

## The severity and prognostic markers of 148 cases of carbon monoxide poisoning by burning charcoal

### 148 宗燒碳引致一氧化碳中毒個案的嚴重程度及預後指標

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**Objective:** To identify and analyse the characteristics of carbon monoxide (CO) poisoning due to burning charcoal in our locality. **Methods:** This was a 3-year retrospective study. All patients presenting with CO poisoning by burning charcoal from August 1999 to December 2002 were recruited. The demographic data, initial vital signs, blood results, treatment regimens and outcomes were collected. Association between clinical outcomes and parameters were calculated. **Results:** The study identified 148 patients (mean age 34.7 years) suffering from CO poisoning by burning charcoal. One hundred and forty-six cases (98.6%) were suicidal. Twenty-five patients (16.9%) were unconscious (GCS  $\leq$  8) on arrival. The mean initial carboxyhaemoglobin (COHb) level was 21.0%. The mortality rate was 5.4%. Fifteen cases (10.1%) required intensive care. Twelve cases (8.1%) had neurological complications and five (3.4%) suffered from delayed neurological sequelae. Initial blood results showing hyperkalemia and acidosis were associated with likelihood of unconsciousness on arrival ( $p = 0.007$ ,  $p = 0.019$  respectively). Hyperkalemia and unconsciousness on arrival were associated with longer hospital stay ( $p < 0.001$ ,  $p < 0.001$  respectively) as well as likelihood of systemic complication ( $p < 0.001$ ,  $p < 0.001$  respectively). There was no relationship between co-ingestion, age, initial COHb level, initial systolic and diastolic blood pressure with consciousness level on arrival ( $p = 0.188$ ,  $p = 0.846$ ,  $p = 0.264$ ,  $p = 0.224$ ,  $p = 0.755$  respectively). Age, initial COHb level, acidosis, initial systolic and diastolic blood pressure did not correlate with the duration of hospital stay ( $p = 0.679$ ,  $p = 0.176$ ,  $p = 0.501$ ,  $p = 0.313$ ,  $p = 0.868$  respectively). **Conclusion:** Suicide almost accounted for all the CO poisonings by burning charcoal in our study group. It caused significant mortality and morbidity. Hyperkalemia, unconscious state and acidosis had prognostic values. (*Hong Kong j.emerg.med.* 2006; 13:6-16)

**目的：**是次研究旨在識別及分析本地燒碳引致一氧化碳中毒個案的特色。**方法：**這是一個為期三年的回顧性研究，招募由 1999 年 8 月至 2002 年 12 月期間因燒碳引致一氧化碳中毒而求診的所有病者；從而收集病人統計數據，初步生命表徵，驗血報告，療程及結果，並計算臨床結果及指標間的關聯。**結果：**研究識別 148 名病者（平均年齡 34.7 歲）因燒碳引致一氧化碳中毒，其中 146 個案（98.6%）為自殺，25 名病者（16.9%）抵院時昏迷（格拉斯哥昏迷指數  $\leq$  8）。最初之碳氧血紅蛋白平均水平為 21.0%，死亡率為 5.4%，15 個案（10.1%）需接受深切治療護理；12 個案（8.1%）有神經上的併發症，其中 5 名（3.4%）更有延遲出現的神經病後遺症。初次驗血結果出現高鉀血症及酸中毒與抵院時昏迷的可能性有關聯（分別為  $p = 0.007$ ，及  $p = 0.019$ ）。高鉀血症及抵院時昏迷與較長住院期（分別為  $p < 0.001$ ，

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$p < 0.001$ ) 及全身性併發症的可能性有關聯 (分別為  $p < 0.001$ ,  $p < 0.001$ ) ; 但同時服藥、年齡、最初之碳氧血紅蛋白水平、最初之收縮及舒張血壓與抵院時清醒程度無關係 (分別為  $p = 0.188$ ,  $p = 0.846$ ,  $p = 0.264$ ,  $p = 0.224$ ,  $p = 0.755$ )。年齡、最初之碳氧血紅蛋白水平、酸中毒、最初之收縮及舒張血壓與住院期並不相關 (分別為  $p = 0.679$ ,  $p = 0.176$ ,  $p = 0.501$ ,  $p = 0.313$ ,  $p = 0.868$ )。總結: 研究者群組中, 近乎全部燒炭引致一氧化碳中毒者皆因自殺所致, 它引致重大的病態和死亡率。高鉀血症、昏迷狀態及酸中毒等有預後的價值。

**Keywords:** Acidosis, hospitalisation, hyperkalemia, length of stay, unconsciousness

**關鍵詞:** 酸中毒、住院、高鉀血症、住院期、昏迷

## Introduction

Burning charcoal is a unique way of suicide in Asia.<sup>1</sup> People commit suicide by burning charcoal in a closed or poorly ventilated space. Carbon monoxide (CO) is a colourless and odourless gas formed from incomplete combustion of charcoal. Burning charcoal, as a method of suicide, was widely spread by the mass media after the first reported case in Hong Kong in November 1998.<sup>1,2</sup> The incidence then rose rapidly and scored the third most common mode of suicide in Hong Kong in 1999 within two months (the first was jumping from height and the second was hanging).<sup>3,4</sup> By 2001, burning charcoal was the second most common mode of suicide in Hong Kong.<sup>5</sup> Unfortunately, burning charcoal might also be involved in homicide.<sup>6</sup>

CO poisoning is a common problem worldwide. In the US, it accounts for 15,000 emergency department attendances and 500 deaths annually for unintentional and non-fire related CO exposure.<sup>7</sup> In the UK, unintentional CO poisoning accounts for 50 deaths per year.<sup>8</sup>

Studies on the clinical aspects of CO poisoning by burning charcoal in our locality were lacking. This study aimed at disclosing the pattern of this unique form of poisoning.

