Scientific Research Publishing

Open Journal of Nephrology, 2025, 15(4), 494-505
https://www.scirp.org/journal/ojneph
ISSN Online: 2164-2869
ISSN Print: 2164-2842

Adult Pulmonary-Renal Syndrome in a Military Centre: A 19-Year Retrospective Review

Sanaa Benbria[1], Kawtar Hassani[1,2], Youssef Zorkani[3], Aya Sobhi[1], Majdoline Errihani[1], Driss ElKabbaj[1,2]

[1]Department of Nephrology-Dialysis, Military Hospital Mohammed V, Rabat, Morocco
[2]Faculty of Medicine and Pharmacy, Mohammed V University, Rabat, Morocco
[3]Department of Naval Health, Moroccan Royal Navy, Rabat, Morocco
Email: drsanaabenbria@gmail.com

How to cite this paper: Benbria, S., Hassani, K., Zorkani, Y., Sobhi, A., Errihani, M. and ElKabbaj, D. (2025) Adult Pulmonary-Renal Syndrome in a Military Centre: A 19-Year Retrospective Review. *Open Journal of Nephrology*, **15**, 494-505.
https://doi.org/10.4236/ojneph.2025.154046

Received: October 3, 2025
Accepted: October 28, 2025
Published: October 31, 2025

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Abstract

Pulmonary Renal Syndrome (PRS) is a rare entity, defined by the association of intra-alveolar hemorrhage and rapidly progressive glomerulonephritis, which histologically translates to extracapillary glomerulonephritis. It requires emergency diagnosis and treatment, and its etiologies are mostly ANCA vasculitis. The objective of this study was to describe the characteristics of renal and pulmonary involvement during PRS, its etiologies and evolution, and to study the overall survival of the patients. This is a descriptive and analytical study that includes all patients admitted for PRS and is analyzed retrospectively from January 2005 to March 2024. There were 18 patients with an average age of 45 years [32;64] and a sex ratio of 1. Regarding renal manifestations, 16 patients out of 18 showed rapidly progressive renal failure, requiring the use of dialysis in the majority of cases. The average serum creatinine level of the patients included was 751 µmol/l, the average proteinuria was 3.2 g/24h, and 15 patients had hematuria. Regarding pulmonary manifestations, 13 patients presented with hemoptysis, 15 patients had intra-alveolar hemorrhage confirmed by chest CT scan, and half of them required mechanical ventilation. The median follow-up of the patients was 38 months [15;137]. The treatment response consisted of pulmonary remission in all patients versus renal remission in only 6 patients, with 3 patients having come off dialysis. Twelve patients progressed to chronic renal failure while 11 patients were still relying on dialysis; there were 6 relapses, mainly pulmonary, and 2 deaths. There was no statistically significant difference in the overall survival of patients who relapsed or not ($p = 0.67$). In conclusion, PRS is a life-threatening emergency in both the short and long term. In our series, renal involvement was as severe as pulmonary involvement, with progression to chronic renal failure in two-thirds of patients and significant dependence on dialysis.

Keywords

Pulmonary Renal Syndrome, Renal Failure, Vasculitis, ANCA

1. Introduction

Pulmonary-Renal Syndrome (PRS) is a rare disorder that is defined as the combination of an Alveolar Hemorrhage (AH) and Rapidly Progressive Glomerulonephritis (RPGN). The characteristic renal histological lesion of PRS is Extracapillary Glomerulonephritis (ECG).

This syndrome was originally described in 1919, along with the description of the first cases of anti-Glomerular Basement Membrane disease (GBM), also known as Goodpasture's syndrome since 1958, leaving the term PRS to the combination of AH and ECG regardless of the etiology [1]. Several mechanisms are involved in the pathogenesis of this syndrome; they involve anti-GBM antibodies, Antineutrophil Cytoplasmic Antibodies (ANCA), and thrombotic microangiopathy [2]. However, ANCA-associated vasculitides are the main cause of PRS (60% - 70%) [3]. It is a diagnostic and therapeutic emergency that can be life-threatening in the short term via pulmonary involvement and also in the long term via renal involvement, especially renal sequelae.

The objective of our work is first to describe the clinical, biological, radiological, and histological characteristics of PRS and then to study the overall patient survival.

