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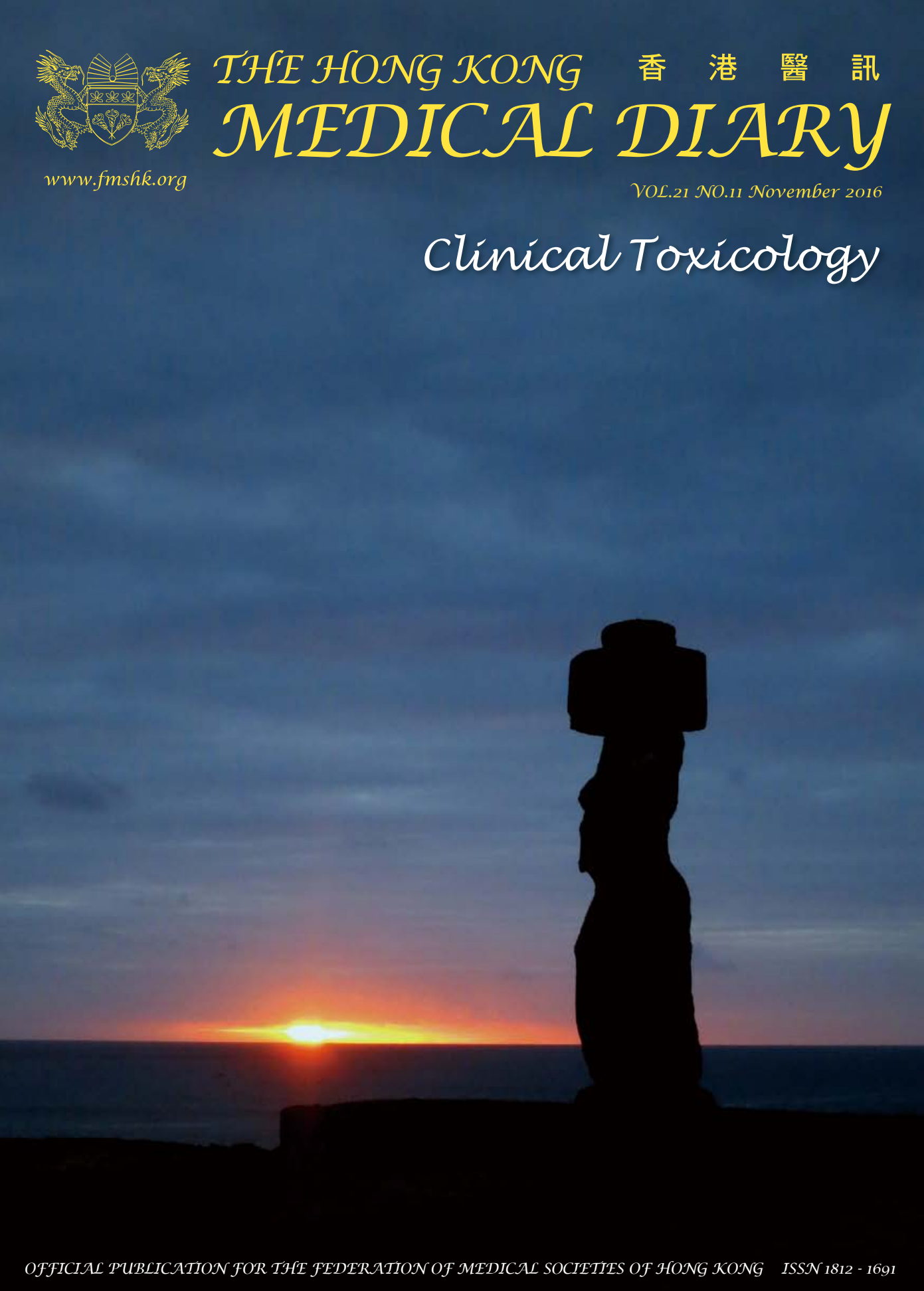
Clinical Toxicology



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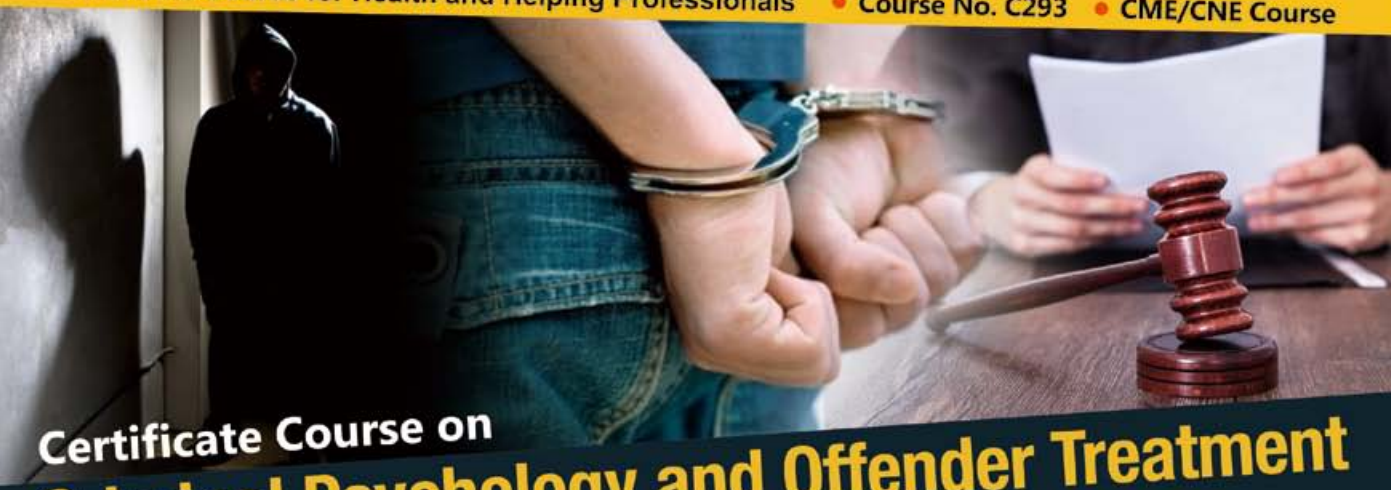

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Hong Kong Clinical Psychologists Association

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4 Nov	Criminal psychology, investigative psychology, and offender treatment: Applications in Hong Kong	Dr Ephraem Tsui Clinical Psychologist
11 Nov	Understanding and treating offenders with common and / or mild psychological problems	Dr Ephraem Tsui Clinical Psychologist
18 Nov	Understanding offenders with personality disorders and / or psychopathy	Ms. W. Y. Kung Clinical Psychologist (tentative to be confirmed)
25 Nov	Understanding and treating sex offenders	Ms. Sarina Lam Clinical Psychologist (tentative to be confirmed)
2 Dec	Understanding and treating violent offenders	Dr. Yvonne Lee Clinical Psychologist (tentative to be confirmed)
9 Dec	Offender assessment for the courts and statutory boards	Dr. Charles Pau & Dr. Ephraem Tsui, Clinical Psychologists (tentative to be confirmed)

Date : 4, 11, 18, 25 November and 2, 9 December 2016 (Every Friday)
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The Cover Shot

Dialogue with the Sun

This photo was taken in the Easter Island in 2012 autumn.

The sun descended just at the horizon, the light of the day faded slowly, revealing the silhouette of a moai.

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The Birth of Clinical Toxicology Subspecialty in Hong Kong

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Editor

Dr Fei-lung LAU

Clinical toxicology was a neglected subject in Hong Kong for decades. While the first poison information centres were established in Europe and North America in the 1950s, the first poison information service (namely the Drug & Poison Information Bureau) in Hong Kong was set up by Prof D M Davies in the Chinese University of Hong Kong in 1988.

As most of the acutely poisoned patients will present to Emergency Departments(ED), Emergency Physicians (EPs) have a leading role in clinical toxicology in many parts of the world including Hong Kong. After the return of a number of senior doctors from overseas toxicology training, the ED of United Christian Hospital (UCH) in 2000 started the first Hong Kong formal toxicology training as biweekly toxicology meetings & audit. It was later accredited as an official monthly training programme of the Hong Kong College of Emergency Medicine (HKCEM) since March 2001.

From 2002, HKCEM started to organise a 2 days clinical toxicology course. By 2016, a total of 10 courses were run and more than 1,000 health care workers had attended the course. With the establishment of the Hong Kong Poison Information Centre (HKPIC) in UCH in July 2005, for the first time in Hong Kong we had full time clinical toxicologists of emergency medicine training background. In the same year HKPIC and HKCEM jointly organised the certificate programme in clinical toxicology which is an eighty hours training programme targeted at clinicians interested in clinical toxicology.

In 2007, HKPIC developed the diploma programme in clinical toxicology. The programme was later recognised in 2011 by the Hong Kong Medical Council as a quotable qualification, namely "Diploma in Clinical Toxicology [Dip Clin Tox (HKCEM & HKPIC)]". By the end of 2015, a total of 39 doctors, all of them EPs, were awarded the diploma.

The programme initially required trainees to work for 6 months full time in the HKPIC, but soon it was extended to 12 months. The trainees needed to answer phone consultations for poisoned patients management and treating poisoned patients in the UCH ED. As some of the EDs had accumulated enough trainers in clinical toxicology, to allow flexibility in toxicology training, 7 EDs including United Christian Hospital (UCH), Queen Mary Hospital (QMH), Pamela Youde Nethersole Eastern Hospital (PYNEH), Caritas Medical Centre (CMC), Queen Elizabeth Hospital (QEH), Tuen Mun Hospital (TMH) and Princess Margaret Hospital (PMH) were accredited for toxicology training for up to 6 months one after another.

With the improved training in clinical toxicology to emergency physicians, the management model of poisoned patients in public hospitals has undergone major changes. While the majority of poisoned patients were admitted as inpatients into medical, paediatric or Intensive care unit wards 15 years ago, most of these poisoned patients are now managed in the emergency medicine wards in all of the toxicology training units. This is important as the early intervention

in EDs provides timely treatment of resuscitation, decontamination and antidotes, all of which would definitely improve the outcome of the poisoned patients, not to mention the additional benefit of more than 50% reduction in the admission rate for poisoned patients. In fact, the first emergency medicine (cum toxicology) ward was established in QMH in 2014 which signified our ED subspecialisation in poisoned patient management.

As more and more of our Emergency Medicine (EM) fellows are trained in clinical toxicology, HKCEM saw the need in developing its first subspecialty in EM, namely clinical toxicology. In 2014, the provisional board of clinical toxicology was formed to push for the establishment of the subspecialty. In March 2016, the Hong Kong Academy of Medicine finally approved the formation of the clinical toxicology subspecialty under the HKCEM. The conferment ceremony of the first fellows in clinical toxicology would be held in the annual general meeting in October 2016, which happens to coincide with the 20th anniversary of the HKCEM.

Since the turn of the century, the development of clinical toxicology in Hong Kong has advanced so fast that we are now the second city in the world after USA that runs a toxicology fellowship training programme. We also boast the best toxicology training centre in Asia receiving trainees from Korea, Malaysia, Singapore and the China Mainland.

To commemorate the setup of our subspecialty, this issue of the Medical Diary is dedicated to clinical toxicology and is written by a number of toxicology-trained EM physicians. They, after the assessment of the vetting committee of clinical toxicology board, would likely be accepted as the first or founding fellows of the clinical toxicology subspecialty of the HKCEM and constitute the backbone of further development of clinical toxicology in Hong Kong.

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Chinese medicine associated poisoning

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Introduction

Chinese medicines (CM) are classified legally in Hong Kong into (1) Chinese Herbal Medicine (CHM) and (2) Proprietary Chinese Medicine (PCM). CHM refers usually to the dried pieces of herbs used for decoction. However, it also encompasses CHM granules (中藥顆粒). CHM is usually dispensed according to the formulae written by CM practitioners. Majority of CHM is prepared from medicinal plant parts but animal products as well as minerals are also utilised. PCM covers the formulated CM that is usually in the forms of big pill(丹), paste(膏), small pill(丸) and powder(散). Historically, PCM was manufactured for the ease of storage and readiness to use particularly for travellers, children and illnesses that need emergent treatment.

Poisoning associated with CHM

Aconite poisoning

The commonest acute CHM poisoning reported in Hong Kong was due to Aconite rootstocks. The rootstocks of a few highly toxic Aconite plant species are used in CHM. The most frequent ones are Fuzi (附子), Chuenwu(川烏), Caowu (草烏). In the raw form, they contain potent sodium channel toxins with higher selectivity for the myocardium than the nervous system. They are usually processed or better known as cured (炮製) through steaming to lower their toxicity and to enhance the wanted therapeutic effect. Even after curing, their therapeutic indexes are still narrow with the recommended upper dose-limits ranging from 3gm -15gm. Long period of boiling is also recommended in the preparation of decoctions with aconite herbs. Aconite poisoning usually presents within 4 hours with a clinical triad of gastrointestinal upset, paresthesia and cardiac arrhythmias. Hypotension and atrioventricular conduction blockage are commonly seen in moderate to severe poisoning. Severely poisoned patients may develop life-threatening ventricular arrhythmias and cardiac arrest. Supportive treatment, inotropes and anti-arrhythmic treatment with amiodarone or magnesium sulphate are usually effective[1]. In severe cases with recurrent ventricular arrhythmia, charcoal haemoperfusion has some reported usefulness. Extra-corporal circulatory support is a reasonable treatment for patients with cardiovascular collapse.

Every year around 10-20 cases of aconite poisoning related to CM are recorded in Hong Kong. The commonest underlying causes are therapeutic overdose and contamination of benign herbs by aconite rootstocks. Each of them accounts for about one-third of all aconite poisonings. Other important causes include inadequate boiling and quality defect of the herbs[2].

