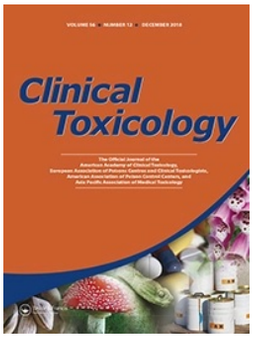

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CLINICAL RESEARCH

The acute paraquat poisoning mortality (APPM) score to predict the risk of death in paraquat-poisoned patients

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ABSTRACT

Context: Mortality prediction in paraquat poisoning is a major issue since most prediction rules are inapplicable if the exact ingestion time cannot be determined and/or the serum paraquat concentration is not readily available, as in most countries. Therefore, we aimed to develop and validate a new prediction rule not requiring these two parameters.

Methods: We designed a 10-year observational cohort study including all consecutive paraquat-poisoned patients managed in two Taiwanese hospitals. We built one cohort to define and one cohort to validate this prediction rule. Parameters independently related to mortality determined using a multivariate analysis were used to formulate the Acute Paraquat Poisoning Mortality (APPM) score.

Results: Overall, 321 paraquat-poisoned patients were included, 156 in the derivation and 165 in the validation cohort. Mortality rates in the derivation and validation cohorts were 73% and 81%, respectively ($p = 0.20$). The three parameters chosen of 28-day mortality at presentation were urine paraquat level >10 ppm (using a colorimetric sodium dithionite-based test; odds ratio (OR), 12.70; 95% confidence interval (CI), 2.64–61.24), white blood cells >13.0 G/L (OR, 5.50; CI, 1.41–21.48) and blood glucose >140 mg/dL [7.8 mmol/L] (OR, 7.45; CI, 1.70–32.86). In the derivation cohort, the area under the ROC curve (AUC-ROC) of the APPM score did not significantly differ from AUC-ROCs of serum paraquat (0.95, $p = 0.25$) and the Severity Index of Paraquat Poisoning (0.95, $p = 0.33$). AUC-ROCs of the APPM score in the derivation and validation cohorts were 0.91 and 0.94, respectively.

Conclusion: We built and validated a reliable score to predict 28-day mortality in paraquat-poisoned patients at presentation, independently from the ingestion time and serum paraquat measurement.

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Paraquat; poisoning; prediction; mortality; predictive score

Introduction

Paraquat, a bipyridylium herbicide widely used in agriculture, is responsible for a high-rate fatality poisoning [1]. Since no therapy has been proved effective to reduce poisoning-related death, mortality prediction remains a major issue, currently based on four available severity scores routinely used at the bedside, that is, Proudfoot's nomogram with Scherrmann's extension [2,3], Hart's nomogram [4], the Severe Index of Paraquat Poisoning (SIPP) [5], and Jones's probability score [6]. Parameters used to calculate these four scores include time elapsed since ingestion and serum paraquat concentration. However, self-reported ingestion time may be inaccurate and serum paraquat measurements not readily available, precluding the determination of these scores at presentation. We therefore designed this study to develop a new simple and reliable mortality prediction rule in paraquat-poisoned patients that does not require ingestion time determination nor serum paraquat measurement.

Methods

Study design and patient selection

We designed an observational two-center cohort study. All consecutive paraquat-poisoned adults admitted to the emergency departments at Linkou Chang Gung Memorial Hospital (LCGMH) and Taipei Veterans General Hospital (TVGH) during the study period (2008–2018) were included. LCGMH and TVGH are academic teaching hospitals located in north Taiwan, with 3000 and 2947 beds and with monthly emergency department visits of ~14,000 and 7000, respectively. Approvals from both hospital ethics committees (201600407B0 in LCGMH, 201605001BC in TVGH) were granted.