2. Materials and Methods

This is a retrospective study conducted at the Department of Nephrology, Dialysis, and Renal Transplantation of the Military Hospital in Rabat, over a 19-year period, from January 2005 to March 2024. The study population was comprised of all adults with PRS who were managed and monitored in the department, regardless of the initial recruitment circumstances.

Pulmonary renal syndrome was retained for any patient with the combination of alveolar hemorrhage and rapidly progressive glomerulonephritis.

The diagnosis of AH is suggested by the clinical triad associating hemoptysis, anemia, and radiological pulmonary infiltrates, with a variable mode of installation (insidious to sudden). Bronchial endoscopy with LBA will confirm the diagnosis when the Golde score (score quantifying the iron load of macrophages using Perls staining) is greater than 100 [4].

RPGN is defined as a clinical syndrome characterized by a rapid loss of kidney function (days to weeks) associated with extensive crescent formation, usually involving >50% of glomeruli on kidney biopsy [5].

Glomerulonephritis. Patients with hemoptysis and/or dyspnea of another origin, as well as those with acute kidney failure of another nature, were excluded from our study. For each patient, we noted their epidemiological data, the initial care

setting, pulmonary and renal data at the time of diagnosis, the use of renal replacement therapy and its delay compared to the time of diagnosis, the use of oxygen therapy, the etiology according to immunological data and/or renal histology, as well as overall management. Short- and long-term clinical and biological monitoring allowed treatment response to be classified into: Renal remission is defined as a stable or improved glomerular filtration rate, while hematuria and proteinuria are present at times of active disease and can resolve completely; their persistence does not necessarily imply active disease. General remission is defined as the absence of manifestations of vasculitis and GN. Relapse is defined as the occurrence of increased disease activity after a period of partial or complete remission; a return or increase of hematuria with proteinuria may indicate a kidney relapse. Treatment-resistant disease is defined as the persistence of or appearance of kidney and/or systemic manifestations of vasculitis while receiving treatment equal in intensity to initial immunosuppressive therapy [6]. Chronic renal failure was defined by a Glomerular Filtration Rate (GFR) less than 60 ml/min/1.73m^2 lasting more than 03 months [7].

3. Results

Eighteen patients were collated, with a median age of 45 years [32;64] and a sex ratio of one. Initial recruitment was done through various departments; however, a third of the patients were recruited in nephrology. The majority of patients reported extrarenal symptoms in their history, which were dominated by joint signs. Concerning pulmonary history, hemoptysis was found in three patients (16.7%) with one case of asthma (5.6%) (**Table 1**).

At the first medical examination, 10 patients (55.6%) experienced the following renal symptoms: microscopic hematuria in 15 patients (83.3%) and oliguria in 5 patients (27.8%). All patients had elevated creatinine. The averages of initial creatinine and proteinuria levels were, respectively, 751 µmol/l and 3.2 g/24h (**Table 1**).

Furthermore, 13 patients (72.2%) experienced hemoptysis, which revealed an alveolar hemorrhage, and 15 patients (83.3%) had both hemoptysis and microcytic anemia (**Table 1**). All patients underwent lung imaging, including chest X-ray, which revealed alveolar syndrome in 13 patients (72.2%), and chest CT, which showed signs of AH in 15 patients (83.3%). Bronchial fibroscopy with bronchoalveolar lavage was performed in 3 patients with an average Golde score of 271 ± 65.5 (**Table 2**). In addition to pneumo-renal involvement, 15 patients (83.3%) had an altered general condition, and 12 (66.7%) had associated signs dominated by joint signs.

ANCA-associated vasculitis constituted the main etiology of PRS, with a specificity for anti-proteinase 3 (PR3) in seven patients (38.9%), and for anti-myeloperoxidase (MPO) in three patients (16.7%), with four (22.2%) cases of ANCA-negative vasculitis, followed by Goodpasture disease in two cases (11.1%), and one case of overlap syndrome of ANCA-associated vasculitis and anti-Glomerular Basement Membrane (GBM) and one case of systemic lupus erythematosus (**Table 3**).

Table 1. Characteristics of the study population.