## Methods

This was a retrospective study. All cases of burning charcoal attending our emergency department, serving a population of approximately one million, from

1 August 1999 to 31 December 2002 were recruited. The emergency department (ED) records and the hospital records were analysed. Demographic data included age, sex, reasons for burning charcoal and co-incident substance ingestion. Initial vital signs were defined as the first set of vital signs documented on the ED record, including blood pressure, pulse rate, Glasgow Coma Scale (GCS) and ECG rhythm. Hypertension was defined as either systolic blood pressure  $\geq 140$  mmHg or diastolic blood pressure  $\geq 90$  mmHg. Hypotension was defined as either systolic blood pressure  $< 90$  mmHg or diastolic blood pressure  $< 60$  mmHg. Blood pressure was measured by manual or automatic sphygmomanometer. Heart rates  $> 100$  bpm and  $< 60$  bpm were regarded as tachycardia and bradycardia respectively. Heart rate and rhythm were documented by 12-lead ECG or rhythm strips recorded by cardiac monitor.  $GCS \leq 8$  was chosen as the cut-off point for unconsciousness. All patients were managed at the resuscitation room after arrival; blood samples were drawn and tested for carboxyhaemoglobin (COHb) level, arterial blood gas, electrolytes, renal and liver functions. Information regarding blood results (including pH, potassium and COHb), modes of oxygen therapy (hyperbaric or normobaric), patient flow (from admission to discharge), in-hospital complications and length of hospital stay, were collected and analysed.

All the data were analysed using the software SPSS for Windows version 11.0. The Mann-Whitney and chi-squared tests were applied to compare the conscious state with clinical features. The Mann-Whitney test and the independent samples t-test were applied to compare clinical outcomes with the duration of

hospital stay. Multiple regression was used to identify clinical features associated with the duration of hospital stay;  $p \leq 0.05$  was defined as statistically significant.

**Results**

**Demographic data**

During the period of study, there were 148 cases of CO poisoning by burning charcoal. The mean age was 34.7 years (SD 11.4, minimum 7 months, maximum 78 years) (Figure 1). Eighty patients (54.1%) were male and 68 (45.9%) were female (male : female, 1.2 : 1).

One hundred and forty-six cases (98.6%) were suicidal. For the two non-suicidal cases, the first was a 7-month-old infant, who suffered from CO poisoning as a result

of his mother committing suicide by burning charcoal at home. The second case involved a 39-year-old mentally ill patient using charcoal as fuel for a barbecue at home.

There were 82 cases (55.4%) with co-incident substance ingestion. Alcohol and benzodiazepines were commonly used substances. Fourteen cases (9.5%) had ingested more than one substance (Figure 2).

**Initial vital signs**

Figures 3 and 4 show the haemodynamic state of the cases. Ninety-five cases (64.2%) had normal blood pressure on arrival. Cardiac arrhythmias were found in 55.4% of cases. The most common rhythm was sinus tachycardia (46.6%). Twenty-five cases (16.9%) were unconscious (GCS  $\leq 8$ ) whereas 84 cases (56.8%) were fully alert (GCS = 15) on arrival (Figure 5).

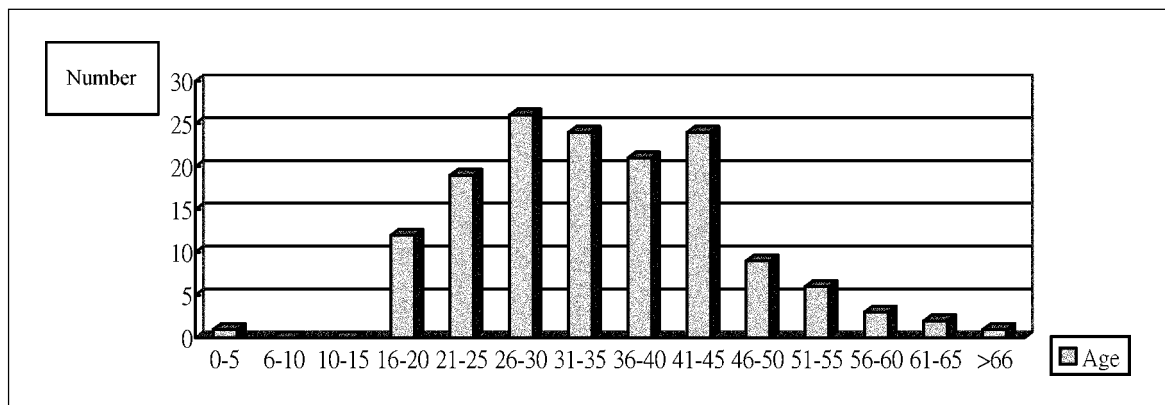


Figure 1. Age distribution.

