PREDICTIVE FEATURES OF CHRONIC KIDNEY DISEASE IN aHUS

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INTRODUCTION

- Chronic kidney disease (CKD) is a frequent and serious complication of atypical hemolytic uremic syndrome (aHUS)[1].
- No reliable biological markers allow a rapid and accurate diagnosis of aHUS, while highly efficient targeted therapy are available[2].
- Consequently, the use of targeted therapies is limited at the acute phase of the disease and rapidly available prognostic surrogate markers are urgently needed[4].

AIM

We aimed to develop a simple accurate model to predict the risk of CKD in aHUS on the basis on clinical and biological features available at hospital admission and treated by dialysis and/or plasma exchange only.

METHODS

- Multicenter retrospective study
- Patients included in our National Registry :
  - **Thrombocytopenia and microangiopathic haemolytic anemia**
  - **Acute kidney injury**
  - **ADAMTS 13 activity > 20%**
- Excluded : HIV infection, cancer, chemotherapy, transplantation, connective tissue disease and malignant hypertension, STEC infection and patients with *E. coli* infection or colonization
- From October 2000 to June 2014
- Outcome : **eGFR < 60mL/min/1,73m²** in 1-years follow-up
- Multivariable **logistic regression** with multiple imputation by chained equation
- Scoring system constructed based on the regression coefficient with internal bootstrap validation

Table 2. Prognosis Score

Serum creatinine (mg/dL)	Point
0-1	0
1.1-3.39	1
3.4-5.64	2
> 5.65	3
Platelets (10^3/µL)	
0-59	0
> 60	1
Mean arterial pressure (mmHg)	
0-105	0
> 106	1

RESULTS

Table 1. Univariate analysis

Variables		CKD (n = 66)	No CKD (n = 44)	*P value*
Arterial pressure (mmHg)	Systolic	160 [146-180]	140 [116-152]	< .001
	Diastolic	90 [80-100]	80 [70-90]	< .001
	Mean	113 [103-123]	97 [89-113]	< .001
Digestive symptoms	Overall	36 (54.5%)	23 (53.5%)	> .9
Neurologic symptoms	Overall	31 (47%)	18 (40.9%)	.56
Blood cell count	Haemoglobin (g/dL)	8.3 [6.9-9.7]	9.4 [8.3-10.5]	.006
	Platelets (10^3/µL)	84 [49-121]	41 [25-59]	< .001
Renal impairment	Serum creatinine (mg/dL)	6.3 [3.7-8.8]	2.8 [1.4-4.1]	< .001
	Renal replacement therapy	54 (81.8%)	10 (22.7%)	< .001
ADAMTS13 (%)		41 [30-62]	51 [40-68]	.08
Infection	Fever	10 (15.1%)	12 (29.2%)	.09
	Documented	9 (13.6%)	15 (34.1%)	.02
Search for genetic abnormalities* / anti-CFH Abs**		24 (36.4%)	12 (27.2%)	.41
Treatment	Plasma exchange (PE)	53 (80.3%)	36 (81.8%)	.9
	Number of PE/patient	11 [5-20]	8 [3-14]	.32
Follow up	Death	4 (6.1%)	1 (2.3%)	
	Time to platelets recovery (d)	11 [5-33]	6 [6-16]	.25

Fig 1. Probability of CKD according score

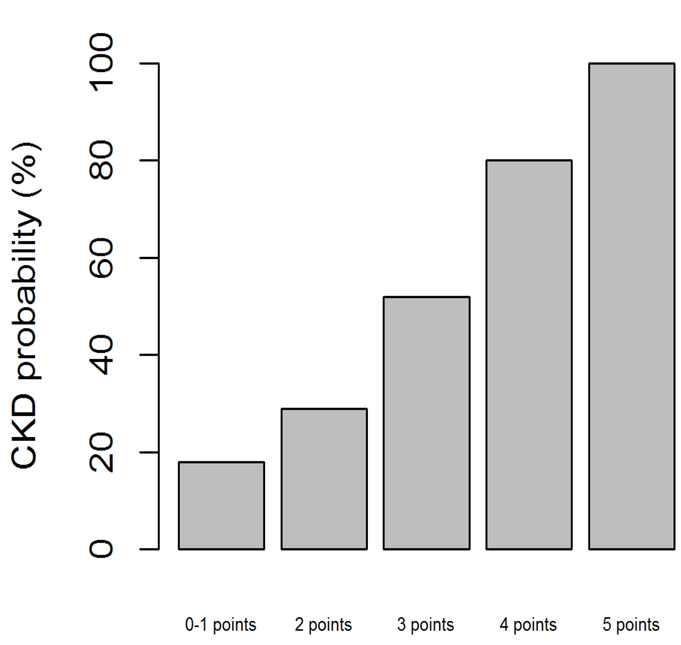
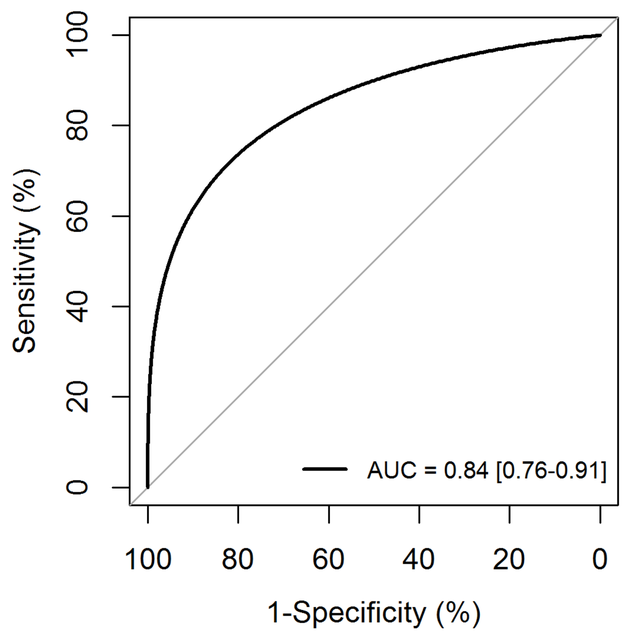


Fig 2. ROC curve



CONCLUSIONS

Global renal prognosis under plasma therapy was poor, with 66 (42 %) of patients who developed severe CKD at 1-year follow-up.

Risk factors for CKD were a **higher blood pressure** on admission (p < .001), a severe renal involvement on diagnosis (p < .001) and renal replacement therapy requirement (p < .001) and a **mildly decreased platelet count** on diagnosis (p < .001).

Patients with a prognostic **score < 2** had no or mild renal involvement (8.3 %), more pronounced thrombocytopenia (27 [8-41] G/L)and a normal or mildly increased MAP (95 [86-100] mmHg). Their outcome was excellent with plasma exchange.

Our clinical score based on admission data is able to predict persistent renal failure under plasma exchange; it should therefore **help identifying patients** suitable for alternative therapies frontline, such as complement blockers.

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