Variable	**n = 18**
Average age (yrs)*	45 [32 - 64]
Gender $	
Male	9 (50 %)
Female	9 (50 %)
General signs $	16 (88.9%)
Hypertension $	5 (27.8%)
Lower limb edema $	6 (33.3%)
Oligoanuria $	5 (27.8%)
Hématurie $	15 (83.3%)
Serun Créatinine level (µmol/l)*	751 [433; 1246]
GFR ml/min/1.75 m^2	5 [3 - 11]
Proteinuria (g/24h)**	3.2 ± 1.98
Dyspnea $	4 (22.2%)
Cough $	2 (11.1%)
Haemoptysis $	13 (72.2%)
Anemia $	15 (83.3%)
Haemoglobin level (g/dl)**	7.2 ± 1.98
Extrarenal Signs $	12 (66.7%)

*expressed in median and inter-quartile range. **expressed as an average ± standard deviations. $ expressed in number and percentage. GFR: Glomerular Filtration Rate.

Table 2. Radiological characteristics of the study patients.

Variable	**n = 18**
Chest X-ray $	
Alveolar syndrome	13 (72.2%)
Interstitial syndrome	4 (22.2%)
Chest CT-scan $	
Ground-glass opacities	9 (50%)
Nodule	3 (16.7%)
Condensation	3 (16.7%)
Fiberoptic bronchoscopy + BAL $	
Golde score ≥ 100	3 (16.7%)

$ expressed in number and percentage. CT: Computed Tomography, BAL: Bronchoalveolar Lavage.

Histological study of renal biopsy using both optical microscopy and IF showed type 1 RPGN in 16.7% (3) of cases, type 2 RPGN in 5.5% (1) of cases, and pauci-immune glomerulonephritis in 66.6% (14) of cases, 7 of which were crescentic, 6 were sclerotic, and one was mixed type (Table 3).

Table 3. Etiologies of pulmonary renal syndrome.

Patient n°	Serological parameters	Histology		Diagnosis
		OM	IF	
1	ANCA-MPO, Anti-GBM	ECGN	Linear IgG deposits	ANCA-associated Goodpasture's sd
2	ANCA-PR3	ECGN	Negative	GPA
3	ANCA-PR3	ECGN	Negative	GPA
4	ANCA-PR3	ECGN	Negative	GPA
5	ANCA-PR3	ECGN	Negative	GPA
6	Negative	ECGN	Negative	GPA
7	ANCA-PR3	-----	-----	GPA (ANCA negative)
8	Anti-MBG	ECGN	Linear IgG deposits	Goodpasture
9	Anti-MBG	ECGN	Linear IgG deposits	Goodpasture
10	ANCA-PR3	ECGN	Negative	GPA
11	Negative	ECGN	Negative	GPA (ANCA negative)
12	Negative	-----	-----	MPA (ANCA negative)
13	Negative	ECGN	Negative	MPA (ANCA negative)
14	ANCA-MPO	ECGN	Negative	MPA
15	ANCA-MPO	ECGN	Negative	MPA
16	ANCA-PR3	ECGN	Negative	GPA
17	ANA, anti-DNA	ECGN	Granulaire Ig deposits	SLE
18	ANCA-MPO	ECGN	Negative	MPA

OM: Optical Microscopy, IF: Immunofluorescence, ANCA: Antineutrophil Cytoplasm Antibodies, PR3: Proteinase3, MPO: Myeloperoxydase, GBM: Glomerular Basement Membrane, ANA: Antinuclear Antibodies, ECGN: Extracapillary Glomerulonephritis. GPA: Granulomatosus with Polyangiitis, MPA: Microscopic Polyangiitis, SLE: Systemic Lupus Erythematosus.

In the case of ANCA vasculitis, induction treatment was initiated based on intravenous cyclophosphamide boluses 15 mg/kg (adapted to age and GFR) at weeks: 0, 2, 4, 7, 10, 13, 16 (19, 21, 24) if necessary, associated with glucocorticoids administered as boluses (15 mg/kg for example without exceeding 1 g, 3 days in a row), with a relay at the dose of 1 mg/kg at least during the first week [6]. Plasmapheresis has been indicated in patients requiring dialysis and/or with diffuse alveolar hemorrhage and hypoxemia [6]. Patients with anti-GBM disease received methylprednisolone boluses: 500 - 1000 mg IV/day for 3 days, switched to oral prednisone: 1 mg/kg/day (max 60 mg/day), with gradual tapering over 6 - 12 weeks, oral cyclophosphamide: 2 mg/kg/day for 2 - 3 months, and plasma exchange for 10 to 14 days [5].