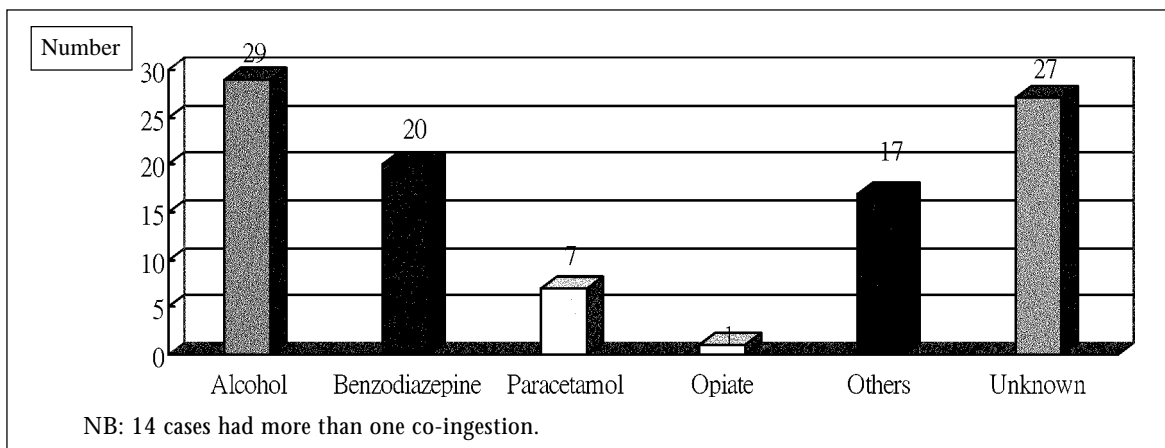


Figure 2. Distribution of co-incident xenobiotic ingestion.

**Blood result**

Blood samples were not drawn in one case of death before arrival with post-mortem changes and five cases of death on arrival without initial vital signs. The diagnoses of burning charcoal in these cases were based

on information from the police or the ambulance staff at the scene. Eight cases had their arterial blood gas and COHb levels checked but not electrolytes. Those cases of death before arrival and death on arrival without initial vital signs and missing data were excluded in the statistical analysis. All the remaining patients had elevated COHb levels with the mean result of 21.0% (SD 14.71) (Figure 6). Figures 7 and 8 demonstrate the distribution of the initial pH and potassium level respectively. Initial hyperkalemia (potassium > 5.1 mmol/L) and acidosis (pH < 7.35) were found to be associated with the likelihood of an unconscious state on arrival at the ED ( $p = 0.007$  and  $p = 0.019$  respectively) (Table 1). There was no association between co-ingestion, age, initial COHb level, systolic and diastolic blood pressure and initial conscious state ( $p = 0.188$ ,  $p = 0.846$ ,  $p = 0.264$ ,  $p = 0.224$ , and  $p = 0.755$  respectively) (Tables 1 & 2).

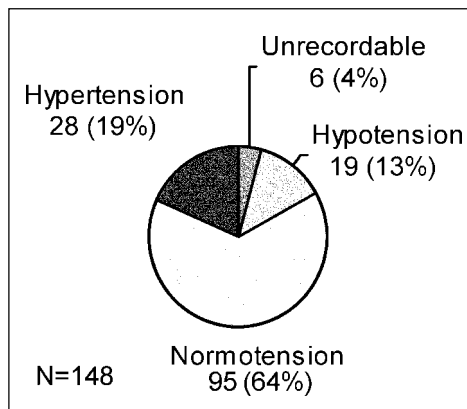
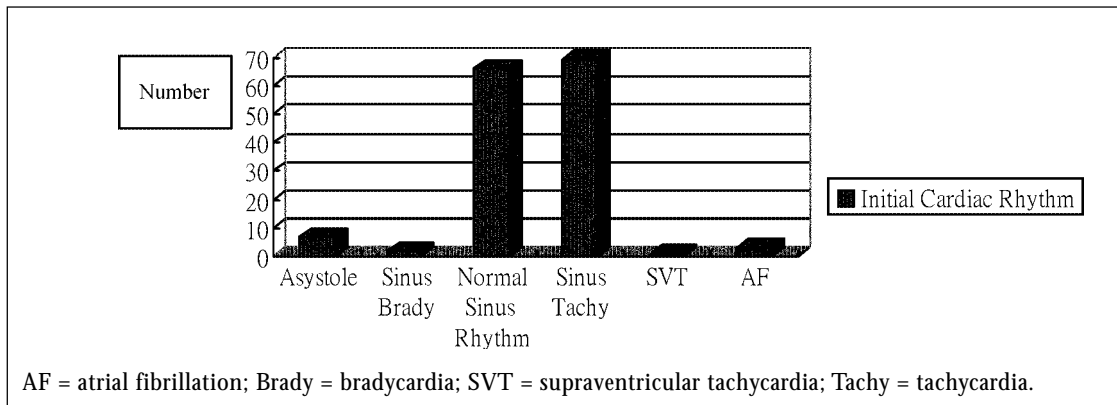


Figure 3. Distribution of initial blood pressure.



AF = atrial fibrillation; Brady = bradycardia; SVT = supraventricular tachycardia; Tachy = tachycardia.