Anti-cholinergic poisoning

Another recurring CHM related poisoning is herb-induced anti-cholinergic poisoning. Patients typically present with anti-cholinergic delirium, confusion with varying degree of peripheral anti-cholinergic signs and symptoms like mydriasis, dry mouth, bloating and urinary retention. Life-threatening anti-cholinergic poisoning due to CM use is rare but possible. The delirium usually lasts for 1-2 days. The specific antidote physostigmine, a centrally acting anti-cholinesterase, can promptly reverse the clinical toxicity if used appropriately.

The commonest cause of CM related anti-cholinergic poisoning in Hong Kong is the substitution of the benign herb Flos Campsis (凌霄花) by Yangjinhua (洋金花 Flos Datura). The latter is made of flowers of the Datura(曼陀羅) species that are well-known to be toxic. Datura plant parts harbour high concentrations of tropane or belladonna alkaloids such as atropine, scopolamine, and hyoscyamine . This probably explains the use of its flower in CHM for asthma and other respiratory conditions. The second commonest cause is contamination of benign herbs by tropane-alkaloid-containing plant parts. One most frequently affected CHM in the past was Cangzhu (倉朮) while a handful of other CHM have been contaminated on occasions[3].

Gelsemium

The toxic shrub or vine Gelsemium elegans (斷腸草) is native to Southern China including Hong Kong. It contains toxic alkaloids known as gelsemines. The use of its rootstocks as CM has been documented but only topically for skin conditions, soft tissue injuries and fractures. When taken orally, it typically causes diplopia due to extraocular muscle weakness, ptosis and bulbar weakness. Associated symptoms like dizziness, nausea and vomiting may or may not present. In severe case, coma and respiratory failure may result. No specific antidote is available but timely respiratory support and airway protection is usually effective. Despite its rapid onset, clinical toxicity usually subsides within 24 to 48 hours[4,5].

Gelsemium poisonings of mild to moderate severity have occasionally resulted from the consumption of soup prepared with Gelsemium-contaminated hairy fig roots (五指毛桃Ficus hirta Vahl). Contamination

happened probably in the process of harvest for the hairy fig roots because the two plants tend to grow nearby in similar geographical locations. More severe or life-threatening poisonings have happened after consumption of CM broth made from Gelsemium being misidentified for benign medicinal plants or parasitic vines that fed on gelsemium elegans[5].

Cardiac glycoside containing CHM

Chen-su (蟾酥 toad venom), a highly toxic CHM is made from the secretion of the venom gland on its skin. It contains rich mix of substances with cardiotoxic as well as hallucinogenic effect that include cardiac glycosides and cardioactive steroids pharmacologically similar to digitalis. The recommended dose is below 30mg (一厘) and is used rather exclusively in the making of pills of PCM. Fatal poisonings happened before when it was mistakenly substituted for a less toxic CHM charred toad-skin in Hong Kong and for donkey-hide-gelatin (阿膠) in the United States[6]. Reported poisonings were severe and often resulted in death due to the occurrence of ventricular arrhythmias and asystole. Digoxin antibody has been reported to be useful in its treatment.

Another herb that has caused poisoning but to a lesser severity is Chuenqi (川七). Chuenqi is a confusing name that refers to at least 3 plant species that are being used as CHM or consumed as food: Panax notoginseng (Sanqi 三七or田七), Tupistra chinensis (川三七or 開口箭) and Anredera cordifolia (滕三七). Among them Tupistra chinensis contains rhodexin, a cardiac glycoside. Poisoning resulted in dizziness, nausea, vomiting and bradycardia.

Aristolochic acid containing CHM

Some Aristolochia plants contain aristolochic acids (AA), particularly AA1 and AA2 can cause DNA adduction and are proven potent carcinogens and mutagens. AA-DNA adducts were found in 60% of upper urinary tract urothelial carcinoma samples from Taiwan so that exposure to AA herbs may have contributed to the exceptionally high prevalence of the carcinoma in the area[7]. AAs are also nephrotoxic. It was first reported to cause rapidly progressive interstitial nephritis in Belgium[8], then followed by similar cases reported from other countries making it once known as "Chinese Herbs Nephropathy". Now, evidence showed that it can cause chronic tubulointerstitial kidney disease that may progress to end-stage renal failure over a period of months to decades after the initial period of exposure[9].

Herbs containing AA include Caulis Aristolochiae Manshuriensis(關木通), Radix Aristolochiae Fangchi (廣防已), Fructus Aristolochiae (馬兜玲), Radix Aristolochiae (青木香) and Herba Aristolochiae (天仙藤). They have been used for centuries before the discovery of their toxicity. Confirmatory diagnosis of AA Nephropathy requires renal biopsy for DNA-AA adduct testing that is not available from clinical laboratory. Treatment is avoidance of further AA exposure while corticosteroids may have values in slowing the disease progression and continual urothelial cancer surveillance may be indicated. Although banned in Hong Kong and the China Mainland, it is possible that Aristolochia herbs might still be used erroneously in CHM or in the manufacture of poor quality PCM. Recent evidence from molecular epidemiological studies also suggested that contamination of wheat by Aristolchia was the likely cause of the mysterious Balkan Endemic Nephropathy[10]. The disease burden of AA nephropathy worldwide should have been underestimated as Aristolochia plants have been and continue to be used in traditional medicines around the world.

Poisoning associated with PCM

Legally speaking, only those PCMs that have been registered with the Department of Health can proclaim themselves PCM in Hong Kong. The unregistered ones, although fulfilling the traditional sense of PCM, should be regarded as health supplements that are receiving less stringent control on its quality than PCM. PCM carries with itself a different profile of toxicity from CHM. The main concerns are (1) exposure to metals that include lead, mercury and arsenic in both registered and unregistered PCM and (2) adulteration with Western drugs in those illicit self-proclaimed PCM products.

In the past, minerals that contain mercury, lead and arsenic compounds were employed as ingredients in PCMs. Most of them are banned in Hong Kong now. Toxic minerals banned in PCM include calomel (輕粉 mercurous chloride), once used against roundworm infestation in the West as well as in China, has caused inorganic mercury poisoning to children in Hong Kong[13]. Arsenic compounds like arsenic trioxide (砒霜), arsenolite(砒石), orpiment(雌黃) are other examples of banned ingredients. An outbreak of subacute arsenic poisoning manifested with peripheral neuropathy and dermatological lesions happened in 2015. The victims were psoriasis patients seeking treatment outside Hong Kong. Lead oxide (紅丹or 鉛丹) has been described as a toxic CM ingredient in the most ancient CM pharmacopeia Shengnong's Herbal Classic(神農本草經). Lead poisonings have been resulted from the PCM made by a CM practitioner[11] and even from the intake of Chinese talisman for illness[12]. Currently, only two metal-containing minerals, realgar and cinnabar (朱砂 mercurous sulphide) are formulated in the registered PCM in Hong Kong. The former was found in 52 and the latter in 64 PCMs respectively[14]. They should have minimal acute toxicity but if consumed in a long term period, there exists a risk for chronic arsenic or lead poisoning.

The adulteration with Western drugs in illicit health supplements with a made-to-believe PCM name and package are posing health risks to most developed countries. They typically targeted persons with chronic illnesses like diabetes mellitus, osteoarthritis, obesity and sex under-potency. The danger is the uninformed risk of adverse drug reactions that are often compounded with supra-therapeutic dosing. Even worse, drugs that have been withdrawn because of their toxicities were frequently used in the adulteration.

Conclusion

The consumption of CM carries with it a low but not negligible risk. In CHM, Aconite, tropane alkaloid containing herbs and Gelsemium repeatedly cause acute poisonings that could be life-threatening. Substitution and contamination of benign herbs by toxic ones are

the underlying causes in the majority of cases. Further research on CM pharmacovigilance is warranted to ensure the short and long-term safety of CHM.

In PCM, the main concern is its containment of metals and adulteration by Western drugs. The underlying causes are often due to unlicensed or illicit products. Good policing of the health supplement market as well as targeted public education would help to combat such PCM related poisonings.

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Mushroom Poisoning in Hong Kong: From Death Cap to Porcini

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Mushroom poisoning is uncommon in Hong Kong despite we consume huge amount of commercially sold cultivated mushrooms every year. The topic doesn't receive much public attention until year 2013, when a couple were poisoned by the amatoxin-containing mushroom self-picked near Shing Mum reservoir[1]. The couple survived through the tragedy, though the wife requiring liver transplantation. In the past 11 years, the Hong Kong Poison Information Centre (HKPIC) has recorded eight cases of amatoxin-containing mushroom poisoning in Hong Kong, resulting in one death and two liver transplantations[2]. Frontline colleagues need to familiar with the telltale signs of this fatal poisoning and know when to seek for assistance.

Clinical presentation and local epidemiology

After reviewing all available local reports, four mushroom poisoning syndromes (see Table 1), together with infective causes of food poisoning and food allergy, were identified to be the cause of all mushroom poisoning cases in the past[2,3,4]. The most common clinical presentation is gastroenteritic mushroom poisoning, which accounts for about 60% of all cases. The reported causative mushroom include *Chlorophyllum molybdites* 綠褶菇 (Fig. 1), *Russula emetic* 毒紅菇, *Macrolepiota neomastoidea* 大環柄菇, *Lepiota atrosquamulosa* 黑鱗環柄菇. Many of these cases were related to mixing up of edible and inedible mushrooms in wild mushroom picking.

Table 1: The four mushroom poisoning syndromes reported in Hong Kong

Syndrome	Clinical features	Prognosis
Gastroenteritic mushroom poisoning	Vomiting and diarrhoea within 0.5-3 hours post-ingestion, and resolved in 6-24 hours	Full recovery with supportive treatment
Amatoxin poisoning	Gastrointestinal symptom onset at 6-24 hours post-ingestion, followed by acute liver and renal injury.	About 40% mortality in Hong Kong
Cholinergic mushroom poisoning	Gastrointestinal upset, with additional cholinergic features including profuse sweating, increased salivation, lacrimation, bradycardia, and blurred vision. Typically early onset within 0.5-2 hours post-ingestion	Full recovery with supportive treatment
Hallucinogenic mushroom poisoning	Neuropsychiatric symptoms including auditory or visual hallucinations, confusion, and delirium. Symptoms can last for 1-2 days.	Full recovery with supportive treatment.

Fig. 1: Chlorophyllum molybdites. The most commonly consumed poisonous wild mushroom. Clinical presentation is gastroenteritic mushroom poisoning.

Fig. 2: Amatoxin-containing mushrooms in Hong Kong

Contradictory to most believes, poisonous wild mushrooms are seldom brilliant in colour. In fact, the appearance of the death cap (*Amanita Phalloides* 毒鵝膏) and the local amatoxin-containing mushrooms (*Amanita farinosa* 小托鵝膏菌, *Amanita exitialis* 致命鵝膏菌)(Fig. 2) are quite benign looking. The practice of wild mushroom consumption in Hong Kong is considered to be risky. This can be illustrated by the fact that over 388 known mushroom species in Hong Kong, fewer than 10% are edible, with a majority have unknown edibility[2,5]. Moreover different edible and inedible mushroom species can share the same habitat and grow together in the wild. Collection of mixed species often happens. For the amatoxin poisoning incident in 2013, the couple had collected predominantly edible fungus (*Auricularia auricula*黑木耳), with pieces of deathly Amanita farinosa mixed in the harvest.

For those experienced readers, they may notice that many deadly mushroom poisoning syndromes reported globally were not included in Table 1. There

are 14 distinctive types of mushroom poisoning found worldwide[6,7]. According to the investigation of 183 mushroom poisoning deaths in Southern China from 1994 to 2012, hepatotoxic syndrome, nephrotoxic syndrome, rhabdomyolytic syndrome, and haemolytic syndrome were reported to be the clinical presentation in these cases[8]. With the exception of hepatotoxic syndrome, other potentially lethal mushroom poisoning syndromes are not yet reported in Hong Kong. With the geographic proximity to Southern China and the Mainland-Hong Kong cultural integration, it is expected that the incidence of wild mushroom poisoning will increase and we may see these mushroom poisoning syndrome in the future.

Amatoxin poisoning

Missing the diagnosis of amatoxin poisoning can result in catastrophic outcomes. The hepatotoxic syndrome caused by amatoxin poisoning is the only life-threatening mushroom poisoning syndrome reported in Hong Kong. The toxins involved in amatoxin poisoning include amatoxins, phallotoxins and other cyclopeptides, which are produced by certain mushroom species of three genera, namely the Amanita, Galeina, and Lepiota[7]. The infamous Amanita Phalloides, also known as the death cap, has been involved in the majority of mushroom poisoning deaths globally. The toxins are heat stable and cannot be destroyed by cooking or other means of food processing. After gastrointestinal absorption, the toxins go through the enterohepatic circulation, and are preferentially absorbed by hepatocytes through a specific transporter mechanism[7]. Once inside the cells, the toxins interfere with RNA polymerase and inhibit protein synthesis, leading to cell death.

The typical clinical presentation of amatoxin poisoning consists of three sequential phases. Phase I is characterised by severe gastroenteritis which starts at 6 to 24 hours post-ingestion. This is the most important distinguished feature of amatoxin poisoning, as other benign gastroenteritic mushroom poisonings present with rapid onset symptoms within 4 hours post-ingestion. Phase II is the transient improvement stage which occurs between 12 to 36 hours post-ingestion. This is followed by phase III which manifested by hepatic and renal failure on 2 to 6 days post-ingestion[7]. According to the local cases of amatoxin poisoning, the most notifiable features (i.e. red flag signs) are:

1. All cases involved the consumption of self-pick wild mushroom[2].
2. Gastrointestinal symptoms began at 6 hours or more after mushroom consumption. The median symptoms onset time was 11 hours[2].
3. Predominant gastrointestinal symptom was typically described as cholera-like profuse watery diarrhoea[7].