Paraquat poisoning was confirmed based on history, suggestive features, positive urine paraquat (using a sodium dithionite reaction-based colorimetric test; normal range, <5ppm; values reported by the laboratory as <5ppm, 5 ppm, 10 ppm, 25 ppm, 50 ppm, and >50ppm) and/or serum

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Supplemental data for this article can be accessed here.

paraquat concentration on admission (using spectrophotometry coupled with sodium dithionite assay; limit of quantification, 0.1 mg/L). Patients with skin exposure to paraquat, any other agrochemical or drug co-ingestion, missing urine and serum paraquat measurement, presentation >24-h postingestion (since outcome could not be evaluated by some scores such as Proudfoot's nomogram [2]) and out-of-hospital cardiac arrest were excluded. Patients from LCGMH were assigned to the derivation set and patients from TVGH to the validation set.

Data collection and covariate definition

A standardized abstraction form was used to collect all variables retrospectively from the electronic medical records. Collected variables included basic demographics, emergency department triage vital signs, physical examination of mucosa lesions on admission, hemoglobin, white blood count (WBC), platelet count, serum glucose, sodium, potassium, bicarbonate, creatinine, urea, alanine aminotransferase, arterial blood gas, serum/urine paraquat concentrations on admission and treatments administered including supportive care, gastric lavage, activated charcoal, hemoperfusion, hemodialysis, pulse therapy (methylprednisolone/cyclophosphamide combination), N-acetylcysteine and vitamin C. SIPP was determined.

Statistical methods

Data are expressed as percentages or median [25th–75th percentiles] unless indicated otherwise. Since data distribution did not meet the normality assumption, univariate comparisons were performed using Mann–Whitney U-tests for numerical variables and Chi-squared tests for categorical variables. Of note, urine paraquat levels were divided into two groups (>10 ppm and ≤10 ppm) for mortality prediction analysis, as previously suggested [3]. Univariate parameters associated with death with $p < 0.1$ were introduced in a multivariate analysis using a backward selection multiple logistic regression model. To examine the model fitness, the Hosmer–Lemeshow goodness-of-fit test was used. Odds ratios and their 95% confidence intervals (CI) were determined to assess the association strength and express the reliability of the study outcomes.

A score based on predictors of death yield by the β-coefficients obtained from the multivariate model was developed from the derivation set then tested on the validation set. Each β-coefficient was divided by 0.4 and rounded to the nearest integer to form the prediction rule allowing the easy scoring of each parameter. Points were first assigned to each predictor and a score was calculated for each patient. Calculated scores were grouped into risk groups. The derived model and prediction rule were validated in the validation cohort and the C-statistic for the validation group was calculated. Thereafter, the prediction model was used to calculate the scores in the validation cohort and the scores used to place patients into the previously defined risk groups. Finally, to assess the clinical utility of the prediction rule, we determined its received operating characteristics (ROC) curve and area under the ROC curve (AUC-ROC). All statistical analyses were performed using SAS version-9.4® (SAS Institute Inc., Cary, NC, USA). *P*-values <0.05 were regarded as significant.

Results

Overall, 321 paraquat-poisoned patients were admitted to LCGMH ($n = 156$) and TVGH ($n = 165$) during the study period. Of these patients, 235 (73%) were enrolled, 121 admitted to LCGMH assigned to the derivation cohort and 114 admitted to TVCGH to the validation cohort (Figure 1). Death rates were 73% and 81% in the derivation and validation cohorts, respectively ($p = 0.20$). Patient characteristics,

