



Clinical and microbiological characteristics of *Pantoea agglomerans* infection in children



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ABSTRACT

Pantoea agglomerans is an environmental Gram-negative bacterium that rarely is responsible for the infections in humans but it is often a causative factor of a number of occupational diseases. This study evaluated the clinical and microbiological characteristics and pathogenicity of *P. agglomerans* in children.

We retrospectively reviewed microbiological test results for all children (1 month old to 18 years old) who were admitted to our pediatric hospital between January 2000 to June 2015 and had positive clinical specimen cultures for *P. agglomerans*. Isolates were identified using conventional tests and the BBL Crystal E/NF ID or MALDI-TOF MS systems. Antibiotic susceptibilities were evaluated using the Kirby-Bauer disc diffusion method.

We identified fifteen positive cultures from 14 patients with confirmed infections. The positive specimens included pus, urine, tracheal aspirate, blood, and central venous line samples that yielded *P. agglomerans*. The median patient age was 8.8 years (range: 1.5 months to 16.5 years), and all patients had underlying comorbidities. Five patients had medical devices, and two devices were removed. The most common *P. agglomerans* infections involved wound infections (35.7%), pneumonia (21.4%), and urinary tract infections (21.4%). Three patients had concomitant infections (*Enterococcus faecium*, *Pseudomonas aeruginosa*, and *Aspergillus fumigatus*). Five patients had anemia. Three patients (21.4%) died, and all three had carbapenem-resistant *P. agglomerans* that was detected after the first week of hospitalization; two cases involved pneumonia, which was ineffectively treated.

P. agglomerans infections may be life-threatening, especially in young patients with pneumonia. Hospital-acquired *P. agglomerans* may have different pathogenicity and clinical features, compared to community-acquired *P. agglomerans*, although further studies are needed to understand the drug-resistance patterns in this bacterium.

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Introduction

Pantoea agglomerans is a yellow-pigmented, rod-shaped Gram-negative aerobacillus that belongs to the *Enterobacteriaceae* family, and has previously been known as *Enterobacter agglomerans* or *Erwinia herbicola* [1,2]. It was reclassified into a new genus in 1989 [3]. *P. agglomerans* is an environmental and agricultural

organism that is frequently isolated from plants, soil, water, and food [1]. This organism is an opportunistic pathogen, and infection usually requires an immunocompromised host [4]. Nevertheless, despite human infections being uncommon, they may be associated with trauma that was caused by penetration with vegetative material during performing of agricultural occupations, gardening or children playing, and also with secondary bacteremia, or nosocomial infections that are related to medical equipments such as intravenous catheters or contaminated intravenous fluids [5–7]. Furthermore *P. agglomerans* is often a causative factor of a number of occupational diseases, caused by the effects of protein allergens and endotoxin produced by this pathogen, with the allergic and/or immunotoxic background [8,9].

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P. agglomerans has been identified as a possible cause of vertebrate animal diseases but compared to humans, there are only few reports for *P. agglomerans* infections in this group. Apart from vertebrate animals, *P. agglomerans* has been isolated from some arthropods and *P. agglomerans* may be also pathogenic for plants [7].

In recent years, the beneficial traits of *P. agglomerans* have been mentioned such as its a set of antibiotics production and the role of the immunopotentiator from *P. agglomerans* 1 (IP-PA1) in the prevention and treatment of for the animals and human diseases or food preservation in contrast to the proven pathologic role of *P. agglomerans* [8].

In the present study, we evaluated the clinical and microbiological characteristics (including carbapenem resistance) of *P. agglomerans* infections among children who were treated at our hospital over the past 15 years due to limited data in childhood.

Patients and methods

Hospital setting

The Hacettepe University İhsan Doğramacı Pediatric Hospital is a 270-bed, tertiary-care, pediatric referral hospital in Turkey. This hospital treats approximately 215,000 outpatients and 11,000 inpatients each year. In the present study, we retrospectively identified all children (1 month old to 18 years old) who were admitted to our hospital between January 2000 and June 2015 and had clinical specimens that provided positive culture results for *P. agglomerans*. This study's retrospective design was approved by the institutional review board of the Hacettepe University Faculty of Medicine.