Figure 4. Initial cardiac rhythm.

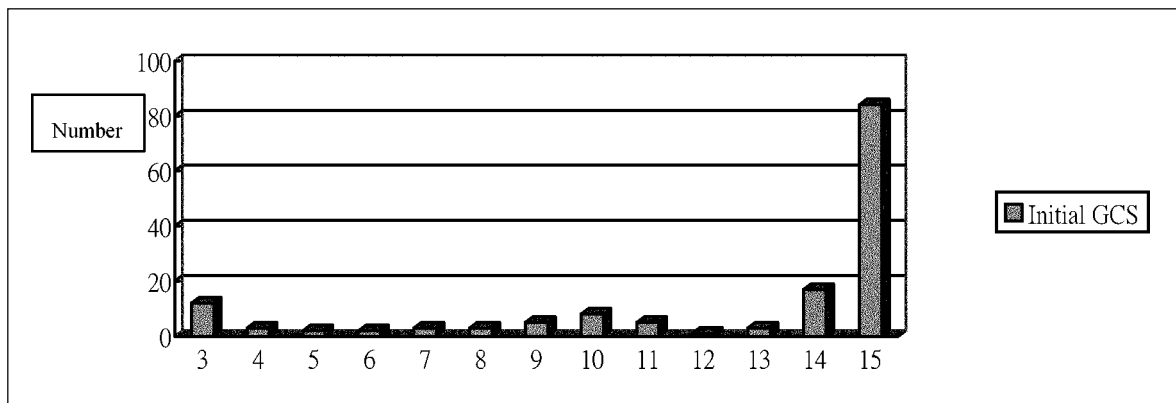


Figure 5. Initial Glasgow Coma Scale (GCS) score.

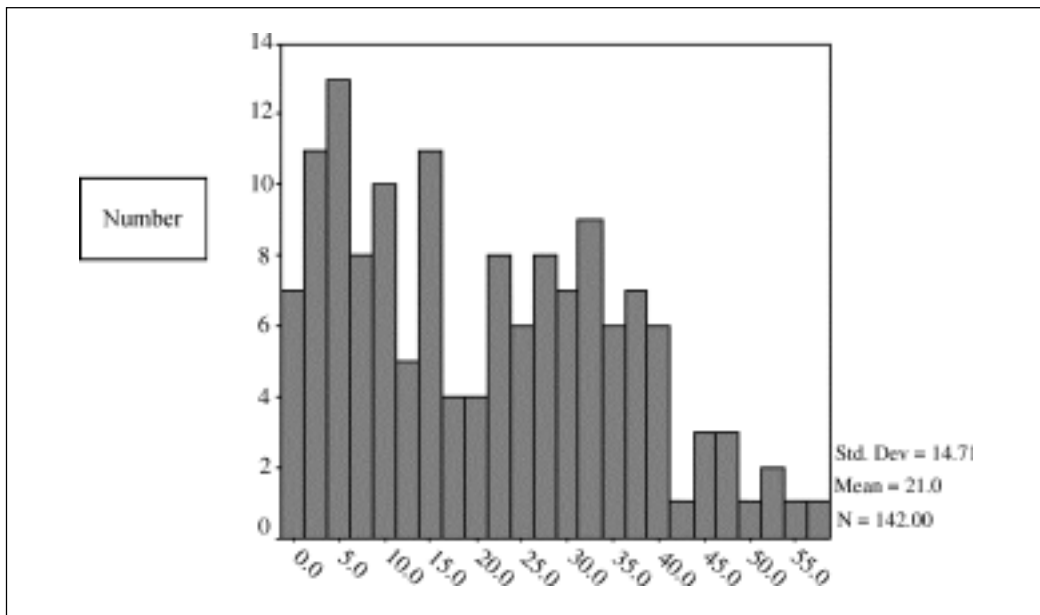


Figure 6. The initial carboxyhaemoglobin level (%).

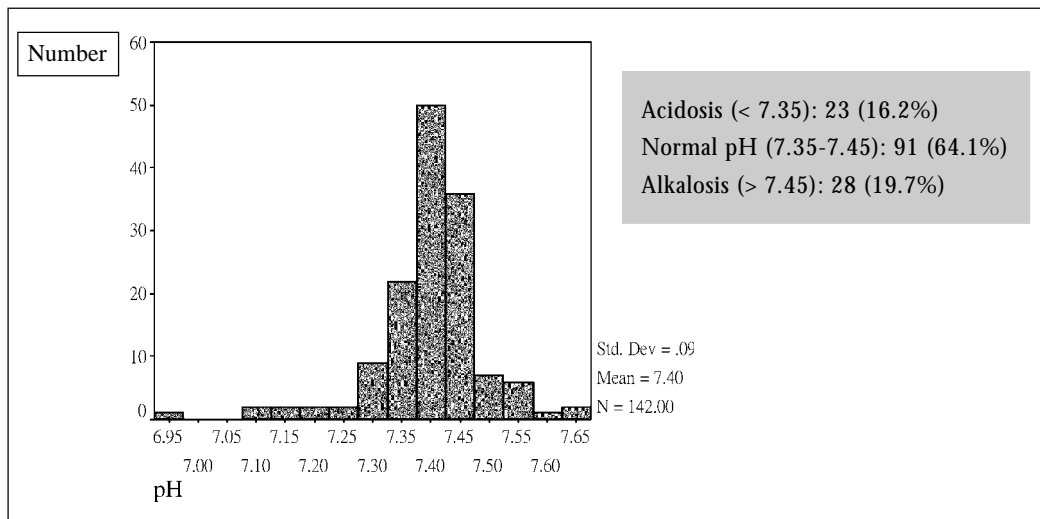


Figure 7. Initial pH.