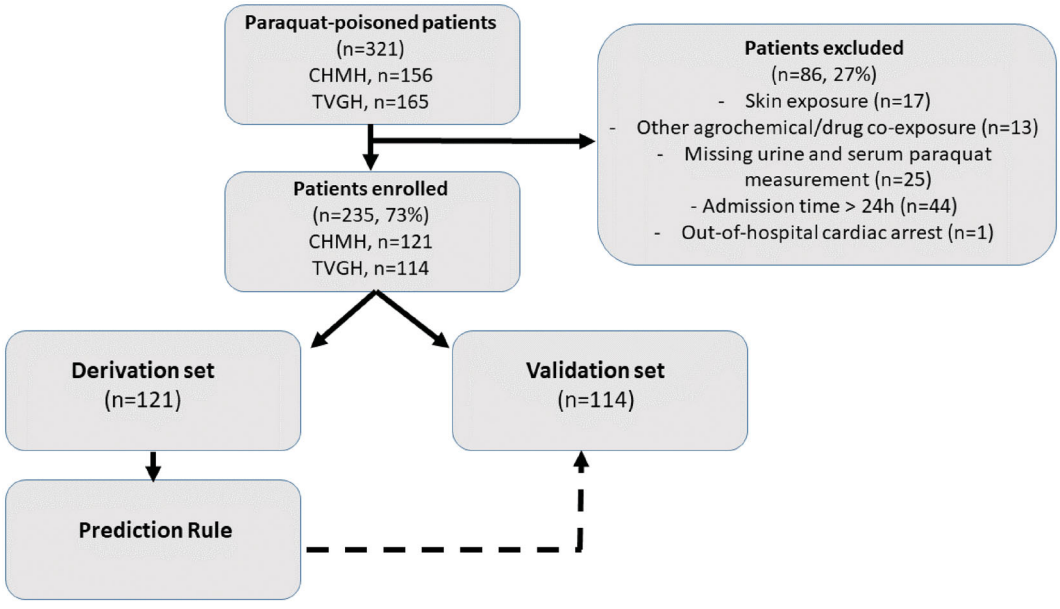

Figure 1. Paraquat-poisoned patient enrollment and assignment to the derivation or validation sets, along with the flow for developing and validating the prediction rule. LCGMH: Linkou Chang Gung Memorial Hospital; TVGH: Taipei Veterans General Hospital.

Table 1. Paraquat-poisoned patient characteristics in the derivation and validation cohorts.

Variable	Patients		p Value
	Derivation ($n = 121$)	Validation ($n = 114$)	
Demographic and clinical characteristics on admission			
Age (years)	50 [38–65]	52 [38–61]	NS
Male, *n* (%)	84 (69%)	80 (70%)	NS
Systolic blood pressure (mmHg)	142 [121–161]	134 [119–156]	NS
Diastolic blood pressure (mmHg)	84 [74–96]	80 [68–93]	NS
Heart rate (/min)	94 [82–108]	89 [75–105]	NS
Body temperature (°C)	36.1 [35.5–36.6]	36.0 [35.6–36.7]	NS
Glasgow coma score (3 to 15)	15 [13–15]	15 [13–15]	NS
Respiratory rate (/min)	20 [18–21]	20 [19–22]	NS
Mucosal lesions, n (%)	38 (31)	33 (29)	NS
Laboratory variables on admission			
White blood cells (G/L)*	15.2 [10.0–21.1]	15.9 [10.9–24.2]	NS
Hemoglobin (g/dL)	14.6 [13.2–16.0]	14.5 [13.4–16.0]	NS
Platelet (G/L)*	255 [213–306]	285 [229–338]	0.01
Serum creatinine (mg/dL)	1.2 [0.9–1.8]	1.4 [0.9–2.3]	0.04
Blood urea nitrogen (mg/dL)	12.5 [9.1–15.4]	13.0 [10.1–17.0]	NS
Alanine aminotransferase (IU/L)	24 [19–38]	28 [20–39]	NS
Arterial pH	7.40 [7.26–7.44]	7.36 [7.28–7.40]	NS
$PaCO_2$ (mmHg)	33 [27–39]	31 [23–37]	0.02
PaO_2 (mmHg)	73 [53–94]	89 [73–111]	<0.0001
Serum bicarbonate (mmol/L)	19.3 [15.3–22.8]	17.5 [11.9–21.2]	0.01
Serum potassium (mmol/L)	3.3 [2.8–3.7]	3.2 [2.7–3.5]	NS
Serum sodium (mmol/L)	140 [138–142]	142 [139–144]	0.001
Blood glucose (mg/dL)**	146 [109–201]	155 [121–201]	NS
Urine paraquat level > 10ppm, n (%)	91 (81)	87 (84)	NS
Treatment modalities			
Hemoperfusion, *n* (%)	73 (60)	35 (31)	<0.0001
Interval to hemoperfusion (h)	8 [6–16]	6 [5–9]	0.03
Cyclophosphamide + MTP, *n* (%)	34 (28)	1 (1)	<0.0001
NAC, *n* (%)	1 (1)	80.(70)	<0.0001
Vitamin C, *n* (%)	0 (0)	53.(46)	<0.0001
Severity			
SIP*p* > 10, *n* (%)	70 (63)	68 (68)	NS
Mortality, *n* (%)	88.(73)	92.(81)	NS

