ORIGINAL ARTICLE

Dettol poisoning and the need for airway intervention

CME

PK Lam 林沛堅
CK Chan 陳志強
ML Tse 謝萬里
FL Lau 劉飛龍

Objectives To (1) characterise the clinical features of Dettol poisoning on a territory-wide basis, (2) assess the need for airway intervention after such poisoning and its time frame after ingestion, and (3) identify predictors for such an intervention.

Design Case series.

Setting Sixteen accident and emergency departments in Hong Kong.

Patients Patients with Dettol ingestion who presented within 48 hours of ingestion from July 2005 to June 2009, derived from the database of the Hong Kong Poison Information Centre.

Results In all, 213 patient records were identified, of which 36 were excluded based on pre-defined criteria and 177 were analysed. Among the latter, the median age was 32 (range, 2-95) years and the male-to-female ratio was 1:2.7 (48:129). Intentional ingestion constituted the majority (95%) of cases. The most common symptoms were related to the local irritative/corrosive effects on the aero-digestive tract, such as gastro-intestinal upset and localised throat pain. Airway intervention was required in 14 (8%) patients. All interventions were performed within 12 hours of Dettol ingestion and three cases involved re-intubation after extubation. Univariate analysis showed that a Glasgow Coma Scale score of <8, older age, a larger amount ingested, lip swelling, lung crackles, and wheezing were all associated with airway intervention. In the multivariate analysis using forward stepwise logistic regression, only coma (Glasgow Coma Scale score of <8) remained statistically significant.

Conclusions Delayed airway obstruction (>12 hours after Dettol ingestion) is unlikely. For those who are intubated, careful assessment of airway adequacy before extubation is strongly recommended to avoid extubation failure and subsequent re-intubation. Patients in coma (Glasgow Coma Scale score of <8) should prompt airway intervention.

New knowledge added by this study

- Airway intervention was required in 8% of patients who ingested Dettol.
- Delayed airway obstruction (>12 hours post-ingestion) was unlikely.
- Coma (Glasgow Coma Scale score of <8) was significantly associated with airway intervention.

Implications for clinical practice or policy

- For those deemed not to require airway intervention at presentation, close monitoring of the upper airway for oedema for 24 hours is probably sufficient.
- For intubated patients, careful assessment of airway adequacy before extubation is strongly recommended in order to avoid extubation failure and re-intubation.

Key words
Chloroxylenol; Disinfectants/poisoning; Intubation, intratracheal; Risk factors

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Accident and Emergency Department, Pamela Youde Nethersole Eastern Hospital, Chai Wan, Hong Kong
PK Lam, FHKAM (Emergency Medicine), Dip Clin Tox
Hong Kong Poison Information Centre, United Christian Hospital, Kwun Tong, Kowloon, Hong Kong
CK Chan, FHKAM (Emergency Medicine), Dip Clin Tox
ML Tse, FRCS (Edin), FHKAM (Emergency Medicine)
FL Lau, FRCS (Edin), FHKAM (Emergency Medicine)

Correspondence to: Dr CK Chan
Email: chanck3@ha.org.hk

Introduction

Dettol, a mixture of 4.8% chloroxylenol, 9% pine oil, and 12% isopropyl alcohol, is a popular household disinfectant in Hong Kong. It is also commonly involved in self-poisoning and has been reported in 10% of self-poisoning–related hospital admissions locally.[1] Ingestion of Dettol can cause central nervous system depression ranging from drowsiness to coma, irritation or corrosion of the aero-digestive tract, aspiration, pneumonia, adult respiratory distress syndrome (ARDS), as well as cardiopulmonary arrest.[2-4] Upper airway obstruction is of particular concern as it is immediately life-threatening and has been reported in many case reports of Dettol ingestion.[5,6]

Some authors have suggested close monitoring of the airway for at least 48 hours post-ingestion, based on the report of a single patient in which upper airway obstruction developed approximately 48 hours after admission.[7] However, a review of that case showed that it actually entailed post-extubation laryngeal oedema, ie upper airway obstruction developed after extubation, which could also occur without Dettol ingestion. For patients who do not undergo airway intervention at presentation, it is not known whether or not delayed airway obstruction is a clinical entity post–Dettol ingestion. Without such information, it may be difficult to determine the duration of close monitoring required.