According to overseas experience, the survival rate of amatoxin poisoning can be achieved 70 to 100% with early diagnosis and specific therapies[7]. Basic supportive care includes vigorous fluid replacement, maintaining electrolytes and acid-base homeostasis, and correction of coagulopathy in intensive care setting. For specific treatment, it can be sub-classified into medical and surgical. N-acetylacysteine (NAC), silibinin, penicillin G, multiple dose activated charcoal, and early charcoal haemoperfusion constitute the mainstay of medical treatment in Hong Kong[7,9]. NAC is believed to be hepatoprotective by acting as an oxygen free radical scavenger. The antidote silibinin works by competitively inhibiting the specific transporter that is responsible for the hepatocytes uptake and enterohepatic recycling of amatoxins. The role of penicillin G in the treatment of amatoxin mushroom poisoning is controversial. The proposed therapeutic mechanisms include blocking amatoxins uptake from hepatocytes and binding to circulating amatoxins. Oral multiple dose activated charcoal can be considered for patient presenting within 3 days post-ingestion. It works by inhibiting amatoxin absorption through intestinal mucosa and reabsorption via the enterohepatic circulation. The use of early charcoal haemoperfusion is controversial and should be considered on a case-by-case basis. For cases progressed into fulminant hepatic failure, liver transplantation is the last resort.

Fig. 3: Dried porcini involved in a recent incident of mushroom poisoning

Porcini poisoning: a new mode of mushroom poisoning in Hong Kong

Wild-harvested porcini (牛肝菌) has been regarded as a delicacy with high nutritional and medicinal value. Pre-packed dried porcini can be bought from local supermarkets and health food stores (Fig. 3). According to the HKPIC record, there were 13 cases of porcini poisoning from July 2005 to June 2015[2]. The poisoning incidence doubled recently, with 8 poisoning incidents involving 14 patients in the past 13 months (July 2015 to August 2016). All cases involved the consumption of commercially purchased dried porcini, with the source of porcini originated from the Mainland China in most cases. Most patients presented with early onset repeated vomiting and abdominal pain, with or without diarrhoea. The clinical diagnosis of gastroenteritic mushroom poisoning was obvious in these cases. Interestingly, several patients presented with neuropsychiatric symptoms without gastrointestinal symptoms after porcini consumption. Visual and auditory hallucinations were commonly

reported in these cases, together with other neurological symptoms including dizziness, malaise and generalised numbness. Mycological identification revealed mixing up of edible porcini and inedible boletes in most cases. Gastroenteritic and hallucinogenic mushrooms were frequently found in these dried porcini samples. Two common culprits, *Boletus luridus* (見手青) and *Heimioporus retisporus* (網孢海氏牛肝菌) were shown in Fig. 4.

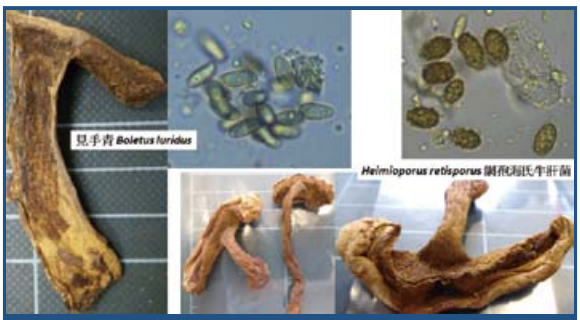

Fig. 4: Gastroenteritic and hallucinogenic mushrooms commonly identified in dried porcini samples.

Courtesy

I would like to thank Professor SW Chiu and the School of Life Sciences, the Chinese University of Hong Kong, for providing us with the mushroom photographs in this article. Their support in urgent mycological identification is essential in our routine management of mushroom poisoning patients.

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Dermatological Quiz

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Specialist in Dermatology and Venereology

Dr Chi-keung KWAN

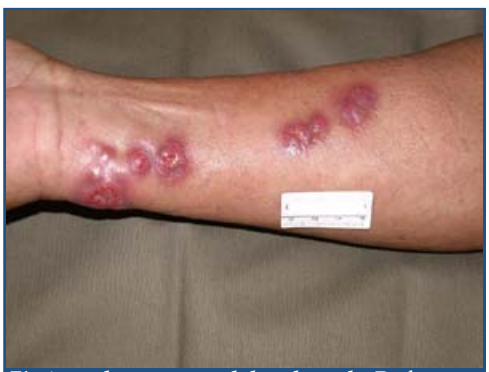

Fig.1: erythematous nodules along the Rt forearm

This 42-year-old man HIV infected patient complained of multiple erythematous lumps that rapidly grew on the right forearm within 6 months. They were not itchy. However, he noted that the nodules appeared one by one from the wrist "spreading" to the ante-cubital fossa. Physical examination revealed multiple erythematous roundish nodules lying on the ventral aspect of the right forearm extending from the wrist to the ante-cubital fossa. Superficial erosions were found on the surface of the nodules(Fig. 1).

Questions

1. How do you describe the pattern of distribution of the skin nodules?
2. What are the differential diagnoses of his skin lesion?
3. What other history do you want to elicit?
4. What investigation are you going to order?
5. How do you treat this patient?

(See P. 37 for answers)

Drug abuse in the acute medical setting

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This article has been selected by the Editorial Board of the Hong Kong Medical Diary for participants in the CME programme of the Medical Council of Hong Kong (MCHK) to complete the following self-assessment questions in order to be awarded 1 CME credit under the programme upon returning the completed answer sheet to the Federation Secretariat on or before 30 November 2016.

Introduction

Patients with drug abuse are commonly encountered in the acute medial setting. "Abuse" was the third most common reason of poisoning for patients recorded by the Hong Kong Information Centre (HKPIC) while amphetamines and ketamine were among the top 20 poisons in the year 2014 (Chan 2015). The most commonly abused substances recorded in HKPIC in 2015 were methamphetamine (28%), ketamine (21%), opioids (19%), benzodiazepines and other sedatives (16%), cough mixture (4%), cocaine (4%) and cannabis (4%) (unpublished data).

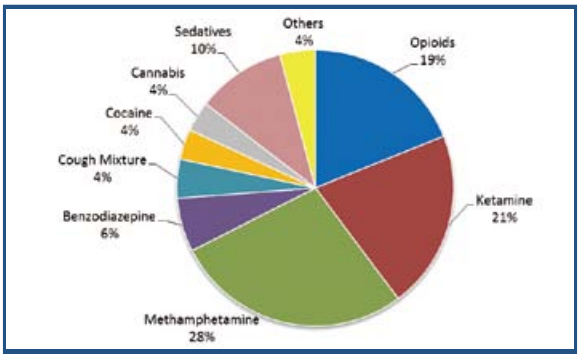

Chart 1: Types of drug abuse recorded by HKPIC in 2015

The rise of amphetamine type stimulants

The United Nations Office on Drugs and Crime estimated global seizures of amphetamine-type stimulants (ATS) of 173 tons in 2014, in which methamphetamine accounted for the majority of it. Globally, ATS are the second most abused drugs (after cannabis) and expert perceptions of trends and treatment admission reports suggested an increase in the use of amphetamines in East and South-East Asia (UNODC 2016).

In Hong Kong, methamphetamine had surpassed ketamine as the most common abusive substance presenting to the acute health care setting since 2014 (Chan 2015). This is in agreement with the general trend of drug abuse of Hong Kong. The Central Registry of Drug Abuse (CDRA) data showed that methamphetamine has been the most common abusive substance in abusers aged under 21 and the second most abused substance in abusers of all ages since 2015 (CDRA 2016).

Most abusers in Hong Kong use the crystalline form of methamphetamine, which has a street name of "ice" or "冰" in Chinese in the form of inhalation - the act of "僕冰". Acute clinical effects of methamphetamine abuse are largely a result of catecholamine excess which include cardiovascular effects (hypertension, tachycardia, myocardial ischaemia, arrhythmias), central nervous system effects (agitation, euphoria, hyperreflexia, hyperthermia, paranoid psychosis, seizures, intracerebral haemorrhage), other sympathomimetic symptoms (diaphoresis, mydriasis, tachypnoea), rhabdomyolysis, acute kidney injury, metabolic acidosis and hyperglycaemia, etc. Multi-organ failure and death may ensue if these systemic signs are not reversed (Jang 2015).

Patients with acute methamphetamine toxicity should be considered a medical emergency. From our experience, a typical methamphetamine abuser with acute toxicity presenting to the emergency department is a patient who is delirious and agitated, with sweating, hyperthermia, hypertension and tachycardia. Adequate number of staff with experience in dealing with an agitated patient should be present in order to protect both the patient and frontline health care workers. Benzodiazepines are the first choice agent of chemical sedation and should be given promptly to patients with overt sympathomimetic effects. Titrated intravenous doses should be given until the patient calms down with improvement of the sympathomimetic effect. Adequate hydration, physical methods of cooling and other supportive measures should be given according to the clinical situations (Jang 2015). Physical restraint alone without chemical sedation should be discouraged as inappropriate physical restraint in a patient with stimulant toxicity may be a risk factor of unexpected death (excited delirium) (Otahbachi 2010).

Apart from the acute toxicity, methamphetamine abusers are well known to have chronic psychiatric consequences, which include psychotic symptoms, persecutory delusions, auditory hallucinations, delusion of parasitosis or formication, "punding" (non-goal directed repetitive activity) and cognitive

defects (relating to learning, episodic memory, speed of information processing etc.) (Rusyniak 2013; Panenka 2013). Damage at dopaminergic and serotonergic axons may occur after repeated exposures to methamphetamine and structural and metabolic brain changes have been detected by magnetic resonance imaging (MRI) and positron emission tomography (PET) in methamphetamine abusers (Chang 2007). For this reason, even though the psychotic symptoms of some methamphetamine abusers may disappear shortly after stopping the use of the drug, some other abusers may have prolonged symptoms of more than 6 months (Glasner-Edwards 2014).

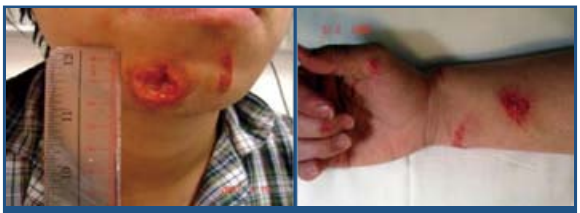

Fig. 1: Delusion of parasitosis - Injuries inflicted by the patient himself while trying to pick out some "worms" under his skin.

Ketamine abuse

Ketamine has been the most commonly abused substance in abusers aged under 21 since 2005, which has only recently been replaced by methamphetamine since 2014/15.

Ketamine is a structural analogue of phencyclidine, a dissociative anaesthetic first introduced in the 1960s. Hallucinations and an "out-of-the-body" sensation similar to near death experience have been noted and ketamine was used for abuse soon after its use as an anaesthetic agent (Reier 1971).

Ketamine is usually snorted in Hong Kong – the act of "索K". In a review of 233 patients presenting to the emergency department, we found that the most common symptom of ketamine misuse was impaired consciousness, followed by abdominal pain, lower urinary tract symptoms and dizziness while the most common abnormal physical findings were high blood pressure, followed by tachycardia, abdominal tenderness and white powder in the nostrils (Ng 2010). In the same series, most patients had only minor complaints and could be managed conservatively in the Accident and Emergency Department. Ketamine abusers usually recover within a few hours and simple supportive measures, including intravenous fluid and the use of benzodiazepines for agitation can be given (Ng 2010; Olmedo 2015).

First noticed and published by clinicians in Hong Kong, patients with chronic ketamine abuse are also associated with urologic (lower urinary tract symptoms, hydronephrosis, renal failure) and hepatobiliary dysfunction (Chu 2007; Wong 2009; Mak 2011; Seto 2011). In another review of 96 chronic ketamine abusers presenting to our outpatient clinic, most of the patients had urinary symptoms (dysuria, urgency and frequency) while others presented with chronic abdominal pain, nasal problems (e.g. septal perforation) and psychiatric symptoms (Chan 2012). For patients with ketamine uropathy, abstinence is an important first step to prevent further damage to the urological tract. Other surgical measures like intravesical instillation therapy, percutaneous nephrostomy and augmentation cystoplasty/neobladder reconstruction may be required (Ma 2015). A multi-disciplinary approach involving social worker, emergency physicians and urologist is required for proper management of the chronic ketamine abusers.

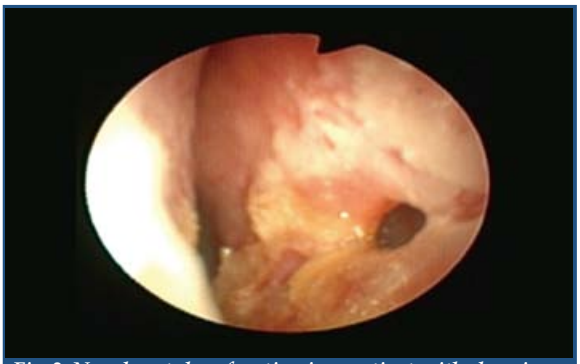

Fig. 2: Nasal septal perforation in a patient with chronic ketamine abuse.

Zopiclone and other sedatives abuse

Zopiclone abuse is a rather unique problem in Hong Kong compared with other developed countries. It is not uncommon to have patients who habitually take 10 to 20 tablets of zopiclone (typically 7.5 mg per tablet) every day. First documented in Hong Kong, massive zopiclone intake (usually more than 50 tablets of zopiclone) may cause methaemoglobinaemia, haemolysis and renal impairment (Fung 2008; Fung 2009). The methaemoglobinaemia may be severe (more than 20%) and requires the use of methylene blue as the antidote.