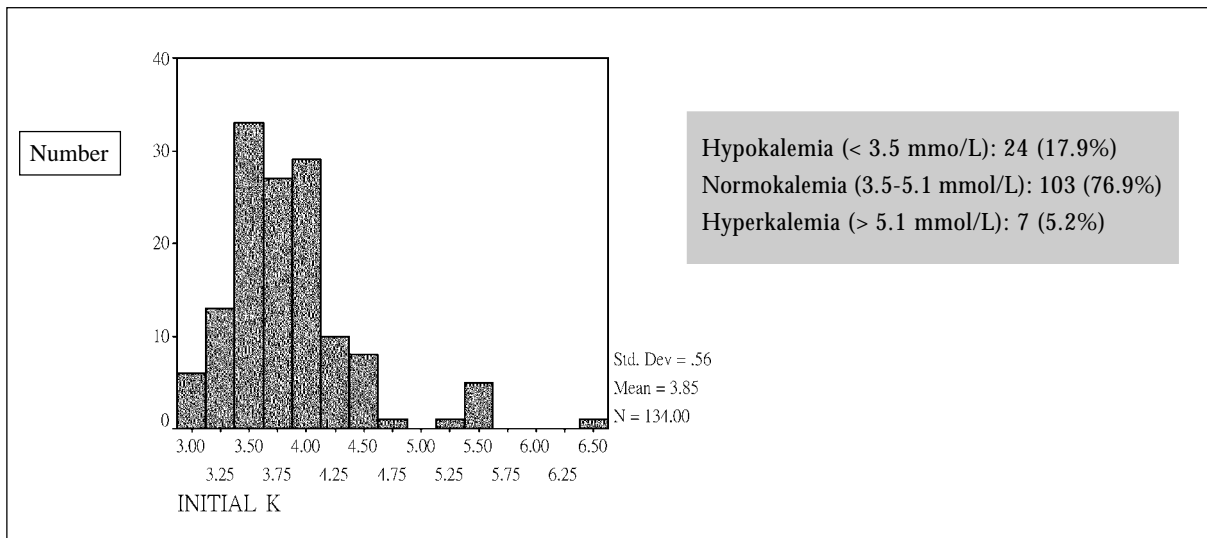
**Treatment**

All patients were given high-flow oxygen during the pre-hospital stage. They were managed at the resuscitation room immediately on arrival at the hospital. Twenty-four cases (16.2%) required mechanical ventilation, five (3.4%) of them had no initial vital sign and required immediate resuscitation. One hundred and twenty-seven cases (85.8%) received 100% or nearly 100% normobaric oxygen. Eighteen cases (12.2%) received hyperbaric oxygen therapy. Two patients (1.4%) refused oxygen treatment. No

treatment was offered to one case (0.7%) with postmortem changes on arrival.

**Patient outcome**

The mortality was 5.4% (8 cases). One (0.7%) was dead before arrival with post-mortem changes. Five death cases (3.4%) arrived at the ED without initial vital signs and failed to respond to active resuscitation. Two cases (1.4%) succumbed during hospital stay. Fifteen cases (10.1%) required intensive care. One hundred and forty-two (95.9%) were admitted to



**Figure 8.** Initial potassium level (mmol/L).

**Table 1.** Clinical parameters and conscious state

| Parameter                         | Consciousness   | Unconsciousness | p-value |
|-----------------------------------|-----------------|-----------------|---------|
| Hyperkalemia (K > 5.1 mmol/L)     | 2/7 (28.6%)     | 5/7 (71.4%)     | 0.007*  |
| Non-hyperkalemia (K ≤ 5.1 mmol/L) | 113/127 (89.0%) | 14/127 (11.0%)  |         |
| Acidosis (pH < 7.35)              | 17/23 (73.9%)   | 6/23 (26.1%)    | 0.019*  |
| Non-acidosis (pH ≥ 7.35)          | 106/119 (89.1%) | 13/119 (10.9%)  |         |
| Co-ingestion                      | 63/77 (81.8%)   | 14/77 (18.2%)   | 0.188** |
| No co-ingestion                   | 60/65 (92.3%)   | 5/65 (7.7%)     |         |

\*Mann-Whitney test; \*\*Chi-squared test

**Table 2.** Clinical parameters not associated with conscious state (N = 142)

| Parameter                       | Conscious state | Mean  | Standard deviation | p-value (Mann-Whitney test) |
|---------------------------------|-----------------|-------|--------------------|-----------------------------|
| Age                             | Conscious       | 34.5  | 11.93              | 0.846                       |
|                                 | Unconscious     | 35.8  | 11.33              |                             |
| Initial COHb (%)                | Conscious       | 24.9  | 16.04              | 0.264                       |
|                                 | Unconscious     | 20.6  | 14.46              |                             |
| Systolic blood pressure (mmHg)  | Conscious       | 112.6 | 34.93              | 0.224                       |
|                                 | Unconscious     | 121.8 | 16.82              |                             |
| Diastolic blood pressure (mmHg) | Conscious       | 71.2  | 23.24              | 0.755                       |
|                                 | Unconscious     | 75.0  | 13.54              |                             |

COHb = carboxyhaemoglobin

hospital after stabilisation at the ED. Twenty-three cases (15.5%) were transferred to the psychiatric hospital after medical treatment (Figure 9). The mean hospital stay was 5.2 days (SD 7.46, mode 3, median 3, 25th percentile 3, 50th percentile 3, 75th percentile 5).