Specimen collection

Microbiological data were retrieved from the microbiology laboratory's electronic records, and the patients' clinical and microbiological data were considered together. Specimens that yielded *P. agglomerans* were collected from venous blood, urinary collection bags (in cases of urinary tract infections with $\geq 100,000$ colony-forming units), incision sites, abscess drainage, and tracheal aspirate. However, six isolates were excluded because of contamination and seven isolates were excluded because of insufficient clinical data. Thus, 15 isolates from 14 patients were included in this study (Fig. 1)

Bacterial identification and antimicrobial susceptibility testing

The blood, catheter, tracheal aspirate, and pus specimens were inoculated onto 5% sheep blood agar and chocolate agar, and urine specimens were inoculated onto 5% sheep blood agar and MacConkey agar. All cultures were incubated at 37 °C for 24–48 h in a 5% CO₂ atmosphere. Gram-negative bacteria from the cultures were identified using conventional tests and the BBL Crystal E/NF ID system (Becton Dickinson Microbiology Systems, Cockeysville, Maryland, USA) or a matrix-assisted laser desorption ionization-time of flight mass spectrometry system (BioMerieux, France). Antibiotic susceptibilities were tested using the Kirby-Bauer disc diffusion method, according to the Clinical and Laboratory Standards Institute guidelines [10,11].

Statistical analysis

All data were analyzed using SPSS software (version 20.0 (SPSS, Inc., Armonk, NY, USA)). Descriptive statistics were used to summarize the baseline patient characteristics. Median values and interquartile ranges (IQR) were calculated for continuous variables

and frequency distributions were calculated for categorical variables.

Results

We identified 36 *P. agglomerans* isolates. However, 13 isolates were excluded because of contamination or insufficient clinical data (Fig. 1), and 8 isolates of newborn patients were excluded. Only 15 isolates from the 14 patients with clinically documented were included. The median age was 8.8 years, (range: 1.5 months to 16.5 years) who were treated during 2000–2015. One patient had two isolates. The male-to-female ratio was 1.8:1, and the patients' demographics, clinical characteristics, and comorbidities are summarized in Table 1. The specimens with detectable *P. agglomerans* included pus (6 specimens, 42.8%), urine (3 specimens, 21.4%), tracheal aspirate (3 specimens, 21.4%), and blood (3 specimens, 21.4%). The most common clinical diagnoses for patients with significant culture growth were wound infections (35.7%), pneumonia (21.4%), and urinary tract infections (21.4%) (Table 1). Three cultures exhibited concomitant pathogens: One pus specimen with *Enterococcus faecium*, one tracheal aspirate with *Pseudomonas aeruginosa*, and one pus specimen with *Aspergillus fumigatus*.

Most patients required hospitalization (85.7%), although 2 patients were treated as outpatients. The median length of hospitalization was 22.5 days (range: 5–292 days; IQR: 7.2–49.5 days), and the median length of hospitalization after the positive culture result was 11 days (range: 0–126 days, IQR: 7.0–30.2 days). Five patients had medical devices (three central venous catheters, one renal double J stent, one dialyzing catheter, and one cardiac pacemaker), although one central venous catheter and the renal double J stent were removed because of growing *P. agglomerans* in the central venous catheter and nephrolithiasis. Patient 12 had both a central venous catheter and a cardiac pacemaker. Two patients (14%) required mechanic ventilation on the date of their positive culture results, and both patients died. Among the 14 patients, 11 patients (78.5%) had medical records with documented laboratory findings of new-onset *P. agglomerans* infection. There was no evidence of colonization prior to onset of infection.

The median values for white blood cell counts, hemoglobin levels, and thrombocyte counts were 8900/ μ L (range: 4300–30,300/ μ L), 10.7 g/dL (range: 8.1–15.3 g/dL), and 361,000/ μ L (range: 55,000–652,000/ μ L), respectively. Five patients had anemia, and one of these patients had systemic lupus erythematosus, hemolytic uremic syndrome, thrombocytopenia (55,000/ μ L), and lymphopenia (300/ μ L). Only two patients had leukocytosis. Ten patients recovered (71.4%), and their treatment was selected based on the susceptibility testing results (Tables 2 and 3).

Three patients exhibited carbapenem-resistant *P. agglomerans* (21.4%), and all three patients died (Table 4). The first patient had cystic fibrosis, was hospitalized because of pneumonia, and required mechanical ventilation during the follow-up. The patient received ciprofloxacin, meropenem, liposomal amphotericin B, vancomycin, ornidazole, and piperacillin with tazobactam. *P. agglomerans* with concomitant *Pseudomonas aeruginosa* was isolated from tracheal aspirate. Although the *Pseudomonas aeruginosa* was susceptible to ciprofloxacin and piperacillin, the *P. agglomerans* was resistant to carbapenems, ciprofloxacin, and piperacillin. The second patient was a 6-month-old hypotonic infant who was born prematurely and was hospitalized for pneumonia, bradycardia, and to diagnose the etiology of the hypotonicity. Carbapenem-resistant *P. agglomerans* was detected on day 12 of the hospitalization, and *Acinetobacter baumannii* was detected on day 36 in a culture of tracheal aspirate. The antimicrobial treatments during the hospitalization were ceftriaxone, meropenem, fluconazole, teicoplanin, vancomycin, and amikacin. That patient ultimately died on day 48