Frontline clinicians may also encounter chronic abusers who have withdrawal symptoms (for example tremor, irritability, sweating, convulsion) if these abusers abruptly stop taking the drugs. Recognition of the history of chronic sedative abuse and an adequate dose of sedation are keys to successfully manage these patients.

Opioids

Opioid abuse, most commonly heroin injection remains a common encounter in the acute care setting as heroin abuse is still the most common drug abuse recorded by CDRA in all age groups (CDRA 2016). Patients with toxicity from opioid abuse typically present with respiratory and central nervous system depression, as well as miosis. Initial management includes airway and ventilatory support, followed by a titrated dose of naloxone (start at a low dose e.g. 0.1 mg IV for opioid dependent patients), to those with profound respiratory suppression. Complications including acute lung injury, compartment syndrome, rhabdomyolysis and injection site related complications (e.g. bleeding and infection) may also occur.

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Cough mixture abuse

Cough mixtures commonly abused in Hong Kong usually contain one or more of the following ingredients: dextromethorphan, sympathomimetics (e.g. ephedrine, pseudoephedrine), opioids, or anti-cholinergic. Patients with acute toxicity from the various ingredients and chronic toxicity with cognitive deterioration may be encountered. Withdrawal symptoms may also occur if the patient stops using the cough mixture abruptly.

Novel psychoactive substances

As of March 2015, the European Monitoring Centre for Drugs and Drug Addiction (EMCDDA) has been monitoring more than 450 novel psychoactive substances (NPS), including synthetic cathinones, synthetic cannabinoid receptor agonists, phenethylamines, tryptamines,arylamines, new opioids and benzodiazepines etc. (EMCDDA 2015). Many of the NPS, like paramethoxymethamphetamine (PMMA), trifluoromethylphenylpiperazine (TFMPP) and NBOMe etc. have been used by abusers in Hong Kong (Ng 2012; Tang 2014; Tang 2015).

Detection and management of patients who abuse NPS are difficult as most of the NPS cannot be detected by routine toxicology analysis and clinical experiences are often limited. Target screening of NPS should be requested and is available in tertiary laboratory (Tang 2015).

Conclusion

Patients with drug abuse are commonly seen in the acute medical setting. With the recent rise in methamphetamine use, it is likely that we would encounter more patients presenting with sympathomimetic toxicity from amphetamine type stimulants. Prompt and appropriate treatment is required to prevent patient morbidity and mortality. Chronic abusers may have psychiatric and other complications which are best managed by a multi-disciplinary approach.

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Drug abuse in the acute medical setting" by Dr Sze-hong NG and complete the following self-assessment questions. Participants in the MCHK CME Programme will be awarded CME credit under the Programme for returning completed answer sheets via fax (2865 0345) or by mail to the Federation Secretariat on or before 30 November 2016. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

Questions 1-10: Please answer T (true) or F (false)

1. The most common drug abused by abusers in Hong Kong aged below 21 is methamphetamine.
2. Most methamphetamine abusers in Hong Kong take the drug by the oral route.
3. Combative methamphetamine abusers with signs of acute intoxication should be sedated with haloperidol.
4. Delusion of parasitosis is one of the psychiatric symptoms in methamphetamine abusers.
5. The most common presentation of ketamine abuser with acute intoxication is impaired conscious level.
6. Augmentation cystoplasty is the first-line treatment for chronic ketamine abusers with uropathy and renal impairment.
7. Massive zopiclone overdose may produce methaemoglobinaemia.
8. Unlike benzodiazepine abuse, withdrawal symptoms in zopiclone abusers have not been reported.
9. An intravenous heroin addict who presents with respiratory failure should be given naloxone 0.4 mg intravenously as the first treatment.
10. Most novel psychoactive active substances (NPS) can be detected by routine toxicology screening of the patient's urine.

ANSWER SHEET FOR NOVEMBER 2016

Please return the completed answer sheet to the Federation Secretariat on or before 30 November 2016 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Drug abuse in the acute medical setting

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MBBS (HK), FHKCEM, FHKAM (Emergency Medicine)

Associate Consultant, Hong Kong Poison Information Centre, Hospital Authority

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Answers to October 2016 Issue

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Envenomation by Indigenous Snakes in Hong Kong

Dr Hin-tat FUNG

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Epidemiology

The yearly number of snakebite cases attending Accident and Emergency Departments of the Hospital Authority was stable at about 130 to 140 in the past three years.[1] Given the scarcity of other sources of medical care, the figures should have covered almost all the venomous snakebites. The number of non-venomous bites, which consists of only a minor proportion of the above figures, however may be under-estimated since some victims may be able to recognise the non-venomous nature of the culprit species and therefore do not seek medical help. This article centres on the envenomation features by the native venomous snakes and the pre-hospital management.

Venomous snake species

In Hong Kong, all sea snakes are venomous but land snakes are not. There is no report on sea snakebites in recent years and thus discussion here would be restricted to only the indigenous land snakes which are divided into three families and nine species:

Family	Species
Viperidae	Bamboo snake 青竹蛇 *Trimeresurus albolabris* Point-scaled pit viper 烙鐵頭 *Protobothrops mucrosquamatus* Mountain pit viper 山烙鐵頭 *Ovophis monticola*
Elapidae	Chinese cobra 飯鏟頭 *Naja atra* King cobra 眼鏡王蛇 *Ophiophagus hannah* Many-banded krait 銀環蛇 *Bungarus multicinctus* Banded krait 金環蛇 *Bungarus fasciatus* MacClelland's coral snake 麗紋蛇 *Sinomicrurus macclellandi*
Colubridae	Red-necked keelback 紅脖游蛇 *Rhabdophis subminiatus*

Of all the above snakes, the bamboo snake, Chinese cobra and many-banded krait in descending order account for almost all the venomous snakebites, while point-scaled pit viper and red-necked keelback are responsible for sporadic incidents. Snakebites more commonly occurs in the evening and in October and November. The majority of bites are outdoor but at times Chinese cobra and many-banded krait bites happen inside village houses.

Pathogenesis and clinical features

Bamboo snake

The venom of bamboo snake produces basically two forms of envenomation: local cellular injury (cytotoxicity) and haemotoxicity. Cytotoxicity is a result of metalloproteinase, hyaluronidase and other components in the venom. Severe pain and gross swelling up to the entire limb are not uncommon. Sometimes paresthesia is noticed around the wound, which may be explained by the minute amount of neurotoxin in the venom. Necrosis is seldom and even if occurs, is small in surface area. Subcutaneous tissue inflammation, and only rarely compartment syndrome, is the reason behind the marked external swelling. Any suspected compartment syndrome should be confirmed by objective measurement to avoid unnecessary fasciotomy.

In contrast to cytotoxicity, haemotoxicty is potentially disastrous if not followed by close monitoring and any indicated therapy. Among the major toxins in bamboo snake are the thrombin-like enzymes. They mimic the serine protease thrombin in human and can activate the coagulation pathway, but without forming normal cross linked fibrin polymers. The end results are over-consumption of fibrinogen and other clotting factors with the formation of only friable clots. Another important action of the venom is inducing thrombocytopenia by means of platelet sequestration and aggregation. The above haemotoxic actions in conjunction with the disintegration of the basement membrane of vasculature linings by the venom may manifest clinically as subcutaneous ecchymosis or in serious cases, internal organ haemorrhage.

The peak of haemotoxicity at least takes several hours to appear because of the gradual nature of absorption of the venom from the bite site to the systemic circulation. Repeat blood tests are mandatory to avoid missing a delayed haemotoxicity.

Chinses cobra

Chinese cobras possess cardiotoxins, which are cardiotoxic to small animals but rarely to humans. The cardiotoxins can directly elicit cell injury and death, as well as indirectly by inducing vasculitis and vascular thrombosis first. In opposition to many other overseas cobras which inflict systemic neurotoxicity, Chinese cobra produces chiefly local cytotoxicity. The neurotoxicity is essentially local paresthesia around the wound or spreading upward along but not exceeding the ipsilateral limb. Despite the benign nature in terms of absence of respiratory paralysis, the local tissue injury is often nasty and skin necrosis is common.[2] As a rule, skin necrosis is not obvious until hours to days later. Pain and swelling alone in the initial post-bite period may give a false sense of security. Ecchymosis

and blisters signify subsequent skin necrosis, which can extend well beyond the fang marks and progress over time. An inadequately disinfected wound is susceptible to bacterial infection, which aside from impairing wound may result in septicaemia. Septicaemia superimposing on extensive a necrotic skin wound was reported being the cause of death in a local patient after a Chinese cobra bite. As with bamboo snake bites, a Chinese cobra bite is frequently followed by gross pain and swelling, and rarely compartment syndrome.

Chinese cobras can spray the venom for a distance of one to two metres accurately onto the eye of its target for the purpose of defence. The dose of the venom is usually smaller than that from a bite. When the threat continues, it can spray multiple times. The cardiotoxins are responsible for the ocular damage. Intense pain and eye discharge are frequent. Corneal erosions may follow.[3] Healing is usually complete provided prompt medical care is sought.

Many-banded krait

Immediate symptoms of a many-banded krait bite include mild pain, trivial or no swelling and local paresthesia. The lethal toxins are systemic neurotoxins, both pre and post-synaptic. The pre-synaptic β-bungarotoxin inhibits release of acetylcholine from the pre-motor endplate of the synapse, through preventing synaptic vesicle endocytosis, uncoupling mitochondria during energy production and influx of intracellular calcium. On the post-synaptic membrane, the α-bungarotoxin irreversibly binds to the acetylcholine receptor. The clinical result is neuromuscular paralysis. The paralysis starts at the ocular muscles (clinically as ptosis, ophthalmoplegia and pupillary dilatation) and descends down to the face, neck, bulbar muscles and respiratory muscles. The onset of neuromuscular paralysis is typically delayed after two to three hours ensuing bite, but can be as fast as 30 minutes or as late as 12 hours. Respiratory paralysis may persist for days to weeks. Autonomic dysfunction such as transient hypertension is possible.

Point-scaled pit viper

As a pit viper, the toxicity of point-scaled pit vipers resembles that of bamboo snakes. Its venom which contains metalloproteinase, phospholipase, protease, C-type lectin etc. can induce oedema, coagulation-fibrinolysis and platelet aggregation. Its bite gives rise to pain, swelling and bleeding diathesis.[4] On the other hand the incidence and severity of skin necrosis are greater than that from bamboo snake bites. Similar to the Chinese cobra, ecchymosis and blisters are the heralding signs of necrosis.

Red-necked keelback

It is a rear fanged snake and has a pair of Duvernoy's glands. The probability of being bitten by the fangs and the capacity of injecting venom from the glands are lower than that of the front fanged snakes all described above. Yet it can elicit alarming haemotoxicity in the presence of just a mild wound swelling. The venom is a prothrombin activator, precipitating hypofibrinogenaemia and secondary fibrinolysis.[5] Extensive skin bruises, internal haemorrhage and haemorrhagic shock have been reported.

Treatments in hospital

Snake antivenom is the antidote against snakebites. The locally stocked snake antivenoms are mostly species specific. Not all snake species have the respective antivenom e.g. red-necked keelback, MacClelland's coral snake. Antivenom is generally indicated for systemic and severe local envenomation.

Supportive treatment is indispensable e.g. mechanical ventilation for respiratory paralysis, wound debridement and skin graft for necrosis, antibiotics for secondary bacterial infection. Blood products are usually not required for haemotoxic snakebites that have antivenom available because the duration of action of antivenom is ordinarily longer.

Prevention of snakebite

1. Do not wear sandals when hiking
2. Wear long trousers when hiking
3. Watch your steps in countryside
4. Minimise walking in grass or swamp
5. Poke the ground in front with a pole when moving in grass
6. Shine a torch when walking outside in dark
7. Minimise piling up objects in village house
8. Never attempt to handle, capture or kill a snake

First aid for snakebite

1. Stay calm: Hypertension and tachycardia expedites systemic absorption of venom.
2. Put the bite wound at the level of the heart: Too high a wound promotes systemic absorption of venom. Too low concentrates the venom at the wound.
3. Immobilisation: No or minimise movement of the injured limb, desirably by a splint. Muscle contraction markedly stimulates systemic absorption of venom.
4. Take off bracelet or ring from the bitten limb.
5. Take a photo of the snake at a safe distance: Never try to catch or kill the snake. This may provoke a second bite.
6. Go to hospital as quickly as possible. Many people cannot differentiate venomous from non-venomous snakes. The worst symptoms of venomous snakebites are frequently not obvious soon after the bite.

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Gas Poisoning

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Introduction

It is always challenging for a doctor to manage a patient with toxic gas poisoning. As unlike a case of overdose by ingestion, there is no straight forward history like name and amount of drugs taken which can be of much help in evaluating the severity of the case. Proper assessment of gas poisoning has to rely on taking a good history that should include gathering circumstantial evidences from all information sources and performing targeted examination based on a strong toxicology knowledge possessed by an experienced clinician. Inhalational toxins can be classified as four categories; simple asphyxiants, respiratory irritants, systemic asphyxiants and smoke. Though less common than drug poisoning in Hong Kong, gas poisoning is an important poisoning killer in Hong Kong with carbon monoxide poisoning accounted for more than one third of the poisoning deaths in the past 5 years.