MTP: methylprednisolone; NAC: N-acetylcysteine; SIPP: Severity Index of Paraquat Poisoning. */mm^3 = G/L × 1000; **mmol/L = mg/dL/18.

laboratory data and administered treatments in these two cohorts are given in Table 1. Forest plots of count variables are shown in Supplemental material (Figure 1S). On admission, patients presented mild acute kidney injury with partially compensated metabolic acidosis and hypokalemia.

Variables associated with mortality rate in the derivation cohort were determined. Interestingly, treatment modalities did not appear to contribute to mortality. Nonsurvivors appeared older (54 years [41–68] *versus* 42 years [38–58], $p = 0.015$), presented on admission with lower body temperature (36.0 °C [35.3–36.5] *versus* 36.4 °C [36.1–37.1], $p = 0.001$), higher WBC (16.9 G/L [11.1–23.1] *versus* 10.3 G/L [7.8–12.4], $p < 0.0001$), higher platelet count (265 G/L [214–307] *versus* 238 G/L [204–260], $p = 0.04$), higher creatinine (1.4 mg/dL [1.0–2.1] *versus* 0.9 mg/dL [0.7–1.0], $p < 0.0001$), higher blood urea nitrogen (13.1 mg/dL [10.4–17.1] *versus* 10.0 mg/dL [6.9–13.1], $p = 0.02$), lower arterial pH (7.38 [7.22–7.44] *versus* 7.41 [7.39–7.45], $p = 0.02$), lower serum bicarbonate (17.6 mmol/L [14.3–20.4] *versus* 23.0 mmol/L [20.6–25.6], $p < 0.0001$), lower $PaCO_2$ (32 mmHg [25–39] *versus* 36 mmHg [33–40], $p = 0.04$), lower serum potassium (3.1 mmol/L [2.7–3.6] *versus* 3.5 mmol/L [3.3–3.7], $p = 0.001$), higher serum sodium (141 mg/dL [139–143] *versus* 139 mg/dL [137–141], $p = 0.002$), higher blood glucose (171 g/dL [125–217] *versus* 107 g/dL [98–122], $p < 0.0001$), higher percentage of urine paraquat level >10 ppm (96% vs 41%, $p < 0.0001$), and with more SIPP >10 (84 *vs* 10%, $p < 0.0001$) (Supplemental material, Table 1S). Based on a multiple logistic regression model, the three parameters contributing to 28-day mortality were urine paraquat concentration >10ppm (OR, 12.70; CI, 2.64–61.24), WBC >13.0 G/L (OR, 5.50; CI, 1.41–21.48) and blood glucose >140mg/dL [7.8 mmol/L] (OR, 7.45; CI, 1.70–32.86) (Table 2).

Thereafter, we defined a new clinical prediction rule, the Acute Paraquat Poisoning Mortality (APPM) score, allowing stratifying patients in four mortality rate groups, that is, low (11%), moderate (58%), high (76%) and very high (100%) (Table 3). By applying this score to the validation cohort, patients could be stratified into low, (4%), moderate (47%), high (95%), and very high (100%) mortality rate groups ($p < 0.0001$). The AUC-ROCs of the APPM score was 0.91 in the derivation and 0.94 in the validation cohorts ($p = 0.035$; Figure 2). The Hosmer–Lemeshow goodness-of-fit statistic was 1 for the model in both cohorts. The AUC-ROCs of the SIPP (0.91 *versus* 0.95, NS) and plasma paraquat (0.94 *versus* 0.95, NS) in the derivation *versus* validation cohorts were determined.