Moreover, much of the current evidence on Dettol poisoning is based on case series from a single local centre,[2-4] and a handful of case reports. Nor is it known whether its conclusions can be generalised to today's Hong Kong population. This study was therefore performed to: (1) characterise the clinical features of Dettol poisoning that prevail on a territory-wide basis; (2) assess the need for airway intervention in Dettol poisoning and its time frame after ingestion; and (3) identify predictors for such interventions.

滴露中毒時氣道介入的必要性

目的 為滴露中毒的臨床徵狀作一全港性總結，評估中毒後施行氣道介入治療的需要與及中毒與治療之間的時間差距，並找出進行介入治療的預測因子。

設計 病例系列。

安排 香港16間急症室。

患者 從香港中毒諮詢中心的檔案中回顧2005年7月至2009年6月期間因吞服滴露並於48小時內送院的病人。

結果 共有213個相關病例，根據預設準則撇除其中36例。餘下的177例中，病人年齡中位數為32歲（介乎2至95歲），男女比例為1:2.7（48:129）。大部分（95%）屬故意吞服的病例。病人最常見的徵狀是氣管及消化道的局部刺激及腐蝕現象，例如腸胃不適及咽喉局部性疼痛。14名病人（8%）接受了氣道介入治療，他們都是在吞服滴露的12小時內接受治療；其中3例在拔除氣管內管後須重新插管。單元回歸分析顯示以下因素與進行氣道介入治療相關：格拉斯哥昏迷評分（GCS）低於8、年齡較大的人士、吞服大量滴露、嘴唇腫脹、胸腔聽診發現痰音和哮鳴。使用逐步回歸的多元回歸分析顯示只有昏迷（即GCS低於8）與氣道介入治療明顯相關。

結論 延遲出現的氣道阻塞在普遍情況下不會在吞服滴露多於12小時後發生。對於需要插管的病人，為避免拔管失敗而須重新插管，應於拔管前仔細評估病人是否能維持呼吸道的通暢。昏迷的病人（即GCS低於8）應盡快施以氣道介入治療。

Methods

This was a retrospective observational study based on the database of the Hong Kong Poison Information Centre (HKPIC). Since its establishment in July 2005, HKPIC has been providing 24-hour telephone consultation services on poison information and management advice in clinical toxicology to Hong Kong public hospitals and private practitioners.[8] It also acts as a portal for reporting all poisoning cases from the Accident and Emergency (A&E) departments under the Hospital Authority. The HKPIC maintains a local poison database, which provides access to information on all locally reported poisoning cases. This database was used to obtain a territory-wide picture of Dettol poisoning.

In this study, records of patients with a history of Dettol ingestion who presented within 48 hours of ingestion between July 2005 and June 2009 were used. Records were excluded if the patient had (1) co-ingested a significant amount of other corrosives or irritants that might also cause mucosal injury resulting in upper airway obstruction; and (2) a doubtful history of Dettol ingestion, possibly entailing another household disinfectant. All recruited and excluded cases were reviewed by the first two authors and eligibility for inclusion in the analysis was determined by consensus.

The patients' electronic medical records were retrieved and reviewed. For those with a complicated clinical progress or information missing from the electronic record, full medical records were retrieved for further review. Data on demographics, clinical presentation, investigations, treatment, the need for airway intervention, and outcomes were extracted using a standardised data collection form. For patients who underwent airway intervention, the maximum time interval from ingestion (as charted in the medical records) was used to assess the time frame of airway intervention. When the time of ingestion was not stated or was uncertain, particularly in patients brought to the A&E department in a state of cardiopulmonary arrest or coma, the authors estimated the time of ingestion based on documented collateral information or other circumferential evidence.

To identify predictors of airway intervention, univariate analysis was performed followed by multivariate analysis. Categorical variables were compared between groups using the Chi squared or Fisher's exact tests as appropriate. Continuous normally distributed variables were compared using the Student's *t* test. For continuous variables that were not normally distributed, differences in medians between groups were compared using the Mann-Whitney *U* test. Variables that showed a significant association ($P<0.1$) with airway intervention in the univariate analysis were further analysed by

TABLE 1. Clinical features of patients with Dettol poisoning with and without other co-ingestants

Symptoms	%	
	All cases (n=177)	Co-ingestion excluded (n=101)
Vomiting	57	62
Throat pain	38	49
Dizziness (GCS* ≥8)	27	26
Abdominal pain	25	26
Coma (GCS <8)	6	2
Hoarseness	6	6
Dyspnoea	4	4
Drooling	3	5
Mild haematemesis	1	1
Diarrhoea	1	2

* GCS denotes Glasgow Coma Scale score

multivariate analysis using forward stepwise logistic regression to adjust for the possible confounding factors. Only those variables that remained statistically significant in the multivariate analysis were regarded as reliable predictors of the need for airway intervention. All statistical analyses were performed using the Statistical Package for the Social Sciences (Windows version 14.0; SPSS Inc, Chicago [IL], US). A two-tailed P value of <0.05 was considered statistically significant.