Twenty-seven complications (excluding death before admission) were identified in 20 cases (13.5%). Twelve patients (8.1%) had neurological complications including five cases of delayed neurological sequelae (DNS) and five cases of hypoxic brain damage. Other complications were rhabdomyolysis, acute renal failure, disseminated intravascular coagulation, post-cardiac arrest, pulmonary embolism, acute duodenal ulcer, and chest infection (Table 3).

In the analysis of the relationship between clinical outcome or clinical parameters and duration of hospital

stay, we excluded one case of death before arrival, five cases of death on arrival without initial vital signs and one case of extreme outlier (a case of hypoxic brain damage hospitalised for 73 days mainly for rehabilitation). The presence of serious neurological complications (DNS and hypoxic brain damage), systemic complications (neurological, respiratory, renal, haematological, gastrointestinal and death after admission) and necessity for mechanical ventilation were associated with longer duration of hospital stay ( $p < 0.001$ ,  $p < 0.001$  and  $p < 0.001$  respectively) (Table 4). The presence of co-ingestion was not associated with longer hospital stay ( $p = 0.375$ ) (Table 4). Using multiple regression, hyperkalemia and unconsciousness on arrival were shown to be associated with longer hospital stay ( $p < 0.001$ , and  $p < 0.001$  respectively;  $r^2 = 0.253$ ). Age, initial COHb level, initial acidosis, systolic and diastolic blood pressure bore no statistically

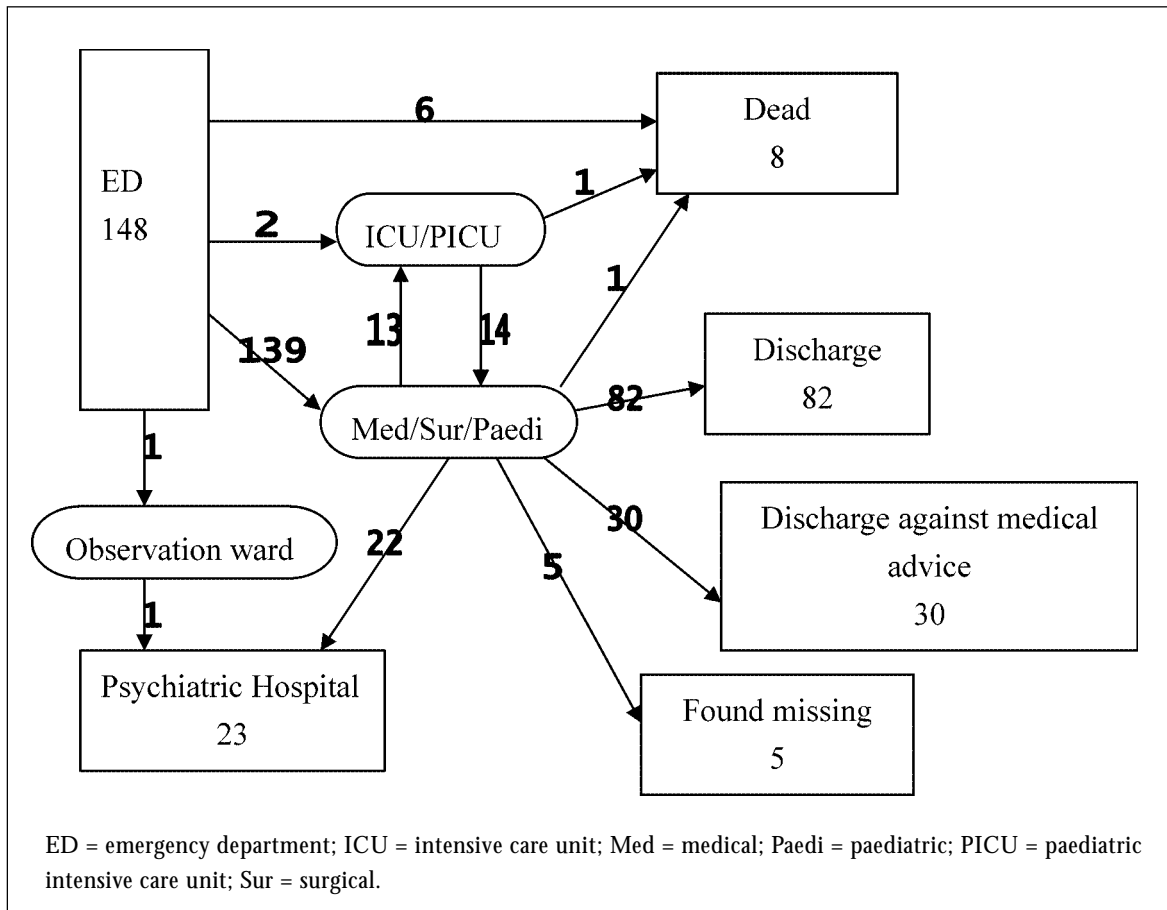


Figure 9. Patient flow from admission to discharge.

significant relationship with the duration of hospital stay ( $p = 0.679$ ,  $p = 0.176$ ,  $p = 0.501$ ,  $p = 0.313$ , and  $p = 0.868$  respectively) (Table 5).