In our study, 14 patients (77.8%) required renal replacement therapy upon diagnosis of the disease, within a median time of four days [0;34], nine patients (50%) needed oxygen therapy, among whom one was under invasive ventilation, and six (33.3%) used therapeutic plasma exchange. All patients received corticosteroids as well as cyclophosphamide as an induction treatment (**Table 4**). The maintenance treatment was based on azathioprine; in cases of relapse, rituximab was used as induction and maintenance treatment [8].

Table 4. Therapeutic management of pulmonary renal syndrome.

Variable	n = 18
Oxygen therapy $	9 (50%)
Oxyden mask	8 (44.4%)
Intubation	1 (5.6%)
Plasma exchange $	6 (33.3%)
Dialysis $	14 (77.8%)
Time to dialysis (days)*	4 [0;34]
Induction $	
Cyclophosphamide IV	16 (88.9%)
Cyclophosphamide OR	2 (11.1%)
Maintenance $	
Azathioprine	13 (72.2%)
Rituximab	0

*expressed in median and inter-quartile range; $ expressed in number and percentage; IV: intravenous, OR: oral route.

Pulmonary response was achieved in all patients, while renal response was achieved in only six patients (33.3%), three of whom got out of dialysis. As for the rest of the patients, 12 (66.6%) progressed to Chronic Kidney failure (CKD), including 11 on renal replacement therapy.

The median follow-up of the patients in this study was 38 months [15;137], and the median overall survival was not achieved (Figure 1).

During the follow-up, six cases (33.3%) of relapses, mainly pulmonary, were reported, and 2 deaths (11%), one due to severe AH and the other to septic shock. The Log-rank test was used to compare the overall patient survival according to relapse occurrences; it did not find any statistically significant difference between the two groups, p = 0.67 (Figure 2).

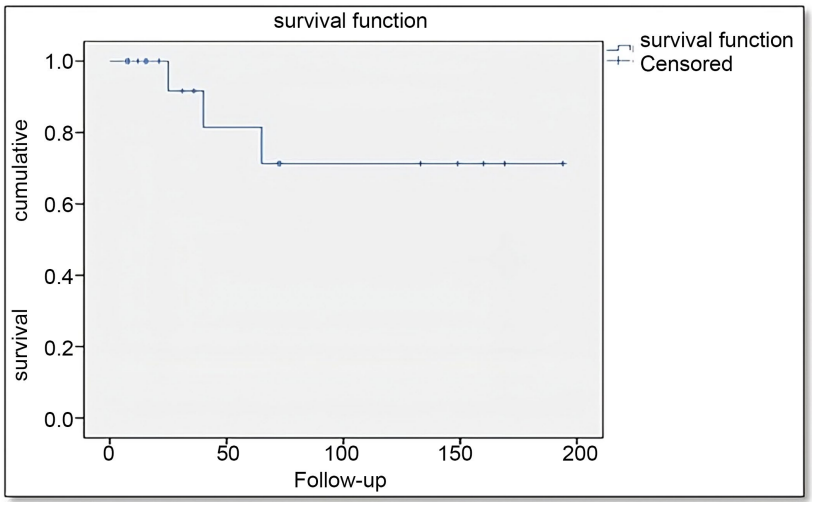

Figure 1. Overall survival of patients with pulmonary renal syndrome.