Results

From July 2005 to June 2009, 213 records were identified from the HKPIC database, of which 36 were excluded. Reasons for exclusion were: Dettol ingestion >48 hours before presentation (n=1); co-ingestion of significant amounts of other corrosives/irritants (1 L of bleaching agent [n=1] and glyphosate [n=1]); Dettol ingestion uncertain and subsequently denied (n=3); and ingestion of disinfectants with chemical compositions different from that of Dettol (n=30).

Regarding the remaining 177 patients, their median age was 32 (range, 2-95) years, and the male-to-female ratio was 1:2.7 (48:129). Intentional Dettol ingestion constituted the majority (95%) of cases. Only eight patients reported accidental exposure and one involved suspected child abuse (a 2-year-old given two mouthfuls by his mother).

The amount of Dettol ingested was unknown in 20 patients. For persons who reported the amounts taken, the median quantity was 100 mL (range, 2.5 mL to 1 L). The median time between ingestion to presentation was 1 hour. Co-ingestion was reported in 43% of the patients, but none involved other corrosives/irritants. A history of psychiatric illness was noted in 26% of the patients and 17% had a history of intentionally taking a drug overdose.

The most common symptoms on presentation were related to the local irritative/corrosive effects on the aero-digestive tract, and included gastro-intestinal upset and localised throat pain. The clinical presentations were similar even after excluding cases with co-ingestion. These findings are summarised in Table 1.

Oral mucosal changes were documented in 24% of the cases. Lip swelling, lung crackles, and pulmonary wheezing were detected in 2%, 3%, and 2% of cases, respectively. No patient had stridor on presentation. Two patients were brought into the hospital in cardiac arrest with circumstantial evidence of Dettol ingestion, both of whom succumbed. Nine patients were asymptomatic after Dettol ingestion, all of whom had intentionally ingested only a small amount (approximately 15-60 mL).

Gastric lavage was performed in three patients: two with co-ingestion and one had ingested Dettol only, for whom the procedure was judged to be not indicated in retrospect. Fortunately, the last patient did not develop complications such as aspiration or airway oedema during her stay in hospital.

Airway intervention was required in 14 (8%) of the patients; endotracheal intubation was performed in 13 cases and fibre-optic–guided nasotracheal intubation in the other. The majority (12/14) of these patients had a low Glasgow Coma Scale (GCS) score on presentation and warranted prompt intubation for airway protection in the A&E department. Two patients were intubated in the intensive care unit (ICU) within 3 to 4 hours of admission, due to deterioration in the GCS score in one case and evidence of upper airway obstruction in addition to a low GCS score in another. During intubation, upper airway obstruction was documented in five of these 14 patients. The time of ingestion was not definitively stated in three patients but was probably <12 hours, all of whom were in a state of cardiopulmonary arrest or deep coma at presentation (Table 2). In our series, there was no evidence of delayed airway obstruction (>12 hours) after ingestion. Whereas post-extubation laryngeal oedema and subsequent extubation failure were not uncommon; re-intubation was performed in three patients (Table 2), on day 7 post-ingestion in one case. A detailed review of the clinical notes showed that all three cases were extubated hastily without prior airway examination. Overall, three patients required subsequent tracheostomy and prolonged ventilation. The median length of artificial airway placement was 1 day (range, 1-92 days).