### ***Relationship between parameter and clinical outcome***

Table 6 illustrates that hyperkalemia and unconsciousness were associated with systemic complications mentioned in Table 3 ( $p < 0.001$  and  $p < 0.001$  respectively), but

not acidosis ( $p = 0.062$ ). Hyperkalemia, acidosis and unconsciousness were associated with DNS ( $p < 0.001$ ,  $p = 0.027$  and  $p < 0.001$  respectively) (Table 7).

## **Discussion**

Our study included 148 patients with definite history of burning charcoal resulting in CO poisoning. There

**Table 3.** Complications during hospital stay\*

| <b>Complication</b>                    |                               | <b>Number of case</b> |
|--|-------------------------------|-----------------------|
| Neurological                           | Delayed neurological sequelae | 5                     |
|  | Hypoxic brain damage          | 5                     |
|  | Right hemiplegia              | 1                     |
|  | Peripheral nerve palsy        | 2                     |
| Rhabdomyolysis                         |                               | 5                     |
| Acute renal failure                    |                               | 2                     |
| Disseminated intravascular coagulation |                               | 1                     |
| Post-cardiac arrest                    |                               | 2                     |
| Pulmonary embolism                     |                               | 1                     |
| Chest infection                        |                               | 2                     |
| Acute duodenal ulcer                   |                               | 1                     |

\*5 patients had more than one complication

**Table 4.** Comparison of length of hospital stay with clinical outcome (n = 141)†

|  | <b>Length of hospital stay (days)</b> |           |               | <b>p-value</b> |
|--|---------------------------------------|-----------|---------------|----------------|
|  | <b>Mean</b>                           | <b>SD</b> | <b>Median</b> |                |
| DNS or hypoxic brain damage (9/141)      | 11.4                                  | 6.0       | 12            | < 0.001*       |
| No DNS or hypoxic brain damage (132/141) | 4.3                                   | 4.4       | 3             |                |
| Systemic complications (18/141)          | 11.2                                  | 9.5       | 10            | < 0.001*       |
| No complication (123/141)                | 3.8                                   | 2.6       | 3             |                |
| Intubated (18/141)                       | 11.4                                  | 9.4       | 9             | < 0.001*       |
| Not intubated (123/141)                  | 3.7                                   | 2.6       | 3             |                |
| Co-ingestion (76/141)                    | 4.4                                   | 3.2       | 3             | 0.375**        |
| No co-ingestion (65/141)                 | 5.1                                   | 6.1       | 3             |                |

DNS = delayed neurological sequelae

\*Mann-Whitney test ; \*\*Independent samples t-test; † Excluded in the analysis: 5 cases of death on arrival, 1 case of death before arrival and 1 case of extreme outlier

**Table 5.** Correlation of clinical parameters with hospital stay using multiple regression (n = 141)\*

| Clinical parameter              | p-value | Regression coefficient (B) | 95% CI (B)    |
|---------------------------------|---------|----------------------------|---------------|
| Initial hyperkalemia            | < 0.001 | 3.192                      | 1.865–4.520   |
| Unconscious state               | < 0.001 | -0.338                     | -0.542--0.134 |
| Age                             | 0.679   | 0.032                      | --            |
| Initial COHb                    | 0.176   | 0.105                      | --            |
| Initial acidosis                | 0.501   | -0.055                     | --            |
| Systolic blood pressure (mmHg)  | 0.313   | -0.033                     | --            |
| Diastolic blood pressure (mmHg) | 0.868   | 0.047                      | --            |

Constant (B) = -3.193; 95% CI = -9.526-3.140

Coefficient of determination ( $r^2$ ) = 0.253

COHb = carboxyhaemoglobin

\*Excluded in the analysis: 5 cases of death on arrival, 1 case of death before arrival and 1 case of extreme outlier

**Table 6.** Systemic complication rates in relation to clinical parameters

|                                   | Systemic complication (%) | No systemic complication (%) | p-value (Chi-squared test) |
|-----------------------------------|---------------------------|------------------------------|----------------------------|
| Hyperkalemia (K > 5.1 mmol/L)     | 5/7 (71.4%)               | 2/7 (28.6%)                  | < 0.001                    |
| Non-hyperkalemia (K ≤ 5.1 mmol/L) | 14/127 (11.0%)            | 113/127 (89.0%)              |                            |
| Acidosis (pH < 7.35)              | 8/23 (34.8%)              | 15/23 (65.2%)                | 0.062                      |
| Non-acidosis (pH ≥ 7.35)          | 11/119 (9.2%)             | 108/119 (90.8%)              |                            |
| Unconsciousness                   | 8/19 (42.1%)              | 11/19 (57.9%)                | < 0.001                    |
| Consciousness                     | 11/123 (8.9%)             | 112/123 (91.1%)              |                            |