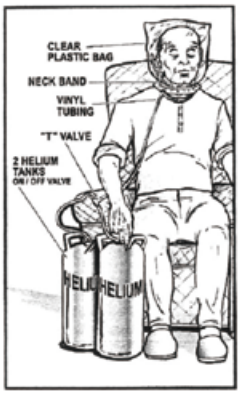

Fig. 1: Exit Bag

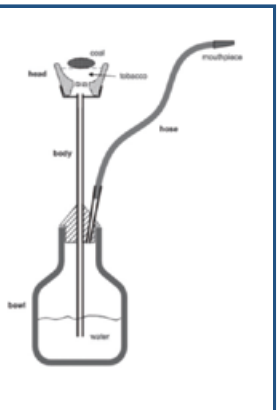

Fig. 2: Structure of Shisha smoking pipe

Simple Asphyxiants

The pathophysiology of simple asphyxiants involves simple displacement of oxygen from the lung resulting in lowering of FiO_2 and hypoxia. By definition a simple asphyxiant is non-irritating and has no direct systemic toxicity. Examples of simple asphyxiants include noble gases (e.g. helium, argon, neon), carbon dioxide, short chain aliphatic hydrocarbons (methane, ethane, butane) and nitrogen. The clinical presentation after exposure is non-specific due to the effect of hypoxia. Most cases of simple asphyxiant poisoning occur in Hong Kong are accidental in nature with the victims being workers in an enclosed area. The rising use of helium for suicide in recent years is of particular concern. How to use helium to commit suicide by creating an 'exit bag' (Fig 1) has been described in details by Derek Humphry in his book 'Final Exit: the Practicalities of Self-Deliverance and Assisted Suicide for the Dying' published in 2002.[1] Our government and other stake holders have been considering every measure to prevent this practice to become endemic.

Respiratory Irritants

Respiratory irritants form acids, bases or free radicals on contact with mucosal water and exert direct damage to the respiratory endothelium. Water solubility is the most important principle to note in understanding the clinical effects of respiratory irritants. Table 1 shows examples of respiratory irritants arranged according to their water solubility. In general high water solubility gases affect the upper airway, eyes, nose and produce fast onset of symptoms. The major risk would be upper airway compromise. Low water solubility gases affect the lower airway and produce delayed onset of symptoms. Unanticipated late deterioration can be a pitfall in managing victims inhaled low water soluble irritants like phosgene.

Table 1: Pulmonary Irritants

High Solubility	Intermediate Solubility	Low Solubility
Ammonia (NH_3)	Chlorine (Cl_2)	Phosgene ($COCl_2$)
Chloramines ($NHCl_2$)	Hydrogen Sulphide (H_2S)	Nitrogen Dioxide (NO_2)
Hydrogen Chloride (HCl)		
Hydrogen Fluoride (HF)		
Sulphur Dioxide (SO_2)		

Chlorine gas exposure is the most common respiratory irritant poisoning we encounter in our clinical practice. Chlorine is a valuable oxidising agent used in industry and disinfection. People having been to swimming pools are familiar with the smell of chlorine but it is difficult for them to differentiate between permissive and toxic levels. Chlorine can also be generated in the bathroom by mixing bleach with any acid (drain opener). In Hong Kong the death of a housewife has been reported as a result of this kind of domestic accident. Chlorine is an intermediately soluble irritant gas and produces acids when dissolves in mucosal water. Concerning treatment, neutralisation therapy by nebulised sodium bicarbonate has been advocated for chlorine gas poisoning on top of the standard measurements like oxygen and beta

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agonist. It has been proven as a safe treatment and with possible benefit.[2] It is worth noting that neutralisation therapy is in general not recommended in Toxicology except in the case of irritant gas poisoning when the potential heat generated by neutralisation can be readily absorbed by our lungs having very large surface area and heat capacity.

Systemic Asphyxiants

Systemic or tissue asphyxiants are absorbed from the lung into the circulation. They impede the utilisation of oxygen at the cellular level. Victims will suffer from cellular hypoxia with lactic acidosis. Examples of tissue asphyxiants include carbon monoxide, hydrogen sulphide and cyanide gas.

In Hong Kong the most common cause of carbon monoxide poisoning is by burning charcoal as a means of committing suicide. In 2014, according to Coroner's report, there were 345 poisoning deaths, of which 148 were due to carbon monoxide poisoning. Other than burning charcoal, carbon monoxide can be generated in any situation when combustion is incomplete. Thus carbon monoxide poisoning has to be suspected in fire victims, and symptomatic patients in enclosed areas with combustion going on like kitchens, bath rooms and cars with running engines. In the less clear cut cases, diagnosis can only be made with clinical suspicion and timely confirmatory testing of carboxyhaemoglobin level. Nowadays Shisha 水煙 (Fig. 2) smoking is becoming more popular in Hong Kong and people think that it is less harmful than smoking cigarettes. In fact the nicotine concentration in Shisha is higher than cigarettes. Shisha smokers also absorb higher concentration of carbon monoxide because of the larger volume of smoke inhaled and longer duration of smoking session. Emergency Physicians and Clinical Toxicologists are now encountering more cases of shisha smokers suffering from carbon monoxide poisoning.

Carbon monoxide is well known to cause damage to our body by its hypoxic mechanism. Carbon monoxide is rapidly absorbed and binds to our haemoglobin with 250 times greater affinity to oxygen. Haemoglobin available to bind oxygen is thus reduced according to the degree of poisoning. Carbon monoxide also binds to myoglobin and impairs the oxygen delivery to myocytes resulting in myocardial and skeletal muscle hypoxia. As other tissue asphyxiants, carbon monoxide binds to cytochrome oxidase and impairs mitochondrial utilisation of oxygen. Finally, the left shift of the oxygen dissociation curve resulted from carbon monoxide poisoning further decreases the oxygen delivery to tissues.

Carbon monoxide's inflammatory mechanism of toxicity is less commonly appreciated and less well understood. In simple terms carbon monoxide induces lipid peroxidation in our CNS with nitric oxide playing a pivotal role in the damage process. Hypoxic damage can cause immediate and persistent neurological damage. On the other hand delayed neurological sequelae (DNS) is another entity of severe carbon monoxide poisoning complication that is believed to be caused by CNS inflammation. Patients having DNS would have neurological function deteriorates after a lucid period of days to weeks and present typically with features of dementia or Parkinsonism. Though debilitating on presentation, DNS usually runs a benign course with most cases resolved in few months to one year.[3]

The mainstay of treatment for carbon monoxide poisoning is good supportive care together with supplementary high flow oxygen therapy. Oxygen reduces the half-life of carboxyhaemoglobin (Table 2). It also increases the oxygen delivery to tissues. Hyperbaric oxygen therapy (HBO) for carbon monoxide poisoning is more controversial. Though it can further decrease the carboxyhaemoglobin half-life, in practice by the time it can be arranged, the hypoxic effect of carbon monoxide has mostly been reversed. It is believed that HBO has a role in preventing delayed neurological sequelae by reducing lipid peroxidation. However studies performed have not firmly established its benefit.[4] The decision for HBO treatment has to be made on a case to case basis by the clinician, with careful consideration to balance the risks and benefits; and sometimes getting the input from the Hong Kong Poison Information Centre or Clinical Toxicologist would be helpful. Some indications for consideration of HBO are listed in Table 3. At present HBO service is provided by the Fire Services Department in Ngong Shuen Chau. There is a certain degree of risk to transfer unstable patients to receive HBO treatment. In 2018 a hospital based HBO service will commence in Pamela Youde Nethersole Eastern Hospital and it is expected that more patients will be benefited from this enhanced service.

Table 2: Half-life of Carboxyhaemoglobin

FiO_2	COHb $T_{1/2}$
Room air	Around 4 hours
High flow O2 in re-breathing mask	90 min
Hyperbaric oxygen at 3 ATM with high flow O2	23 min

Table 3: Indications for consideration of hyperbaric oxygen therapy

Evidence of end organ damage	- LOC, coma, seizure - Confusion, cognitive defects, neurological deficits - Myocardial ischaemia, arrhythmias - Persistent neurological symptoms after normobaric oxygen therapy
Evidence of end organ damage	>25% >15% in pregnant woman, child

In low concentration hydrogen sulphide acts as a respiratory irritant while in high concentration it is a tissue asphyxiant. In concentration of >700ppm, it can cause immediate knock down and death of a victim. It carries the characteristic rotten egg smell. In Hong Kong hydrogen sulphide poisoning has been infrequently reported in manhole workers. It can also occur in situations when the sewage pipe system is misconnected or damaged. Management of hydrogen sulphide poisoning is mainly supportive. As hydrogen sulphide can cause rapid knock down and death within a short period of time, prevention should be a better strategy to adopt, and rescuers safety should be emphasised. Cyanide toxicity will be covered in the next session.

Smoke inhalation

Fire accidents occur not infrequently in Hong Kong. Smoke inhalation is the major cause of death in fire accidents. However in the management of fire victims, the top priority still goes to the management of airway and ventilation. Other management considerations would include burn, hyperthermia, trauma and dehydration. Thus a multi-specialty team approach to manage fire victims would be optimal.

Smoke consists of heated air, suspended solid and liquid particles, and gas products from combustion. Gas combustion products vary according to the materials inside a building being burnt. Commonly found toxic gases generated include carbon dioxide, carbon monoxide, hydrogen sulphide, hydrogen chloride, cyanide and etc. Clinicians should consider cyanide gas poisoning in managing fire victims. Cyanide is a tissue asphyxiant that causes cellular hypoxia. The hints to cyanide gas poisoning include clinical features of hypoxia, lactic acidosis and high venous PO_2. However confirmatory tests for cyanide poisoning may not be readily available. Nowadays with the availability of hydroxocobalamin, a relatively harmless but effective cyanide antidote, in most A&E departments, treatment for suspected cyanide gas poisoning can be given more liberally based on strong clinical suspicion without the need to worry too much about the harmful effects of treatment.

Conclusion

Clinicians should consider inhalational poisoning in patients who present with non-specific signs and symptoms, especially when the incident occurs in an enclosed space or victims come as a group. Identification of the culprit gas can sometimes be achieved by asking a targeted history from the patient, and seeking help from useful information sources like job supervisor and relevant government departments. Good supportive care with close monitoring is the mainstay of care. Certain gas poisoning may require consideration of specific treatment. (Table 4)

Table 4: Principles of management of gas poisoning

1.	Ensure rescuers safety, remove victims from exposure
2.	Pre-hospital basic life support and provision of supplementary oxygen
3.	Good supportive care with continuation of oxygen therapy in hospital
4.	Identify nature or class of culprit gas and severity of exposure. Anticipate clinical course of patient
5.	Consider specific treatment for certain gas poisoning, e.g. antidote for cyanide gas poisoning

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Hong Kong Society of Breast Surgeons

Hong Kong Society of Breast Surgeons (HKSBS) was established in 2012 dedicated to develop and promote breast surgery care in Hong Kong. It is led by Dr Polly Cheung and a council made up of breast surgeons from both private and public sectors.

President:	Dr Polly Cheung		
Vice president:	Dr Chan Wing Cheong		
Honorary Secretary:	Dr Sharon Wing Wai Chan		
Honorary Treasurer:	Dr Hung Wai Ka		
Council Members:	Dr Ava Kwong	Dr Bonita Law	Dr Fiona Leung
	Dr Yuen Ho Yan	Dr Marcus Ying	

We have continued to conduct clinical meetings in collaboration with HA and private hospitals, on topics of current interest to breast surgeons. We have also introduced information on new technologies and surgical skills relevant to breast disease through courses and workshops delivered by overseas experts.

In the coming years, we will continue to organize clinical meetings, workshops and courses, bringing in new technology and encouraging experience sharing among members. Information on future activities and record of past events, please visit our website (www.hkbreastsurgeons.com).

We encourage new fellows to join to enjoy the benefit of attending future meetings and workshops.

Target members
Ordinary members: Surgeons with interest in breast surgery
Associate members: Medical health professionals who are interested in breast surgery

Antidotes

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Clinical management for poisoning primarily includes exposure termination, supportive measures, decontamination, antidote use, enhanced elimination and other specific interventions. In general, an antidote exerts its action through kinetic or dynamic interactions with the poison. The timely use of antidotes can be life-saving in certain situations such as cyanide poisoning. However, not all poisons have an antidote, and even if available, its use has to be clinically decided based on individual benefit-risk consideration. The Hong Kong Poison Information Centre (HKPIC) recorded an average of about 4,000 poisoning cases per year and about 12% of them were given antidotes as treatment[1]. Commonly used antidotes include N-acetylcysteine (NAC), naloxone, sodium bicarbonate (NaHCO3), and benzodiazepine. The antidote use pattern in Hong Kong is similar to that in the United States[2].

Fig. 1. N-acetylcysteine (NAC)

N-Acetylcysteine (NAC) (Fig. 1) is the cornerstone therapy for paracetamol poisoning. A full course of intravenous infusion starts with the initial portion (150mg/kg) given over 15 to 60 minutes. It is then followed by the 2nd portion (50mg/kg over 4 hours) and 3rd portion (100mg/kg over 16 hours). The decision for NAC treatment is generally made according to the risk assessment based dose, time of exposure, blood paracetamol level, liver function tests and clinical symptomatology. NAC induced anaphylactoid reactions such as flushing, urticarial rash, tachycardia, bronchospasm may occur in up to 20% of its recipients[3]. It is usually mild; management include temporarily stop the NAC and administration of anti-histamine and corticosteroids. NAC can be restarted at a slower rate after the reactions have subsided. Continuous NAC treatment is considered in selected cases with liver injury and detectable paracetamol level after the full course. Besides, NAC may also be used to treat other poisonings such as amatoxin, chloroform, carbon tetrachloride, acrylonitrite, cyclophosphamide, and paraquat with limited evidence. Pregnancy is not a contraindication to NAC use.

Fig. 2. Naloxone

Naloxone (Fig. 2) is a competitive opioid receptor antagonist with its primary antidote effect is to reverse CNS/respiratory depression in opioid overdose. The initial dose commonly used is 0.4mg - 2mg in adult (0.1mg/kg in children, up to 2mg) and preferably given intravenously although other routes of administration including intramuscular, intranasal have been used. For a patient who is a chronic opioid user, it is advisable to start with a low dose (0.05-0.1 mg) and titrates according to clinical response to avoid precipitating withdrawal. Repeated doses or infusion may be used since the half-life of most opioids is much longer than that of naloxone. If no response is achieved after 2mg for natural opioids (e.g. morphine) or 10mg for synthetic opioids (e.g. tramadol), opioid poisoning as the sole diagnosis is considered to be unlikely. Besides opioid poisoning, naloxone is occasionally effective in reversing the toxic effect of clonidine or other alpha-2 agonist, valproic acid and captopril overdoses. In general, use of naloxone is considered safe in opioid non-dependent patients.