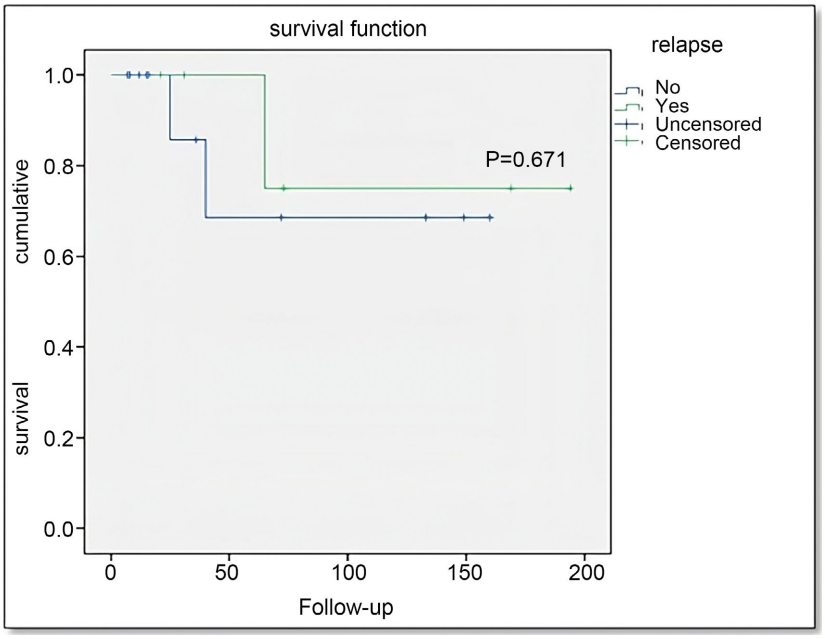

Figure 2. Survival of patients with pulmonary renal syndrome based on relapses.

4. Discussion

Over a period of 19 years, this work included 18 patients from different departments (ICU, nephrology, pneumology, neurology), with a median age of 45 years [32;64], 10 years younger than what is reported in the literature [9]-[11].

According to a meta-analysis that included nine studies, PRS affects men and women equally (53% and 47%, respectively) [11] [12], which is consistent with the results of our series.

The clinical picture is associated with prominent respiratory symptoms and usually poor renal symptoms. Indeed, according to a monocentric Indian study including 25 PRS patients, hemoptysis was present in 68% of cases, and 88% of patients were dyspneic, half of whom required mechanical ventilation [13]. Respiratory impairment can sometimes be more discreet, such as a simple cough, dyspnea, or hemoptoic sputum [4]. The patients in our series showed mainly pulmonary signs, with 13 patients out of 18 having hemoptysis, 9 of whom had acute respiratory failure that required oxygen therapy. However, the absence of obvious respiratory symptoms does not eliminate the diagnosis, since occult alveolar hemorrhage may be present and will need the use of radiology and/or endoscopy to be documented [4] [14]. According to a meta-analysis by West *et al*., 51 out of the 62 chest CT scans showed abnormal signs, with the presence of ground-glass opacities in 94% of patients [12]. In our series, 15 patients out of 18 had AH signs in their chest CT scans. Bronchoscopy with bronchoalveolar lavage was often used to confirm the diagnosis of diffuse pulmonary hemorrhage, discarding at the same time any pulmonary infection [10] [13].

Renal symptoms are often inconspicuous and not very specific. In the Indian study, 13 patients out of 25 had kidney damage, but only four reported clinical

signs [13]. About a third of our patients had lower extremity edema with oligoanuria.

In another cohort of 75 patients with ANCA-associated vasculitis, grouped according to the presence or absence of diffuse alveolar hemorrhage, renal involvement was more severe with a higher average serum creatinine level and greater hematuria in the AH group [15]. This is consistent with our results, since 90% of our patients had an RPGN presentation that led to dialysis in 61% of cases.

Renal biopsy has both diagnostic and prognostic value and must be carried out as soon as possible in the absence of contraindications, but must in no case delay the treatment. Extracapillary glomerulonephritis is found in the majority of cases; however, in some cases, other lesions can be found using optical microscopy, pointing towards other etiologies such as intracapillary thrombi or necrotizing granulomas. Immunofluorescence is crucial for etiological diagnosis depending on whether or not immunoglobulin and/or complement deposits are present and, if so, their appearance and location. Thus, we distinguish three types: type 1 extracapillary GN, which corresponds to anti-GBM disease (with linear IgG deposits along the glomerular basement membrane); type 2 extracapillary GN, with immune complex deposits including primary glomerulopathies, lupus, post-infectious glomerulonephritis; and finally, type 3 extracapillary GN, which includes ANCA-associated vasculitides, also known as pauci-immune GN given the absence of significant deposits at the IF [16].

In Gallagher's study of 14 patients, 10 had a renal biopsy. Optical microscopy showed extracapillary GN in nine patients. Immunofluorescence was performed on all samples; eight samples of the nine showed no evidence of deposition, confirming the diagnosis of pauci-immune GN, which is consistent with our results [9].