Univariate analysis showed that coma (GCS score <8) [odds ratio (OR)=405; 95% confidence interval (CI), 41-3969; P<0.001], physical signs of lip swelling

TABLE 2. Clinical details of 14 patients who required airway intervention after Dettol ingestion*

Patient No.	Sex/ age (years)	Reported amount of Dettol ingested (mL)	Co-ingestion	GCS on admission	Evidence of airway obstruction	Place of intubation	Estimated maximum time lag between ingestion and intubation (hours)	Outcome
1	F/67	Unknown	Detergent	3/15	Yes	A&E	<3	Prolonged ventilation; survived
2	F/80	Unknown	Sleeping pills, nifedipine, and metoprolol	3/15	Not mentioned	A&E	<12	Died
3	F/59	Unknown	No	11/15 → 5/15	Oedematous vocal cord	ICU	4-5	Survived
4	M/66	500	No	3/15	Laryngeal oedema	A&E	1	Extubated on day 4 but required re-intubation soon after extubation; survived
5	F/84	Unknown	Ethanol	3/15	No	A&E	1-2	Extubated on day 7 but required re-intubation soon after extubation, prolonged ventilation; survived
6	F/68	500	Librium, mianserin, trazodone	7/15	Congested oedematous airway	A&E	1	Survived
7	F/48	20	Tricyclic antidepressant	8/15	No	A&E	2	Survived
8	M/29	750	No	15/15 → 7/15	No	ICU	2-3	Survived
9	F/60	Unknown	Listerine	3/15	Loose foreign body and vomitus in throat	A&E	1-2	Extubated on day 2 but required re-intubation on day 4; survived
10	M/63	400	No	13/15 → 8/15	No	A&E	5	Survived
11	F/77	Unknown	Acetaminophen, thymol gargle, quetiapine, nitrazepine, zolpidem, acetaminophen/ phenyltoloxamine	3/15	No	A&E	1-2	Died
12	M/39	500	Rodenticide	7/15	No	A&E	Likely <12	Survived
13	F/27	Unknown	Carbon monoxide poisoning (carboxyhaemoglobin, 30%)	3/15	No	A&E	Likely <12	Survived
14	M/71	Unknown	No	3/15	No	A&E	1-2	Died

* GCS denotes Glasgow Coma Scale score, A&E accident & emergency department, and ICU intensive care unit

(OR=13; 95% CI, 2-104; P=0.032), lung crackles (OR=15; 95% CI, 3-81; P=0.007), and pulmonary wheezing (OR=27; 95% CI, 2-320; P=0.017) were significantly associated with having airway intervention. Those who underwent airway intervention were older (median age, 65 years; interquartile range [IQR], 46-73 years vs 32 years; IQR, 26-47 years; P<0.001) and had ingested larger amounts of Dettol (median volume, 500 mL; IQR, 200-500 mL vs 60 mL; IQR, 37.5-200 mL; P=0.004). The amount of Dettol ingested was dropped in multivariate analysis because there were too many missing values on this valuable, especially for those serious cases who presented in coma and required intubation, for whom estimation of the amount of Dettol ingested was not feasible. In the multivariate analysis, only coma (GCS score <8) [OR=194; 95% CI, 16-2289; P<0.001] remained significantly associated with airway intervention.

The majority of patients (n=168) were hospitalised, and had a median length of stay of 34 (range, 1-2712) hours. Nine patients were discharged directly after A&E department assessment and did not return. In all, 15 patients were admitted to the ICU, where their median length of stay was 54 (range, 13-474) hours. The clinical course was complicated by shock, aspiration, and pneumonia in 3%, 5%, and 4% of the patients, respectively. One patient developed ARDS and two developed acute coronary syndrome. Transient mild renal impairment (defined as serum creatinine level higher than the upper limit of normal for their institution) was reported in 6% of cases. In all, five patients endured cardiopulmonary arrest

after Dettol ingestion, four of whom died, two having taken co-ingestants; the overall mortality rate was 2%.

Discussion

The authors believe that this is the largest published local case series of Dettol poisoning. By utilising the HKPIC database, we were able to assess the whole clinical spectrum of poisoning for this product on a territory-wide basis, by retrieving data for both hospitalised and non-hospitalised patients via various A&E departments in Hong Kong. Furthermore, this is the first study to assess the overall use of airway interventions for this condition and the time frame for such a procedure in unselected patients with Dettol poisoning.

The clinical presentations of Dettol poisoning in this case series were similar to those reported in a previous local study,[2] suggesting a consistent pattern of effects after oral ingestion that is applicable to a wide array of subjects. Serious complications (aspiration, pneumonia, ARDS, shock, acute coronary syndrome, and cardiopulmonary arrest) were noted in 7% of cases, which was slightly less than that previously reported in a local study of hospitalised patients,[2] and could be explained by the inclusion of milder cases that were not hospitalised. The authors therefore contend that this series provides a better overall risk profile across the entire spectrum of Dettol poisoning.