NaHCO3 is an antidote with multiple indications and mechanisms. Firstly, NaHCO3 is commonly available as an 8.4% solution, which makes it essentially hypertonic sodium (even more hypertonic than hypertonic saline!). Thus, it can be used to treat poisoning caused by sodium channel blocking poisons such as tricyclic antidepressants, cocaine, propranolol and many others. For this indication, it is preferably to be given in rapid intravenously infusion (initial dose as 1-2 mEq/kg or 1-2ml/kg of 8.4% NaHCO3) or even in intravenous

bolus in life threatening situations. Besides, the bicarbonate component is able to cause serum and/or urinary alkalisation. Serum alkalisation is particularly important for tricyclic antidepressants and salicylate poisoning while urinary alkalisation can enhance the excretion of salicylate, phenobarbital, methotrexate, chlorpropamide and 2,4-dichlorophenoxyacetic acid. Repeated doses or infusion may be required in selected case with cautious on its contraindications including significant alkalaemia (pH>7.55) or sodium/fluid overload. Another less common indication for NaHCO3 use via the nebulising route is to treat chlorine gas inhalation based on limited data.

Interestingly, benzodiazepine, as a common pharmaceutical poison in overdose, can also be used as an effective antidote for stimulants poisoning (e.g. cocaine, methamphetamine), treatment for poison induced seizures, sedative of choice for most forms of poison induced agitation or neuromuscular excitation, and treatment for ethanol/sedative withdrawal. Although often discussed as a class for its antidote effect, understanding the subtle differences exists in the pharmacokinetics and pharmacodynamics for each particular benzodiazepine is useful for their optimal use. For example, midazolam is preferred if a short duration of action is needed and it also has the advantage in situations without intravenous access, intravenous lorazepam is highly effective for seizures while continuous infusion of midazolam is preferred for status epilepticus[4]. Another point worthy to note is that the dose of benzodiazepine used for treatment of stimulants poisoning can be large; a total dose of 100mg diazepam within its first few hours of management is not uncommonly encountered in our clinical practice. The pearl is close monitoring and titration with the clinical response.

Antidotes play an important role in the treatment of poisoning. The list of antidotes is diverse, ranging from some commonly used drugs such as atropine to certain rarely used, but critical drugs such as botulinum antitoxin. In Hong Kong, the Hospital Authority (HA) stocks a wide variety of antidotes at 3 levels (Table 1), which based on their frequency of use; urgency of administration when indicated; the presence of alternative antidotes and cost-effectiveness. The Chief Pharmacist's Office (CPO) reviews the list annually based on the best practice at the time of review and coordinates the procurement of the centrally maintained antidotes. Most of the rarely used antidotes are unregistered products in Hong Kong, hence special arrangement for importation is deemed necessary. To maintain the antidotes at recommended levels, stock replenishment will be required once they are used or approaching their expiration. In this connection, close monitoring of the stock status is essential.

The three levels of antidotes in HA are managed as follows: Level I antidotes (HA Acute Hospital Level) sets out the individual hospital requirement. Each hospital with Accident & Emergency Department (AED) should maintain the stock of antidotes at the recommended level. Level II antidotes (HA Cluster Level) sets out the cluster requirement. The cluster management determines the optimal stock distribution within the cluster, having regard to the anticipated demands and the time required for stock transfer (no more than 4 hours). Level III (HA Central Level) sets out the central stock requirement. The HKPIC maintains the stock and provides expert advice on the use of the antidotes in this level.

In summary, antidotes play an important role in poison management. Hong Kong is proud to have an up to date useful antidote list, which is comparable or even more comprehensive than that in the overseas counterparts.

Level I antidotes

Acetylcystesine (NAC)	Activated charcoal	Atropine	Bromocriptine
Calcium	Calcium gluconate gel	Cyproheptadine	Dantrolene
Desferrioxamine	Digoxin fab	Dimercaprol (BAL)	Dimercaptopropane sulphonate (DMPS)
Ethanol	Flumazenil	Glucagon	Idarucizumab
Intralipid 20%	Methylene blue	Naloxone	Penicillamine
Physostigmine	Polyethylene glycol	Pralidoxime chloride	Pyridoxine
Protamine	Sodium bicarbonates	Sodium nitrite	Sodium thiosulphate
Vitamin K1			

Level II antidotes

Calcium EDTA	Calcium folinate	Hydroxocobalamin	L-Carnitine
Octreotide	Phentolamine		

Antivenom		
Bungarus fasciatus 抗金環蛇蛇毒血清	Bungarus mulitcinctu / Naja naja (atra) 抗銀環蛇/抗眼鏡蛇蛇毒血清	Green Pit Viper

Level III antidotes

Acetamide	Botulism trivalent antitoxin	Fomepizole	Methionine
Prussian blue	Silibinin	Silymarin	Succimer (DMSA)

Antivenom			
Deinagkistrodon acutus 抗五步蛇毒血清	King cobra	Russell's viper	Scorpion
Stone fish	Tiger snake	Tr. muscrosquamatus + Tr. gramineus 抗龜殼花及赤尾舶蛇毒血清	

Table 1

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Scale Modelling

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Chairman, Scientific Affair Committee, Hong Kong College of Emergency Medicine

Dr Yiu-cheung CHAN

Hobbies are important in the maintaining of an individual's mental, social and physical good health. One of the popular hobbies in Hong Kong since the 1970s is scale modelling, and I am a big fan of it, particularly military scale models. Scale modelling trains my fine motor skills, patience (from none to a bit now) and other attributes and may have been one of the reasons for my choosing to be an emergency physician.

Scale modelling has a long history. In the 4-5th century BC, Greeks, Romans and Persians used scale replicas of enemy fortifications and coastal defence lines to plan battles in their years of world domination. In World War II (WWII), scale models were used to identify enemy aircraft, vessels and armoured fighting vehicles (AFV). After WWII, with the advance in technologies and materials, the industries of scale modelling blossomed. Tamiya (田宮),better known as "Double stars" (雙星) to most of us, was established in 1946. The Hong Kong Society of Scale Modelling (formerly as the Hong Kong Military Modelling Society) was established in 1972, it is the organiser of the annual Hong Kong modelling open competition since 1992. Similar to our annual medical conference, there is a main theme for each year's competition. The 24th competition will be held in November this year with the main theme of the Vietnam War.

A scale model represents its object in a certain ratio usually displayed as "1: XX". Scale modelling has various "specialties" such as airplane, train/railway, house/building, ship, and military. Common ratios in military models are 1:35/1:48 for AVF, 1:350/1:700 for naval ships, and 1:32/1:48/1:72 for aircraft. For each category, it can be further "subspecialised" according to different criteria such as nation (German/US), class (Tank/Artillery/Armored Car) or time period (pre-WWII/WWII/Modern). My favourite "subspecialty" is German self-propelled artillery in WWII. (Fig. 1)

Most scale modellers start modelling by constructing directly from the manufacturer's kit. The steps generally include planning, construction/reconstruction, and painting/weathering. Planning involves studying of the construction manual, researching the subject (e.g. Panzer IV tanks in WWII) as well as looking for fine details that may be wrong/missing in the manufacturer's kit. "House officer" or beginners in the hobby usually follow the exact steps in the construction manual. "Senior medical officers" are more experienced and may take references (Fig. 2) and add a lot of details. For the "Professors", they may scrap build part or whole of a scale model from a range of raw materials by themselves. Common raw materials for scale models are plastic, resin and metal. Other "add-on" materials are commonly brass photo-etch parts. Different tools and techniques are used for constructing models from different materials. Common basic tools include the flush cutter, craft knife similar to surgeon scalpels. (Swann-Morton craft knives are really surgical scalpels!), tweezers, sand papers, cutting mat, polystyrene cement glue and putty. More advanced tools include pliers, probes, clamps, drills (sounds like in operating theatres!), cyanoacetate glue, epoxy resin, photo-etch cutting and folding tools. Construction steps generally include cutting the parts from sprue, removing scratches and defects from the parts, gluing the parts together, filling gaps with putty, polishing, and applying add-on details materials. The use of magnifying tools is highly recommended for those modellers of "senior medical officer" grade or beyond. Last year, I found a model shop in Akihabara, Tokyo where a whole floor of it was selling all kinds of magnifying modelling tools. I am convinced of the ageing problem in Japan!

Fig. 1 - 1:35 Wespe (German self-propelled artillery in WWII)

Fig. 2 - Reference books for Panzer

After completing the basic construction/reconstruction and add-on details, the next step is painting. Over the decades, the modelling community has developed a wide range of painting and finishing techniques. However, the most important 1st step is to ascertain good ventilation; the paints are poisons! (I knew that decades ago even before I studied clinical toxicology!). Knowledge of the characteristics of different paints is important; paints contain different pigments, binders, solvents and additives, which affect solubility (oil/water), time of drying and others. Common paints used in modelling include acrylic, enamel, oil and water paints. Painting can be by the air-brush or hand brush; steps in painting involve a pre-painting wash (to remove dust and grease), use of the primer, base colour application, darker/lighter shading (to increase contrast), camouflage, detailed painting on tiny items, washes, protective layer, applying decals and unit marking (usually numbers or symbols), dry brushing and finishing with an outer protective layer. Depending on the class of models (ships, AVF or aircraft), the next step is weathering. Weathering adds a touch of reality; such as dirt and dust for AVF, rusting for ships, and all other kinds of weathering effects that happen in real life.

One of the issues in scale modelling is finding a place to display the finished models. It is usually easy for the small scale AVF or ships, however large scale models, especially aircraft in consideration of their wing size, are particularly problematic. "Mini-storage" seems to be an option before the recent tragic fire incident in Hong Kong.

Regrettably, with my entry into middle age (hopefully still be there) and onset of presbyopia a few years ago, I am now basically a scale model collector rather than a maker. Fortunately, collection is still a recognised hobby that may boost up my mental health.

Find yourself a lovely hobby and enjoy!

Fig. 3 - 1:700 IJN Fuyuzuki (Japan Destroyer in WWII) - Before Painting
[To show the use of add-on materials to improve details]

Fig. 4 - 1:700 KMS Prinz Engen (German Heavy Cruiser in WWII)

Fig. 5 - 1:35 Panzer IIA (German tank in WWII)

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
		★FMSHK Officers' Meeting **1**	★HKMA Central, Western & Southern Community Network - Certificate Course on Dermatology (Session 5) - Cosmetic Dermatology for GP **2**	★HKMA Hong Kong East Community Network - The Importance of DM Nephropathy ★HKMA Kowloon East Community Network - Chemotherapy for Metastatic Prostate Cancer – How Early to Start? **3**	★Joint Surgical Symposium - Nerve Monitor in Thyroid Surgery ★HKMA Shatin Doctors Network - Common Sports Injury in Upper Limb ★HKMA Yau Tsim Mong Community Network - Certificate Course on Urology (Session 1) - PSA and TRUS Biopsy **4**	**5**
★HKMA Swimming Gala **6**	**7**	★HKMA Kowloon West Community Network - First 1000 Days of Allergy Prevention ★MPS Workshop - Achieving Safer and Reliable Practice ★HKMA Council Meeting **8**	★Hong Kong Neurosurgical Society Monthly Academic Meeting –Image Guided DBS **9**	★HKMA Hong Kong East Community Network - Seminar on Management of Common Breastfeeding Problems: What Primary Care Doctors Need to Know and Practice? ★HKMA KECN, HKCFP & UCH - Certificate Course for GPs 2016 (Session 5): Management of Degenerative Joint Diseases ★HKMA Structured CME Programme with HKS&H Session 10: Anaesthesiologists as Perioperative Physicians **10**	★HKMA Shatin Doctors Network - Sarcopenia in Elderly ★HKMA Yau Tsim Mong Community Network - Certificate Course on Urology (Session 2) - Better LUTS Management, Better Days for Your Patients **11**	★24th Annual Scientific Meeting of Hong Kong College of Radiologists ★Refresher Course for Health Care Providers 2016/2017 ★MPS Workshop - Achieving Safer and Reliable Practice **12**
★24th Annual Scientific Meeting of Hong Kong College of Radiologists ★Post-stroke Management: Integrative Medicine **13**	**14**	**15**	**16**	★HKMA Hong Kong East Community Network - Update on GERD Management ★HKMA New Territories West Community Network - Lecture Series on Clinical Oncology (Session 1): Recent Advancement Cancer Treatment: Immunotherapy - PD-1/PD-L1 Pathway ★MPS Workshop - Mastering Shared Decision Making Workshop ★FMSHK Executive Committee Meeting ★FMSHK Council Meeting ★FMSHK 31st Auunal General Meeting ★HKFMS Foundation 17th Auunal General Meeting **17**	★HKMA Yau Tsim Mong Community Network - Certificate Course on Urology (Session 3) - A New Step for the Past 30 Years OAB Management **18**	★18th Beijing/Hong Kong Medical Exchange **19**
★18th Beijing/Hong Kong Medical Exchange **20**	**21**	★HKMA Kowloon West Community Network - Better LUTS Management, Better Days for Your Patients **22**	**23**	**24**	★HKMA Yau Tsim Mong Community Network - Certificate Course on Urology (Session 4) - Microscopic Hematuria and Stone Disease **25**	**26**
★HKMA Family Sports Day **27**	**28**	★HKMA Tai Po Community Network - Overview of Psoriasis and Treatment Options **29**	**30**			