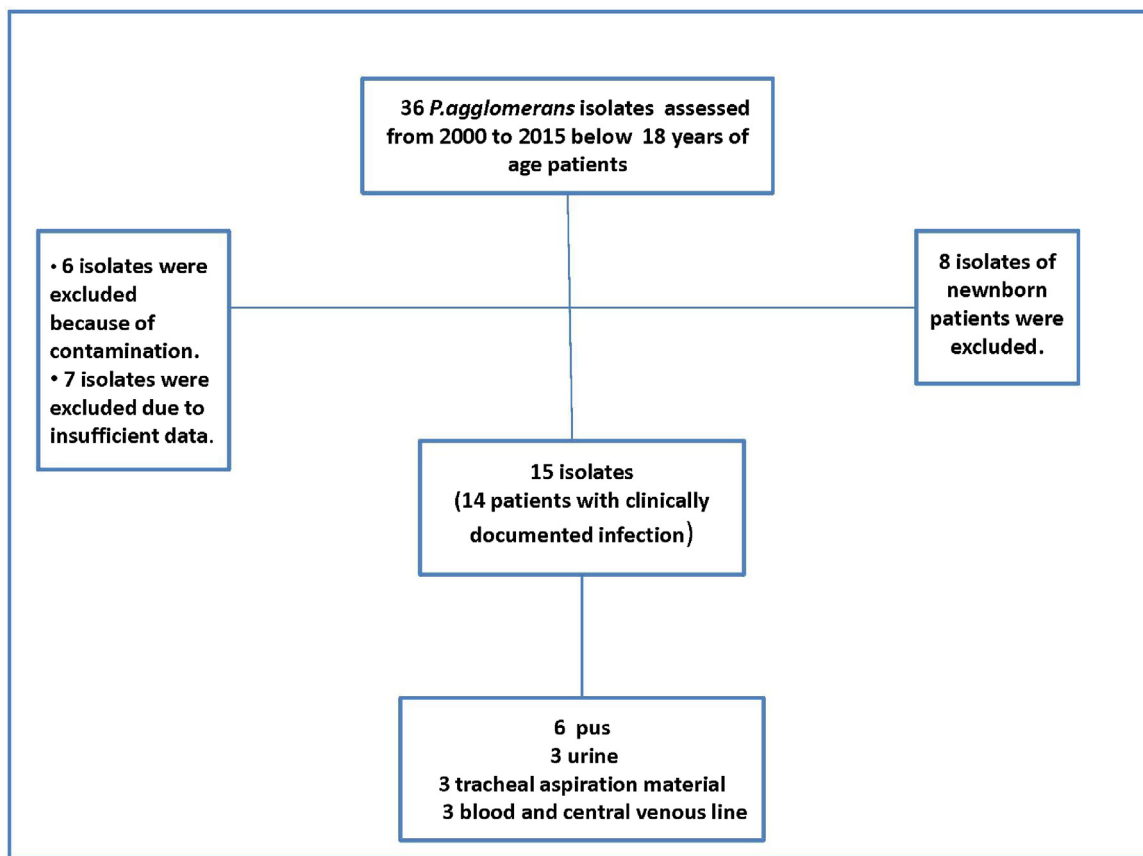


Fig. 1. A Consort diagram showing the numbers of included/excluded isolates and patients.

Table 1

The demographics, clinical characteristics, and comorbidities of the included patients.

