<input type="checkbox"/> Normal	<input type="checkbox"/> Non-reactive pupils	<input type="checkbox"/> Myoglobinuria	<input type="checkbox"/> Excessive secretion
<input type="checkbox"/> Other _____	<input type="checkbox"/> Nystamus	<input type="checkbox"/> Haematuria	<input type="checkbox"/> Other _____
	<input type="checkbox"/> Corneal Injuries	<input type="checkbox"/> Other _____	
	<input type="checkbox"/> Other _____		

(continued on page 253)

Appendix 1. (cont'd)

BLOOD TEST	TOXICOLOGY SPECIMEN	ECG	CXR
<input type="checkbox"/> Hypoglycaemia	<input type="checkbox"/> Gastric Aspirate	<input type="checkbox"/> Sinus Tachycardia	<input type="checkbox"/> Pneumothroax
<input type="checkbox"/> Hyperglycaemia	<input type="checkbox"/> Clotted Blood	<input type="checkbox"/> Sinus Bradycardia	<input type="checkbox"/> Pneumomediastinum
	<input type="checkbox"/> Urine	<input type="checkbox"/> Terminal 40ms sign	<input type="checkbox"/> Pulmonary edema
<input type="checkbox"/> WBC >15	<input type="checkbox"/> Other	<input type="checkbox"/> QRS > 100ms	<input type="checkbox"/> Aspiration
<input type="checkbox"/> Thrombocytopenia		<input type="checkbox"/> QRS > 120ms	<input type="checkbox"/> Other
<input type="checkbox"/> Agranulocytosis	Sent to Lab	<input type="checkbox"/> QRS > 160ms	
<input type="checkbox"/> DIC		<input type="checkbox"/> QTc > 440ms	
		<input type="checkbox"/> QTc > 500ms	
<input type="checkbox"/> High anion metabolic acidosis		<input type="checkbox"/> SVT	AXR
<input type="checkbox"/> Respiratory acidosis	DRUG LEVEL	<input type="checkbox"/> AF	
<input type="checkbox"/> Respiratory alkalosis		<input type="checkbox"/> Ventricular arrhythmia	<input type="checkbox"/> Radioopaque drugs +ve
	<input type="checkbox"/> Paracetamol	<input type="checkbox"/> VEB	<input type="checkbox"/> Ileus
<input type="checkbox"/> AST/ALT > 1000 IU		<input type="checkbox"/> ST scooping	<input type="checkbox"/> Other
<input type="checkbox"/> Lactate > 2 mmol/l	<input type="checkbox"/> ASA	<input type="checkbox"/> 2 nd or 3 rd degree heart block	
<input type="checkbox"/> CPK > 1000		<input type="checkbox"/> Other	
<input type="checkbox"/> Cr increased 2X from baseline	<input type="checkbox"/> Ethanol		
<input type="checkbox"/> Hyponatremia	<input type="checkbox"/> Others		
<input type="checkbox"/> Hypernatremia		URINE	CT BRAIN
<input type="checkbox"/> Hypokalemia		<input type="checkbox"/> Fluorescence +ve	<input type="checkbox"/> Infarct
<input type="checkbox"/> Hyperkalemia		<input type="checkbox"/> Ketone +ve or more	<input type="checkbox"/> Haemorrhage
<input type="checkbox"/> Hypocalcaemia		<input type="checkbox"/> Triage kit	<input type="checkbox"/> Cerebral Edema
<input type="checkbox"/> Hypercalcaemia		(specify)	
<input type="checkbox"/> Other			<input type="checkbox"/> Other

Decontamination	Antidotes	Other treatment
<input type="checkbox"/> Ipecac	<input type="checkbox"/> Antivenin/Antitoxin	<input type="checkbox"/> Alkalinization
<input type="checkbox"/> AC single	<input type="checkbox"/> Atropine	<input type="checkbox"/> Anti-arrhythmia
<input type="checkbox"/> AC multiple	<input type="checkbox"/> BAL	<input type="checkbox"/> Anti-convulsants
<input type="checkbox"/> GL	<input type="checkbox"/> Benzodiazepines	<input type="checkbox"/> Anti-emetic
<input type="checkbox"/> WBI	<input type="checkbox"/> Calcium	<input type="checkbox"/> Anti-histamine
<input type="checkbox"/> Cathartic	<input type="checkbox"/> Deferoxamine	<input type="checkbox"/> Bronchodilator
<input type="checkbox"/> Irrigation/washing	<input type="checkbox"/> Dig Fab	<input type="checkbox"/> CPR
<input type="checkbox"/> Others	<input type="checkbox"/> DMPS	<input type="checkbox"/> Cardioversion
	<input type="checkbox"/> EDTA	<input type="checkbox"/> Defibrillation
	<input type="checkbox"/> Ethanol	<input type="checkbox"/> HD
	<input type="checkbox"/> Flumazenil	<input type="checkbox"/> HP
	<input type="checkbox"/> Folate	<input type="checkbox"/> Intubation
	<input type="checkbox"/> Glucogan	<input type="checkbox"/> IVF
	<input type="checkbox"/> Dextrose	<input type="checkbox"/> Oxygen
	<input type="checkbox"/> HBO	<input type="checkbox"/> Rapid Cooling
	<input type="checkbox"/> Hydroxocobalamin	<input type="checkbox"/> Pacing
	<input type="checkbox"/> Insulin	<input type="checkbox"/> Ventilator
	<input type="checkbox"/> Methylene Blue	<input type="checkbox"/> Sedation
		<input type="checkbox"/> Transplantation
		<input type="checkbox"/> Others

Disposal

- Discharge Discharge after observation Admission to general ward after observation
 Admission to ICU/CCU after observation Admission to psychiatric ward after observation
 Admission to general ward directly Admission to ICU/CCU directly DAMA/Disappeared Death

Outcome

- No effect Mild effect Moderate effect Major Effect Death Unknown
 Outcome relation to poisoning Definite Probable Possible Not related

HK Poison Information Centre Recommendation

Signed out by _____ on _____