Date / Time	Function	Enquiry / Remarks
1 TUE 8:00PM	**FMSHK Officers' Meeting** Organiser: The Federation of Medical Societies of Hong Kong; Venue: Gallop, 2/F, Hong Kong Jockey Club Club House, Shan Kwong Road, Happy Valley, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
2 WED 1:00 PM	**HKMA Central, Western & Southern Community Network - Certificate Course on Dermatology (Session 5) - Cosmetic Dermatology for GP** Organiser: HKMA Central, Western & Southern Community Network; Chairman: Dr. CHAN Hau Ngai, Kingsley; Speaker: Dr. CHAN Hin Lee, Henry; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
3 THU 1:00 PM	**KMA Hong Kong East Community Network - The Importance of DM Nephropathy** Organiser: HKMA Hong Kong East Community Network; Chairman: Dr. AU Chi Lap, Simon; Speaker: Dr. LAM Man Fai; Venue: The Hong Kong Management Association, Room 201, 2/F, Pico Tower, 66 Gloucester Road, Wan Chai, Hong Kong	Ms. Candice TONG Tel: 2527 8285 1 CME Point
1:00 PM	**HKMA Kowloon East Community Network - Chemotherapy for Metastatic Prostate Cancer – How Early to Start?** Organiser: HKMA Kowloon East Community Network; Chairman: Dr. AU Ka Kui, Gary; Speaker: Dr. CHAN Leung Cho; Venue: Lei Garden Restaurant (利苑酒家), Shop no. L5-8, apm, Kwun Tong, No. 418 Kwun Tong Road, Kwun Tong, Kowloon	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
4 FRI 8:00 AM	**Joint Surgical Symposium - Nerve Monitor in Thyroid Surgery** Organiser: HKMA Organizers: Department of Surgery, The University of Hong Kong & Hong Kong Sanatorium & Hospital; Venue: Hong Kong Sanatorium & Hospital; Chairman: Dr. Brian LANG; Speakers: Professor LO Chung-Yau, Dr. Brian LANG	Department of Surgery, Hong Kong Sanatorium & Hospital Tel: 2835 8698 Fax: 2892 7511 1 CME Point
1:00 PM	**HKMA Shatin Doctors Network - Common Sports Injury in Upper Limb** Organiser: HKMA Shatin Doctors Network; Chairman: Dr. MAK Wing Kin; Speaker: Dr. LAU Yan Kit; Venue: Chairman Room, Level 2, Royal Park Hotel, 8 Pak Hok Ting Street, Shatin, Hong Kong	Ms. Ivy LEE Tel: 2895 9730 1 CME Point
1:00 PM	**HKMA Yau Tsim Mong Community Network - Certificate Course on Urology (Session 1) - PSA and TRUS Biopsy** Organiser: HKMA Yau Tsim Mong Community Network; Chairman: Dr. LEE Wai Lun; Speaker: Dr. FU Kam Fung, Kenneth; Venue: Pearl Ballroom, Level 2, Eaton, Hong Kong, 380 Nathan Road, Kowloon	Ms. Candice TONG Tel: 2527 8285 1 CME Point
6 SUN 2:00 PM	**HKMA Swimming Gala** Organiser: The Hong Kong Medical Association; Venue: Michael Clinton Swimming Pool, Hong Kong Polytechnic University, 30 Renfrew Rd, Kowloon Tong, Kowloon	Mr. Ian KWA Tel: 2527 8285
8 TUE 1:00 PM	**HKMA Kowloon West Community Network - First 1000 Days of Allergy Prevention** Organiser: HKMA Kowloon West Community Network; Chairman: Dr. CHAN Siu Man, Bernard; Speaker: Dr. CHIU Cheung Shing, Daniel; Venue: Crystal Room IV-V, 3/F., Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan, N.T.	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
6:30 PM	**MPS Workshop - Achieving Safer and Reliable Practice** Organiser: The Hong Kong Medical Association & Medical Protection Society; Speaker: Dr. CHENG Ngai Shing, Justin; Venue: Eaton Hotel, 380 Nathan Road, Yau Ma Tei, Kowloon	HKMA CME Dept. Tel: 2527 8452 2.5 CME Points
9:00 PM	**HKMA Council Meeting** Organiser: The Hong Kong Medical Association; Chairman: Dr. CHOI Kin; Venue: HKMA Wanchai Premises, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Ms. Christine WONG Tel: 2527 8285
9 WED 7:30AM	**Hong Kong Neurosurgical Society Monthly Academic Meeting –Image Guided DBS** Organizer: Hong Kong Neurosurgical Society; Chairman: Dr CHAN Tat Ming, Danny; Speaker: Dr YEUNG Kam Tong; Venue: Seminar Room, G/F, Block A, Queen Elizabeth Hospital	Dr. LEE Wing Yan, Michael Tel: 2595 6456 Fax. No.: 2965 4061 1.5 points College of Surgeons of Hong Kong
10 THU 1:00 PM	**HKMA Hong Kong East Community Network - Seminar on Management of Common Breastfeeding Problems: What Primary Care Doctors Need to Know and Practice?** Organiser: HKMA Hong Kong East Community Network and Primary Care Office of the Department of Health; Chairman: Dr. YIP Yuk Pang, Kenneth; Speaker: Dr. FOK Oi Ling, Annie; Venue: The Hong Kong Management Association, Room 201, 2/F, Pico Tower, 66 Gloucester Road, Wan Chai, Hong Kong	Ms. Candice TONG Tel: 2527 8285 1 CME Point
1:00 PM	**HKMA KECN, HKCFP & UCH - Certificate Course for GPs 2016 (Session 5): Management of Degenerative Joint Diseases** Organiser: HKMA Kowloon East Community Network & Hong Kong College of Family Physicians & United Christian Hospital; Chairman: Dr. David CHAO; Speaker: Dr. HO Hon Shuen;Venue: Conference Room, G/F, Block K,United Christian Hospital (UCH), 130 Hip Wo Street, Kwun Tong, Kowloon	Ms. Polly TAI / Ms. Cordy WONG Tel: 3949 3430 (Ms. TAI) / 3949 3087 (Ms. WONG) 1 CME Point
2:00 PM	**HKMA Structured CME Programme with HKS&H Session 10: Anaesthesiologists as Perioperative Physicians** Organiser: The Hong Kong Medical Association & Hong Kong Sanatorium & Hospital; Speaker: Dr. KUA Seng Wee, Jeffrey; Venue: Function Room A, HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	HKMA CME Dept. Tel: 2527 8452 1 CME Point
11 FRI 1:00 PM	**HKMA Shatin Doctors Network - Sarcopenia in Elderly** Organiser: HKMA Shatin Doctors Network; Chairman: Dr. MAK Wing Kin; Speaker: Dr. CHAN Chun Chung; Venue: Jasmine Room, Level 2, Royal Park Hotel, 8 Pak Hok Ting Street, Shatin, Hong Kong	Ms. Mandy LO Tel: 2859 8759 1 CME Point
1:00 PM	**HKMA Yau Tsim Mong Community Network - Certificate Course on Urology (Session 2) - Better LUTS Management, Better Days for Your Patients** Organiser: HKMA Yau Tsim Mong Community Network; Chairman: Dr. CHAN Wai Keung, Ricky; Speaker: Dr. WONG Kwok Tin, Martin; Venue: Pearl Ballroom, Level 2, Eaton, Hong Kong, 380 Nathan Road, Kowloon	Ms. Candice TONG Tel: 2527 8285 1 CME Point

Date / Time	Function	Enquiry / Remarks
12 SAT 8:30AM *(13)*	**24th Annual Scientific Meeting of Hong Kong College of Radiologists** Organiser: Hong Kong College of Radiologists; Venue: Hong Kong Academy of Medicine Jockey Club Building, 99 Wong Chuk Hang Road, Aberdeen, HKSAR, China	Tel: 2871 8787 Fax: 2871 8898 Email: hkcr@hkam.org.hk
2:15 PM	**Refresher Course for Health Care Providers 2016/2017** Organiser: Hong Kong Medical Association & HA-Our Lady of Maryknoll Hospital; Speaker: Dr. KWAN Wing Shan, Iris; Venue: Training Room II, 1/F, OPD Block, Our Lady of Maryknoll Hospital, 118 Shatin Pass Road, Wong Tai Sin, Kowloon	Ms. Clara TSANG Tel: 2354 2440 2 CME Points
2:30 PM	**MPS Workshop - Achieving Safer and Reliable Practice** Organiser: The Hong Kong Medical Association & Medical Protection Society; Speaker: Dr. CHENG Ngai Shing, Justin; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept. Tel: 2527 8452 2.5 CME Points
13 SUN 2:30 PM	**Post-stroke Management: Integrative Medicine** Organizer: Hong Kong Association for Integration of Chinese-Western Medicine; Speaker(s): (1) Prof. Shen Jiangang (2) Dr. Peng Zengfu (3) Prof. Pang Yiu Chung (4) Dr. Lau Yuk Lun (5) Dr/CMP Or Ka Hang; Chairman: Dr Yu Chau Leung and Prof. Cheung Tak Fai; Venue: Lecture Theatre, M/F, HA Building, 147B Argyle Street, Kowloon	Miss Y.C. Yeung Tel: 3119 1858 Fax: 2301 2414 Point(s) Pending CME accreditation by 3 points CME (CMP)
17 THU 1:00 PM	**HKMA Hong Kong East Community Network - Update on GERD Management** Organiser: HKMA Hong Kong East Community Network; Chairman: Dr. AU YEUNG Shiu Hing; Speaker: Dr. HSU Shing Jih, Axel; Venue: The Hong Kong Management Association, Room 201, 2/F, Pico Tower, 66 Gloucester Road, Wan Chai, Hong Kong	Ms. Candice TONG Tel: 2527 8285 1 CME Point
1:00 PM	**HKMA New Territories West Community Network - Lecture Series on Clinical Oncology (Session 1): Recent Advancement Cancer Treatment: Immunotherapy - PD-1/PD-L1 Pathway** Organiser: HKMA New Territories West Community Network; Speaker: Dr. CHEUNG Foon Yiu; Venue: Atrium Function Rooms, Lobby Floor, Hong Kong Gold Coast Hotel, 1 Castle Peak Road, Gold Coast, Hong Kong	Miss Hana YEUNG Tel: 2527 8285
6:30 PM	**MPS Workshop - Mastering Shared Decision Making Workshop** Organiser: The Hong Kong Medical Association & Medical Protection Society; Speaker: Dr. Fung Shu Yan, Anthony; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept. Tel: 2527 8452 2.5 CME Points
7:00 PM	**FMSHK Executive Committee Meeting** Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
7:00 PM	**FMSHK Council Meeting** Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
8:00 PM	**FMSHK 31st Auunal General Meeting** Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
8:30 PM	**HKFMS Foundation 17th Auunal General Meeting** Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898

Date / Time	Function	Enquiry / Remarks
18 FRI 1:00 PM	**HKMA Yau Tsim Mong Community Network - Certificate Course on Urology (Session 3) - A New Step for the Past 30 Years OAB Management** Organiser: HKMA Yau Tsim Mong Community Network; Chairman: Dr. LAM King Hei, Stanley; Speaker: Dr. WONG Chun Wing, Simon; Venue: Pearl Ballroom, Level 2, Eaton, Hong Kong, 380 Nathan Road, Kowloon	Ms. Candice TONG Tel: 2527 8285 1 CME Point
19 SAT (20)	**18th Beijing/Hong Kong Medical Exchange** Organiser: The Hong Kong Medical Association & Chinese Medical Association; Chairman: Dr. CHAN Nim Tak, Douglas; Speaker: Various Venue: Versailles Ballroom, 3/F, Regal Kowloon Hotel, 71 Mody Rd, Tsim Sha Tsui East, Hong Kong	Miss Ellie FU Tel: 2527 8285 8 CME Points
22 TUE 1:00 PM	**HKMA Kowloon West Community Network - Better LUTS Management, Better Days for Your Patients** Organiser: HKMA Kowloon West Community Network; Chairman: Dr. LAM Ngam, Raymond; Speaker: Dr. WONG Ming Ho, Edmond; Venue: Crystal Room IV-V, 3/F., Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan, N.T.	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
25 FRI 1:00 PM	**HKMA Yau Tsim Mong Community Network - Certificate Course on Urology (Session 4) - Microscopic Hematuria and Stone Disease** Organiser: HKMA Yau Tsim Mong Community Network; Chairman: Dr. LAM Tzit Yuen, David; Speaker: Dr. SO Chun; Venue: Pearl Ballroom, Level 2, Eaton, Hong Kong, 380 Nathan Road, Kowloon	Ms. Candice TONG Tel: 2527 8285 1 CME Point
27 SUN 1:30 PM	**HKMA Family Sports Day** Organiser: The Hong Kong Medical Association; Venue: Stanley Ho Sports Centre, 10 Sha Wan Drive, Sandy Bay, Hong Kong	Miss Denise KWOK Tel: 2527 8285
29 TUE 1:45 PM	**HKMA Tai Po Community Network - Overview of Psoriasis and Treatment Options** Organiser: HKMA Tai Po Community Network; Speaker: Dr. LEE Tsz Yan, Samson; Venue: Chiuchow Garden Restaurant(潮江春) Shop 001-003, 1/F, Uptown Plaza (新達廣場), No.9 Nam Wan Road, Tai Po	Miss Hana YEUNG Tel: 2527 8285 1 CME Point

Federation News

Federation President Cup Basketball Tournament 2016

The Federation President Cup Basketball Tournament for 2016 was held at the Wanchai Southorn Stadium on 5 & 12 June 2016. There were eight participating teams, including the Federation's Invitational Team, Sanofi-Aventis Hong Kong Ltd, Hong Kong Medical Association, Hong Kong Urological Association, Hong Kong Medical Supplies Ltd, Pfizer Corporation Hong Kong Ltd, Hong Kong Clinical Psychologists Association and Jacobson Pharma Corporation Ltd.