Airway intervention was undertaken in 14 (8%) of the cases. Detailed review of clinical notes showed that the common indication was airway protection in the face of a low or deteriorating GCS score, while evidence of upper airway obstruction was only documented in five patients. All such patients had immediate airway placement in the A&E department or intubation in the ICU within a few hours of admission. No delayed upper airway obstruction was encountered more than 12 hours after ingestion, regardless of co-ingestion of other substances.

In the past, it was suggested that close monitoring of the airway should continue for at least 48 hours after Dettol ingestion, based on a single case report in which upper airway obstruction developed approximately 48 hours after admission.[7] In that patient, the airway obstruction was attributed to a delayed effect of Dettol resulting in progressive airway inflammation and oedema. However, a review of the case showed that the patient was actually intubated at presentation for airway protection because of coma, and developed upper airway obstruction subsequent to extubation.[7] Moreover, it is well-recognised that endotracheal intubation per se can cause local mucosal injury resulting in post-extubation laryngeal oedema and extubation failure, even in the absence of Dettol ingestion. The reported frequency of such post-extubation laryngeal oedema ranges from 2 to 22%, depending on the diagnostic criteria used.[9] Re-intubation is deemed necessary in 1 to 5% of cases,[10] and is more common if the original tracheal intubation persists longer than 36 hours.[11] Whilst it is possible that Dettol ingestion may have contributed to post-extubation laryngeal oedema and extubation failure in that patient by virtue of mucosal injury, the extent of its contribution is unclear. Thus, the conclusion that Dettol ingestion alone causes delayed upper airway obstruction up to 48 hours after ingestion may not be true; local tissue effects after any initial endotracheal intubation should first be excluded.

Arguably, two of the patients that underwent re-intubation (Patient Nos. 5 and 9; Table 2) had no documented evidence of airway oedema when first intubated. Their initial intubation, which was performed for airway protection in the face of a low GCS score, might have masked the onset of airway oedema developing during the period of intubation, and therefore delayed airway oedema due to Dettol cannot be ruled out. Yet, none of the patients who did not undergo airway intervention in the first 12 hours after Dettol ingestion developed delayed airway obstruction.

Based on these findings, we suggest that for those who have ingested a few mouthfuls of Dettol only, airway intervention is usually not necessary. For those who do not require emergency airway management on presentation or soon after admission, close monitoring for upper airway obstruction for 24 hours is probably sufficient. For those who require intubation on presentation or shortly after admission, the adequacy of the airway should be properly assessed before extubation. Ideally the cuff-leak test[12] should be used, and preparations for possible extubation failure should be at hand.

In our series, univariate analysis showed that coma (GCS score <8), older age, a large amount of Dettol ingested, lip swelling, lung crackles, and wheezing were all associated with airway intervention. In the multivariate analysis that adjusted for confounding factors, only coma was found to be a reliable predictor for airway intervention. The fact that only one predictor was identified in this study could be due to the small number of the events (only 14/177 subjects had airway interventions). For the same reason, the 95% CIs of ORs were wide. Yet the significant association with coma signifies that for Dettol ingestion, coma, rather than upper airway obstruction, is the major driver of early airway intervention.

This study was limited by its retrospective design and the heterogeneity in the quality of documentation in different hospitals. We tried to minimise information loss by carefully reviewing individual cases and to standardise the information gathered using preset clinical criteria and a standardised data

extraction form. In addition, cases not reported to HKPIC were not included in the study, although we believe this number was small. Furthermore, the estimates of the time of ingestion might not have been accurate, particularly in the three intubated patients for whom the time of ingestion was not stated in the medical records. However, judging from the critical nature of their poisoning, prolonged delay to hospital presentation (>12 hours after ingestion) seems very unlikely. Since all such patients underwent either immediate intubation in the A&E department or in the ICU soon after admission, we believe that such interventions are not likely to be necessary >12 hours post-ingestion. Finally, the observed requirement for airway intervention and its time frame might have been affected by the presence of co-ingestants.

Conclusions

Dettol poisoning resulted in serious complications in 7% of patients, including death. Airway intervention was deemed required in 8% of patients, and all such interventions probably ensued within 12 hours of ingestion. Delayed airway obstruction of more than 12 hours post-ingestion was unlikely. For those deemed not to require airway intervention at presentation, close monitoring for upper airway oedema should continue for up to 24 hours post-ingestion. For intubated patients, careful assessment of airway adequacy is strongly recommended before extubation so as to avoid extubation failure and re-intubation.

Declaration

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