**Table 7.** Rate of delayed neurological sequelae (DNS) in relation to clinical parameters

|                                   | DNS          | No DNS          | p-value (Chi-squared test) |
|-----------------------------------|--------------|-----------------|----------------------------|
| Hyperkalemia (K > 5.1 mmol/L)     | 1/7 (14.3%)  | 6/7 (85.7%)     | < 0.001                    |
| Non-hyperkalemia (K ≤ 5.1 mmol/L) | 4/127 (3.1%) | 123/127 (96.9%) |                            |
| Acidosis (pH < 7.35)              | 3/23 (13.0%) | 20/23 (87.0%)   | 0.027                      |
| Non-acidosis (pH ≥ 7.35)          | 2/119 (1.7%) | 117/119 (98.3%) |                            |
| Unconsciousness                   | 3/19 (15.8%) | 16/19 (84.2%)   | < 0.001                    |
| Consciousness                     | 2/123 (1.6%) | 121/123 (98.4%) |                            |

was a wide range of severity from mild dizziness to respiratory arrest or death. We mainly focused on the initial presentations, blood results and their relationship to the hospital stay. Turner et al reported that initial acidosis predicted longer treatment in carbon monoxide poisoning. They conducted a retrospective review of 40 cases of carbon monoxide poisoning treated with hyperbaric oxygen. They found that patients receiving more than one hyperbaric

oxygen therapy session had significantly higher initial hydrogen ion concentrations than those who recovered from one session only.<sup>9</sup> CO poisoning appeared to result in hypoxia at the cellular level.<sup>10</sup> Acidosis and hyperkalemia, markers of significant tissue hypoxia, were found by our study to be associated with an unconscious state. Unconsciousness indicates severe carbon monoxide poisoning and is suggestive for hyperbaric oxygen therapy in adults and children,<sup>11,12</sup>

and it is considered imperative in pregnant women.<sup>13</sup> Eighteen cases (12.2%) in our study group received hyperbaric oxygen therapy. The effectiveness of hyperbaric oxygen therapy in the prevention of neurological sequelae is controversial.<sup>14</sup> Although there is no evidence for routine hyperbaric oxygen therapy, there may be benefits in those severely poisoned.<sup>14</sup> Recent animal researches favoured a new biochemical marker, S100B over unconsciousness in predicting survival.<sup>15</sup>

According to the coroner's report published by the Hong Kong Government, 'the numbers of suicidal deaths by CO poisoning (commonly called "burning charcoal") in Hong Kong for year 1999, 2000, 2001 and 2002 were 89, 154, 250 and 252 respectively'. This mode of suicide was first singled out in the coroner's report in 2000. In comparison, the suicidal deaths for the years 1999, 2000, 2001 and 2002 were 882, 915, 988 and 1,025 respectively.<sup>16</sup> There is a huge difference between the rate of mortality in our study group and the government's figures (8 vs. 745). The most important reason was that most of the deaths were sent to the government mortuary directly instead of the ED. An important message is that the 5% mortality rate in our study group might give the public and medical personnel a false impression that burning charcoal is not a highly lethal mode of suicide, but in fact, it causes significant mortality.

Worse clinical outcomes (DNS, hypoxic brain damage, systemic complications and necessity for mechanical ventilation) were associated with a longer duration of hospital stay (Table 4). Unconsciousness and hyperkalemia were associated with a longer hospital stay and higher systemic complication rates (Tables 5 & 6). As there were only five cases of DNS in our study group; the validity of its association with clinical parameters (hyperkalemia, acidosis and unconsciousness), though statistically significant, was questionable (Table 7).

Half of our study group had co-ingestion. We observed that co-ingestion was not associated with longer hospital stay or unconsciousness. Unfortunately, the documentation of co-ingestion details (e.g. amount of co-ingestion, duration of ingestion) was incomplete

and poor. Some of the substances, for example salicylate, can cause acid-base abnormalities that would confound the results.

The mortality and morbidity rates (5.4% and 13.5% respectively) were substantial. Five of our cases (3.4%) suffered DNS while other epidemiological studies revealed DNS incidences of 10–30%.<sup>13</sup> The lack of detailed neurological examination and neuropsychiatric tests during the acute management and follow-up sessions might result in a failure to pick up subtle changes. Our actual morbidity might be higher.

Our study only concentrated on part of the problems of intentional carbon monoxide poisoning by burning charcoal. Future prospective studies should assess the effectiveness of hyperbaric oxygen therapy, neuropsychiatric syndromes, the use of neuropsychiatric tests and biochemical markers.

## Conclusion

Suicide accounted for nearly all CO poisonings by burning charcoal in our study group. Burning charcoal caused significant mortality and morbidity. Initial hyperkalemia and unconsciousness were good prognostic markers, while initial acidosis might have prognostic values.

## Acknowledgement

The authors are grateful to Dr. Sammy PS Ng, Centre for Health Protection, Department of Health, for providing information on burning charcoal mortality in Hong Kong and Ms. WY Sung, statistician of Tuen Mun Hospital, in providing advice in statistical work.

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