This year, the Federation brought a new exhibition match to the tournament by inviting the Presidents / CEOs from each team to shoot five times at the free throw line. Dr Eliza Cheung, Chairperson of HKCPA won the game and 16 FMSHK T-shirts for all her team members. After two days of competition, Pfizer successfully defended their title to become the basketball champion of 2016.

We congratulate the winning teams and express our sincere gratitude for the support from all the participating teams and guests. We look forward to seeing you again at the Federation President Cup Soccer Five & Basketball Tournament in 2017!

Photographs taken at the match have been uploaded onto the Federation's website http://www.fmshk.org

The results of the Basketball Tournament were as follows:

Champion	**Pfizer Corporation Hong Kong Ltd**
1st Runner Up	**Jacobson Pharma Corporation Ltd**
2nd Runner Up	**Hong Kong Clinical Psychologists Association**
Top Scorer	**Mr Sui-lun NG, Jacobson Pharma Group Ltd**

FMSHK Annual Scientific Meeting 2016

On 3 July 2016, the Federation of Medical Societies of Hong Kong successfully held the Annual Scientific Meeting 2016 at the Sheraton Hotel and Towers, with the theme of "Holistic Care in the Era of Specialty Based Medicine".

A total of 15 medical talks were delivered by a panel of distinguished speakers. They shared with us the latest developments on Holistic Care through a wide range of topics including Cardiovascular Disease, Diabetes, Paediatric Epilepsy, Allergy, Autistic, Geriatrics, as well as Oncology, all of which aimed to enhance the knowledge on treatment or palliation for these advanced conditions.

FMSHK was much privileged to have the Officiating Guest, Dr Donald Li, President of Hong Kong Academy of Medicine; and the Honourable Guests, Dr the Hon LEONG Che-hung, GBM, GBS, OBE, JP; Prof the Hon Joseph LEE, PhD, RN, SBS, JP, Legislative Councillor (Health Services); Dr the Hon LEUNG Ka-lau, Legislative Councillor (Medical); Dr Mario CHAK, President of FMSHK and Dr Raymond Lo, Immediate Past President of FMSHK to officiate the opening ceremony.

Dr Donald LI and Dr Mario CHAK led all the guests to read out the key message - ***"To achieve disciplinary, integrative & personalized medicine, providing full physical and psychosocial support!"*** to signify the opening of the Annual Scientific Meeting 2016.

We would like to take this opportunity to express our sincere gratitude to our Officiating Guest, Honourable Guests, Co-chairmen, Chairpersons and Speakers for their contributions that made the event a great success. Our gratitude also extends to various sponsors for their generous support. We look forward to seeing you in our Annual Scientific Meeting in 2017!

Opening Ceremony

Session 1 - Cardiovascular Disease and Metabolic Syndrome

Session 2 - Diabetes Mellitus

Lunch Symposium - Allergy Prevention

Session 3 - Paediatric Epilepsy

Session 4a - Allergy and Autistic Spectrum Disorder

Session 4b - Geriatrics

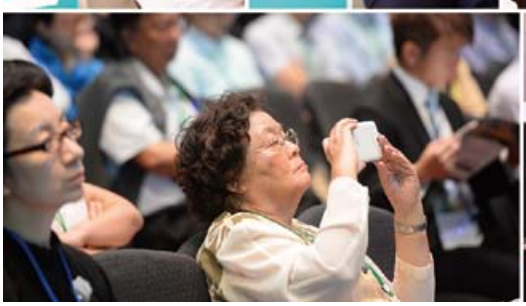
Session 5a - HIV Infection and Mental Health




Session 5b - Oncology








Sponsors




香港醫學組織聯會基金「香港晚期病患及寧養紓緩服務」意見調查結果

意見調查目的

鑑於公眾社會對晚期病患者的需求及選擇越見關注，香港醫學組織聯會基金早前進行一項有關「晚期病患及寧養紓緩服務」的意見調查，希望透過是次調查向公眾及醫護人員索取意見，藉此提高社會各界對寧養紓緩服務的認知，及向晚期病患者提供醫療服務及保障的關注。

調查分兩個階段進行，第一階段由香港醫學組織聯會基金委託香港中文大學傳播與民意調查中心，以隨機抽樣的方式，利用電腦輔助電話訪問系統，由訪問員進行電話訪問，成功個案共775個; 第二階段調查由香港醫學組織聯會基金，以郵寄方式將同一份問卷寄發，對象主要為香港註冊的醫生及牙醫，成功個案共799個。

調查結果

Ⅰ. 寧養紓緩服務

「寧養紓緩服務」指為晚期病患者提供全面的身心照顧服務，包括症狀控制、身體護理、情緒和心靈輔導等。

Q1. 你有否聽過「寧養紓緩服務」?

	公眾	醫療界別
有	14.80%	74.09%
沒有	84.50%	21.28%
不知道	0.70%	3.00%
拒絕回答	0.00%	0.13%
空白或多於一個答案	0.00%	1.50%
總和	100.00%	100.00%

Q2. 你認為香港社會為「寧養紓緩服務」提供的資源足夠嗎?

	公眾	醫療界別
足夠	4.20%	0.75%
一般	20.80%	15.27%
不足夠	50.60%	65.96%
不知道	24.40%	16.77%
拒絕回答	0.00%	0.25%
空白或多於一個答案	0.00%	1.00%
總和	100.00%	100.00%

Q3. 你認為「寧養紓緩服務」對晚期病患者重要嗎?

	公眾	醫療界別
重要	86.30%	91.49%
一般	7.90%	3.38%
不重要	1.00%	0.13%
不知道	4.60%	3.88%
拒絕回答	0.20%	0.25%
空白或多於一個答案	0.00%	0.88%
總和	100.00%	100.00%

Q4. 你認為現時由公立醫院提供的寧養紓緩服務足夠嗎?

	公眾	醫療界別
足夠	4.70%	1.00%
一般	25.90%	14.89%
不足夠	47.10%	65.83%
不知道	22.30%	16.90%
拒絕回答	0.00%	0.38%
空白或多於一個答案	0.00%	1.00%
總和	100.00%	100.00%

Q5. 你認為現時由私營機構提供的寧養紓緩服務足夠嗎?

	公眾	醫療界別
足夠	5.70%	0.75%
一般	29.5%	12.14%
不足夠	29.4%	55.94%
不知道	35.3%	29.66%
拒絕回答	0.1%	0.63%
空白或多於一個答案	0.00%	0.88%
總和	100.00%	100.00%

Q6. 你了解「寧養紓緩服務」是怎樣幫助晚期病患者嗎?

	公眾	醫療界別
了解	8.30%	30.54%
一般	28.50%	45.43%
不了解	61.20%	17.40%
不知道	1.80%	5.26%
拒絕回答	0.20%	0.25%
空白或多於一個答案	0.00%	1.13%
總和	100.00%	100.00%

Q7. 你清楚可以從甚麼途徑接觸「寧養紓緩服務」嗎?

	公眾	醫療界別
清楚	8.10%	16.77%
一般	17.60%	33.67%
不清楚	72.20%	39.42%
不知道	2.00%	8.51%
拒絕回答	0.10%	0.25%
空白或多於一個答案	0.00%	1.38%
總和	100.00%	100.00%

Q8. 你認為晚期病患者最適合在那裡休養及度過餘生?

	公眾	醫療界別
家中	52.90%	53.94%
醫院	8.80%	0.88%
寧養院	33.20%	31.16%
不知道	4.90%	4.38%
拒絕回答	0.20%	1.13%
空白或多於一個答案	0.00%	8.51%
總和	100.00%	100.00%

Q9. 你(醫療界別)認為自己最適合在那裡休養及度過餘生？

	醫療界別
家中	67.33%
醫院	0.75%
寧養院	18.90%
不知道	6.63%
拒絕回答	1.75%
空白或多於一個答案	4.63%
總和	100.00%

Q10. 你認為現時社會上為晚期病患者提供的家居支援服務足夠嗎?

	公眾	醫療界別
足夠	3.7%	0.63%
一般	21.6%	10.89%
不足夠	60.9%	75.09%
不知道	13.8%	11.64%
拒絕回答	0.00%	0.50%
空白或多於一個答案	0.00%	1.25%
總和	100.00%	100.00%

Q11. 整體來說，你認為現時的保險計劃對晚期病患者在經濟及生活需要上的保障足夠嗎？

	公眾	醫療界別
足夠	6.30%	0.38%
一般	26.50%	7.88%
不足夠	52.50%	71.84%
不知道	14.30%	17.77%
拒絕回答	0.40%	0.88%
空白或多於一個答案	0.00%	1.25%
總和	100.00%	100.00%

Q12. 對於要照顧晚期病患者的家人，你認為香港現時為他們提供的支援足夠嗎？

	公眾	醫療界別
足夠	3.20%	0.25%
一般	25.50%	10.39%
不足夠	62.50%	79.10%
不知道	8.60%	9.01%
拒絕回答	0.20%	0.63%
空白或多於一個答案	0.00%	0.63%
總和	100.00%	100.00%

調查結果

II.「預前醫療指示」

「預前醫療指示」是病人預先為自己作出醫療決定，例如晚期病患在生命危急時是否搶救。

Q13. 你聽過「預前醫療指示」嗎?

	公眾	醫療界別
有	12.10%	71.84%
沒有	86.50%	23.28%
不知道	1.40%	4.01%
拒絕回答	0.00%	0.25%
空白或多於一個答案	0.00%	0.63%
總和	100.00%	100.00%

Q14. 假如你有嚴重病患，你會考慮作出預前醫療指示嗎?

	公眾	醫療界別
會	80.10%	88.36%
不會	11.30%	2.25%
不知道	8.30%	7.88%
拒絕回答	0.30%	0.88%
空白或多於一個答案	0.00%	0.63%
總和	100.00%	100.00%

Q15. 你贊成政府為「預前醫療指 示」立法，令其具有法律效力，即是醫護人員及家人必須遵從病人的預前醫療指示嗎？

	公眾	醫療界別
贊成	62.70%	67.33%
一般	22.10%	12.64%
不贊成	11.30%	11.76%
不知道	3.80%	5.88%
拒絕回答	0.10%	1.75%
空白或多於一個答案	0.00%	0.63%
總和	100.00%	100.00%

總結

總結1 - 市民對「寧養紓緩服務」認知不足

1. 多達八成公眾未曾聽過「寧養紓緩服務」，近七成半醫療界別人士聽過。
2. 逾六成市民不了解「寧養紓緩服務」是怎樣幫助晚期病患者，只有30.5%醫療界別人士了解、45.4%醫療界別人士對「寧養紓緩服務」有一般了解。

總結2 - 認同「寧養紓緩服務」重要普遍認為服務及資源不足

1. 有九成受訪者認為「寧養紓緩服務」對晚期病患者重要。
2. 有六成受訪者認為現時由公立醫院提供的寧養紓緩服務不足。
3. 有四成半受訪者認為現時由私營機構提供的寧養紓緩服務不足。
4. 有六成受訪者認為香港社會為「寧養紓緩服務」提供的資源不足。

總結3 - 希望在家中度過餘生但照顧者支援不足

1. 逾五成受訪者認為晚期病患者最適合在家裡休養及度過餘生。
2. 有七成受訪者認為現時社會上為晚期病患者提供的家居支援服務不足夠。
3. 有六成受訪者認為現時的保險計劃對晚期病患者在經濟及生活需要上的保障不足夠。
4. 有七成受訪者認為香港現時為照顧晚期病患者的家人所提供的支援並不足夠。

總結4 - 建議「預前醫療指示」具有法律效力

1. 只有一成多公眾人士聽過「預前醫療指示」，而醫療界別則有逾七成受訪者聽過。
2. 面對自己有嚴重病患時，有八成半受訪者會考慮作出預前醫療指示。
3. 逾六成受訪者贊成政府為「預前醫療指示」立法，令其具有法律效力，即是醫護人員及家人必須遵從病人的預前醫療指示。

本會誠邀各界就香港晚期病患及寧養紓緩服務調查結果，提出寶貴的建議。歡迎與本會秘書處胡小姐聯絡。

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Answers to Dermatological Quiz

Answer:

1. The nodules spread along the lymphatic drainage under the skin. It is sometimes described as sporotrichoid lymphocutaneous spread and commonly happens on the upper limbs. It often starts distally on the hand or wrist after a minor injury or insect bite. Then the lesions arise proximally along the lymphatic vessels in a roughly linear distribution.

2. The sporotrichoid lymphocutanous spread is often caused by uncommon infections. Organisms commonly causing sporotrichoid spread are Sporothrix schenckii, Mycobacterium marinum, Nocardia and Leishmaniasis. Other deep fungal infections like blastomycosis, histoplasmosis, cryptococcosis and coccidioidomycosis may also cause sporotrichoid spread.

3. Sporotrichoid spread is often started from trivial trauma or injuries. So this kind of history should be asked although most patients could not recall it. It is more commonly seen in immunocompromised patients. Therefore, a history like malignancy, organ transplant, HIV infection or on immunosuppressive agents are important. Further occupational history is also important because most of these organisms are found in the rural area. Thus, farmers, gardeners, landscapers, fishermen are at higher risks.

4. Skin biopsy for histology, tissue for acid-fast bacilli culture and tissue for fungal culture are useful in confirming the diagnosis and identifying the causative organism. Test for HIV test if the patient's status is not known.

5. Sporotrichosis and other fungal infections can be treated by itraconazole. For atypical mycobacterial infections, one may use rifampicin or minocycline. Minocycline or cotrimoxazole can be used for Nocardia. Leishmania may be treated by stibogluconate.

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