Systemic necrotizing ANCA-associated vasculitides are the main cause of PRS (66.5%) [2]. Typically, proteinase 3 (PR3)-ANCA and cytoplasmic (c-ANCA) profiles are observed in granulomatosis with polyangiitis (GPA), while Myeloperoxidase (MPO)-ANCA and perinuclear (p-ANCA) profiles dominate in Microscopic Polyangiitis (MPA) and in approximately 40% of Eosinophilic Granulomatosis with Polyangiitis (EGPA) cases [17]. Patients with PRS are more frequently positive for PR3 than for MPO [10] [11], which confirms the results obtained by our work.

Among the etiologies of SPR, the association of vasculitis with anti-MPO ANCA and anti-GBM disease (14.3%) comes second, followed by anti-GBM disease (3.8%) [2]. In our series, among the three anti-GBM disease cases we had, one was associated with anti-MPO ANCA. In 24.5% of cases, other conditions causing PRS were found, such as Systemic Lupus Erythematosus (SLE), which is particularly severe with a mortality close to 50% [18], catastrophic antiphospholipid syndrome, periarteritis nodosa, HIV-related vasculitis, cryoglobulinemic vasculitis and Henoch-Schönlein purpura [2]. We reported only one case of SLE in our study.

Symptomatic respiratory and renal management is necessary in any patient with PRS. According to Gallagher and the Indian study, mechanical ventilation was used in 47.5% and 56% of cases, respectively, which is consistent with our study [9] [13].

The use of dialysis in the PRS varies; it reaches 51.2% according to Bulut *et al*. [11]. The need for dialysis in our series was greater (61%).

Plasma Exchange (PE) is part of the standard treatment for Goodpasture's syndrome, as are corticosteroids and cyclophosphamide, whereas for ANCA-associated vasculitides, they are indicated when the initial serum creatinine level is higher than 300 µmol/l or if dialysis and/or severe AH is required [19]. According to the MEPEX trial, the addition of PE to corticosteroids and cyclophosphamide in patients with ANCA-associated vasculitis showed a significant benefit on short-term kidney function (less than 12 months) [20]. However, the international PEXIVAS trial did not obtain a significant benefit from PE in either mortality or terminal CKD [10] [21]. In our study, only patients with severe alveolar hemorrhage and advanced renal failure received PE.

As for the specific treatment, there are no prospective clinical studies on the treatment of PRS because it is a life-threatening emergency that requires therapy to be started upon admission, and also given the small size of the series and the heterogeneity of the diagnoses. Treatments are empirical, extrapolated from the treatment of extracapillary glomerulonephritis or codified treatments for the pathology responsible for the PRS.

The sequelae of an episode of PRS are mainly renal and depend on the serum creatinine level at the time of diagnosis. In the Gallagher study, after two years of follow-up, 25% of patients remained dependent on dialysis [9], whereas in our series the renal response was inferior to the pulmonary response, with dialysis dependence in 61% of patients after a median follow-up of 36 months. Relapses are common in anti-PR3 ANCA-associated vasculitides [10] [21]; the relapse rate in our study was higher given the frequency of ANCA PR3 vasculitis [11].

Although the small numbers involved prevent meaningful statistical analysis for group comparisons, the mortality rate in our series remains lower than reported in the literature. Indeed, the initial mortality rate of PRS ranges from 10 to 50%, and is especially correlated to the intensity of alveolar hemorrhage [10] [22]-[24]. Long-term prognosis depends on renal response to treatment [23] [24].

This study has several important limitations. First, its retrospective, single-center design inherently carries a risk of selection and information bias. Second, despite the rarity of SPR, the sample size remains modest. Finally, the lack of long-term follow-up data limits the assessment of overall patient outcomes. Future research should include larger, multicenter, prospective studies to validate these findings.

5. Conclusion

PRS is a rare condition requiring early diagnosis and urgent therapeutic manage-

ment. In our series, this syndrome affects a relatively young population without gender predominance and with pulmonary symptoms as its main clinical manifestation. However, renal damage was severe and consisted of a rapidly progressive kidney failure presentation with frequent use of renal replacement therapy. ANCA-associated vasculitis was the main etiology in our study. The combination of corticosteroids, cyclophosphamide ± plasma exchange allowed a good pulmonary response; however, the renal response was lower with a significant dependency on dialysis.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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