Discussion

We validated a new simple score, the APPAM score, to predict the risk of death in paraquat-poisoned patients. The extremely high fatality rate in paraquat poisoning emphasizes the importance of mortality prediction, with four

Table 2. Independent predictors of death in paraquat poisoning identified by multivariate analysis.

Variable	β[a]	Odds Ratio	95% confidence interval	Points
Intercepts	−2.57			
Urine paraquat > 10 ppm	2.54	12.70	2.64 – 61.24	6
Serum glucose > 140 mg/dL [7.8 mmol/L]	2.01	7.45	1.70 – 32.86	5
White blood cell count > 13.0 G/L	1.70	5.50	1.41 – 21.48	4

[a]β vector of weight (or regression coefficient) corresponds to the outcome. The β-coefficient for each variable was divided by 0.4 and rounded to the nearest integer to form the prediction rule.

Table 3. Predictive values of the Acute Paraquat Poisoning Mortality (APPM) score based on mortality rates observed in the derivation ($n = 121$) and validation ($n = 114$) cohorts.

	Low risk	Moderate risk	High risk	Very high risk
APPM score	0, 4, 5	6, 9	10, 11	15
Derivation cohort, died/total (%)	2/18 (11%)	11/19 (58%)	16/21 (76%)	37/37 (100%)
Validation cohort, died/total (%)	4/17 (24%)	7/15 (47%)	21/22 (95%)	48/48 (100%)

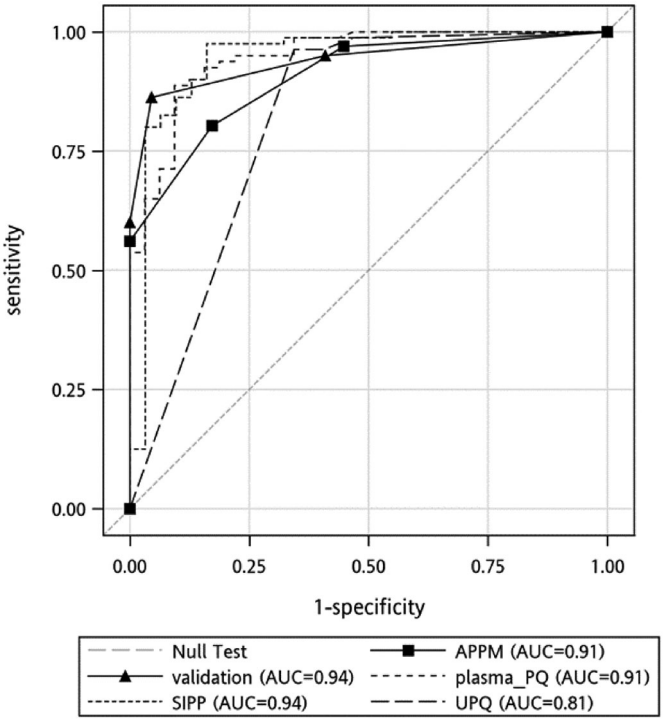

Figure 2. The receiver operator characteristics (ROC) curves of the Acute Paraquat Poisoning Mortality (APPM) score (area under the curve (AUC), 0.91), the Severe Index of Paraquat Poisoning (SIPP; AUC, 0.95) and plasma (AUC, 0.95) and urine paraquat concentrations (AUC, 0.81) in the derivation cohort and of the APPM in the validation cohort (AUC, 0.94). There was no statistically difference between AUCs of the APPM score *versus* the SIPP and serum paraquat concentration ($p = 0.33$ and $p = 0.25$, respectively).

currently available severity scores [2–6]; however, all require the time of ingestion, not always accurate, and the real-time serum paraquat concentration, not always available. By using readily available biomarkers correlated to mortality such as body temperature, respiratory index, WBC, serum potassium, blood lactate, serum creatinine, liver function tests and/or urine paraquat level using a colorimetric test, alternative scoring systems were developed [6–15]. However, the serum paraquat and time post-ingestion remained key parameters required in all these scores.