Case	Gender	Age	Underlying comorbidities	Hospitalization	Site	Infection type	Concomitant pathogen
1	Female	3 years	Focal segmental glomerulosclerosis, neurometabolic disease?	Yes	Central venous catheter	Catheter infection	Nil
2	Male	15,5 years	Intestinal perforation	Yes	Pus	Wound infection	<i>Enterococcus faecium</i>
3	Male	7,5 years	Non-hogkin lymphoma	Not stated	Both central venous catheter and peripheral blood	Bacteremia	Nil
4	Female	16,5 years	Osteosarcoma	Yes	Pus	Wound infection	Nil
5	Male	10 years	Cyctic fibrosis	Yes	Tracheal aspiration material	Pneumonia	<i>Pseudomonas aeruginosa</i>
6	Male	14 years	Precocious puberty	No	Pus	Fruncele	Nil
7	Male	12 years	Meningomyelocele, hydrocephaly, neurogenic bladder, nephrolithiasis	Yes	Urine	Urinary system infection	Nil
8	Female	7 years	Chickenpox	Yes	Pus	Wound infection, abscess	Nil
9	Male	2 months	Phenylketonuria	Yes	Urine	Urinary system infection	Nil
10	Male	4,5 years	Vesicoureteral reflux	Yes	Urine	Urinary system infection	Nil
11	Male	15,5 years	Neurofibromatosis type 2, head trauma	Yes	Pus	Wound infection	Nil
12	Male	6 months	Prematurity, hypotonic infant	Yes	Tracheal aspiration material	Pneumonia	Nil
13	Female	12,5 years	Systemic lupus erythematosus, hemolytic uremic syndrome	Yes	Pus	Wound infection	<i>Aspergillus fumigatus</i>
14	Female	2 months	Foreing body aspiration	Yes	Tracheal aspiration material	Pneumonia	Nil

because of the progression of pneumonia and acute renal failure. The third patient was 12.5 years old and had systemic lupus erythematosus, hemolytic uremic syndrome, palate erosion, and wound infections in the nasal cavity. *P. agglomerans* and *Aspergillus fumigatus* were detected in pus from the nasal cavity, and the patient was treated using amikacin, meropenem, ceftazidime, and voriconazole.

The *P. agglomerans* isolate was carbapenem-resistant and sensitive to amikacin. The patient ultimately died on day 34 because of the progression of pneumonia after the onset of *P. agglomerans* growth. Both *Aspergillus fumigatus* and *P. agglomerans* were considered contributing pathogens.

Table 2
Treatments and other patient features.

Case	Carbapenem resistance (imipenem/meropenem)	Mechanic ventilation	Device	Device removal or surgery	Antibiotic treatment (day)
1	No	No	Central venous catheter	Central venous catheter removal	Ampicillin–sulbactam (10)
2	Not stated	No	No	No	Ornidazole (28), cefaperozone sulbactam (28), ciprofloxacin (21), flucanazole (19), amikacin (21), tobramycin (7)
3	No	No	Central venous catheter	No	Not stated
4	No	No	No	Surgery (debridement)	None
5	Yes	Yes	No	No	Ciprofloxacin (35), meropenem (34), liposomal amfotericin B (26), vankomicin (12), ornidazole (13), piperacillin with tazobactam (3)
6	No	No	No	No	Ciprofloxacin (7), mupirocin (local)
7	No	No	Renal double j stent	Renal double j stent removal	Ceftriaxone (5)
8	No	No	No	No	Ampicillin–sulbactam (8), clindamycin (6)
9	No	No	No	No	Amikacin (7)
10	No	No	No	No	Ceftriaxone (5)
11	No	No	No	No	Piperacillin with tazobactam (10), ampicillin–sulbactam (6), amoxicillin clavulanate (7)
12	Yes	No	Central venous catheter, cardiac pacemaker	No	Ceftriaxone (31), meropenem (17), fluconazole (31), teicoplanin (31), vancomycin (9), amikacin (9)
13	Yes	Yes	Dialysing catheter	No	Amikacin (23), meropenem (46), ceftazidime (10), voriconazole (49)
14	No	No	No	No	Amikacin (11), ampicillin–sulbactam (17)

Table 3
Treatment effectiveness, antimicrobial susceptibility, and outcomes of *P. agglomerans* infection.

	Treatment suitability to the antimicrobial susceptibility for <i>P. agglomerans</i>	Outcomes
The patients that <i>P. agglomerans</i> was isolated after the first week of hospitalization		
Case 1	Yes	Recovered
Case 5	No	Died
Case 12	No	Died
Case 13	Yes	Died
The patients that <i>P. agglomerans</i> was isolated in the first week of hospitalization		
Case 2	Yes	Recovered
Case 4	No	Recurrence of wound site infection
Case 7	Yes	Recovered
Case 8	Yes	Recovered
Case 9	Yes	Recovered
Case 10	Yes	Recovered
Case 11	Yes	Recovered
Case 14	Yes	Recovered
<i>P. agglomerans</i> infection in outpatients		
Case 3	Unknown	Recovered
Case 6	Yes	Recovered

Table 4
Susceptibilities of the *P. agglomerans* from the three patients who died.