General physiological scores used to evaluate multi-organ function including the Acute Physiology and Chronic Health Evaluation II (APACHE II), the Expanded Simplified Acute Physiology Score II (MSAPS IIe), and the Sequential Organ Failure Assessment (SOFA) score were also used but compared poorly to scores based on serum paraquat [16–18]. MSAPS-IIe modified by excluding the 24-h urine paraquat amount showed no significant differences with serum paraquat concentration or SIPP; however, several parameters used in this score were not related to paraquat poisoning. The SOFA score was modified, but the main additional parameter was serum paraquat concentration [17]. Finally, machine-learning processes for outcome prediction were designed but not yet validated [19].

Our simple APPM score presents advantages, as it relies on three biomarkers readily available in most emergency departments (Table 4). It maintained a good predictive ability for death in the validation cohort. Like other validated scores, our score was good at predicting high-risk mortality but not at excluding low-risk mortality. AUC-ROCs of SIPP, serum paraquat concentration and APPM score were not significantly different, suggesting that APPM score can reliably replace previous scores if serum paraquat concentration or ingestion time are not available.

Our study has limitations due to its retrospective design. To limit inaccuracy in data retrieving, we used uniform reporting forms and collected laboratory rather than nonobjective clinical history data. The presumed dose and concentration of the ingested paraquat solution were rarely available. We acknowledge that the derivation and validation cohorts should have been chosen more optimally at random from the entire population rather than using the hospitals to define the datasets. Some data significantly differed between the two cohorts. For instance, the observed difference in PaO_2 values could be attributed to a certain degree of venous and arterial blood mixture when puncturing the artery, possibly due to different sampling practices at the two hospitals. The observed tendency to hypothermia could also be related to imperfect body temperature measurement in the emergency department, as observed in real life. Since we included paraquat-poisoned patients admitted within 24 h, AAPM score can only be applied for patients referred within 24-h postingestion. Hence, we do not suggest using it for delayed presentations. Finally, antioxidant therapies including N-acetylcysteine and vitamin C used to treat our paraquat-poisoned patients seemed ineffective. Our findings clearly suggest that banning of this highly lethal herbicide from the market should be the only effective solution to reduce the

Table 4. Application of the Acute Paraquat Poisoning Mortality (APM) score in two patients.

	History	Laboratory data	APPM score	Predicted mortality	Outcome
Patient 1	M, 61 years Suicidal attempt Ingestion of an unknown amount of paraquat ~30 min before ED visit	WBC, 8.4 G/L* Blood glucose, 171 mg/dL** Urine paraquat level, 50 ppm	11 (0 + 5 + 6)	High risk	Dead 3 days after ICU admission
Patient 2	M, 62 years Suicidal attempt Ingestion of ~1 mouth of praraquat ~1 h before ED visit	WBC, 6.3 G/L* Blood glucose, 135 mg/dL** Urine paraquat level, 5 ppm	0 (0 + 0 + 0)	Low risk	Alive Discharged 29 days after admission

ED: emergency department; ICU: intensive care unit; WBC: white blood cell count. */mm^3 = G/L × 1000; **mmol/L = mg/dL/18.

consequences of acute ingestions, as already shown in other neighboring countries [20,21]. Interestingly, the first-stage ban on the import and production of paraquat from February 2018 was very recently shown to be followed by an estimated 37%-reduction in pesticide suicide rate (rate ratio, 0.63, CI, 0.54–0.74) predominantly related to a 58% reduction in paraquat suicide rate (rate ratio, 0.42; CI 0.33–0.54) in Taiwan [22].

To conclude, we defined and validated the APPM score, a readily available score usable everywhere based on three simple parameters on admission, that is, serum glucose concentration, WBC and urine paraquat concentration. Its predictive ability did not significantly differ from those of currently used scores requiring determination of the ingestion time and serum paraquat concentration. Its definitive validation still requires a multicenter prospective study.

Disclosure statement

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