Patients	Patient 1	Patient 2	Patient 3
Ceftazidime	R	R	I
Gentamicin	R	R	R
Piperacillin	R	R	R
Mezlocillin	R	R	NS
Cefoperazone	I	R	R
Cefepime	R	R	R
Ciprofloxacin	I	R	R
Imipenem	R	R	R
Meropenem	R	R	R
Tobramycin	R	R	S
Carbenicillin	R	R	R
Levofloxacin	NS	R	NS
Amikacin	R	R	S

R: Resistant, I, Intermediate, S: Sensitive, NS: Not stated.

Patient 8 presented with cellulitis and an abscess near the hip after a varicella infection. *P. agglomerans* was isolated from the abscess drainage material, and the patient was successfully treated using ampicillin–sulbactam and clindamycin.

Patient 14 required hospitalization because of foreign body aspiration (a chickpea) and pneumonia. *P. agglomerans* was isolated from the tracheal aspirate, and the patient was successfully treated using amikacin and ampicillin–sulbactam.

Discussion

In literature previous reports have described pathogenic *P. agglomerans* being isolated from cases of wound infection, abscess, bacteremia, pneumonia, urinary tract infection, septic arthritis, osteomyelitis, peritonitis, choledocholithiasis, dacryocystitis, and endophthalmitis [1,6,12–21]. Furthermore, other reports have described associations between *P. agglomerans* and contaminated intravenous fluid, total parenteral nutrition, propofol (an anesthetic agent), blood products, and powdered infant formula [6,22]. However, there are few reports of lower respiratory tract infections or urinary infections that involved *P. agglomerans*. Cruz et al. have reported three pediatric cases of polymicrobial urinary tract infections that involved *P. agglomerans*, although all of those patients survived [6]. Cheng et al. and Shubov et al. have also reported two adult patients with pre-existing conditions (bladder cancer and heart–lung transplant) who successfully recovered from *P. agglomerans* pneumonia [1,13]. In the present study, we identified three cases of *P. agglomerans* urinary tract infection (not polymicrobial) and all of those patients recovered, although we also identified three cases of pneumonia-associated *P. agglomerans* infection; two of these cases involved polymicrobial infection and ultimately resulted in death. A previous study revealed that pulmonary disease was prominent in an outbreak of *P. agglomerans* at a neonatal intensive care unit (related to infected parenteral nutrition solutions), and the mortality rate was 87.5% [23]. Thus, these findings indicate that lower respiratory tract infections that involve *P. agglomerans* may be a life-threatening condition in children.

All of the patients in the present study presented with comorbidities, and none of the patients had experienced penetration with vegetative material, although *P. agglomerans* was isolated from the tracheal aspirate from one case (Patient 14; chickpea aspiration). Two patients had medical devices that were removed (a renal double J stent in Patient 7 and a central venous catheter in Patient 1), and both patients subsequently recovered. Two patients who died had devices that were not removed, although *P. agglomerans* was not isolated from those devices (a central venous catheter, a cardiac pacemaker, and a dialyzing catheter). Cruz et al. evaluated 21 patients with central venous line-related bacteremic episodes that involved *P. agglomerans* [6], and although 19 of these patients survived, 14 patients had polymicrobial infections that required line removal [6]. In contrast, Cheng et al. have reported that the persistence of a catheter was not associated with adverse outcomes or relapses in cases of *P. agglomerans* infection [1]. Therefore, the relationship between the course of *P. agglomerans* infection and the presence and/or removal of medical devices remains unclear.

Sporadic outbreaks of *P. agglomerans* infection, especially in hospitals, have been reported [5,24]. In the present study, *P. agglomerans* was detected in five cases after the first week of hospitalization, and these cases appear to have been nosocomial infections, based on the timing of the infections. Similarly, Richard et al. evaluated 10 *P. agglomerans* strains that were isolated from the stool samples of adult patients who were hospitalized in the same ward, and 9 of these 10 strains were acquired during the hospitalization [25]. The authors considered the possibility of cross-contamination during the study period, as none of the identified strains caused a documented infection. However, 7 of the 10 isolated *P. agglomerans* strains expressed an extended-spectrum β -lactamase phenotype. In the present study, we identified cases with carbapenem-resistant *P. agglomerans* in tracheal aspirate and nasal cavity samples after the first week of hospitalization, and these patients were hospitalized for >2 weeks. Therefore, these findings suggest that *P. agglomerans* can be isolated in both the community and healthcare settings. No concrete evidence has identified a discrete evolutionary link occurred between plant-associated and clinical *P. agglomerans* isolates [26,27], although pathogenic factors can presumably be acquired through horizontal gene transfer that is mediated by plasmids and other mobile elements in the healthcare setting [28]. Moreover, carbapenem-resistant *Enterobacteriaceae* are becoming more common worldwide, and infection with these bacteria is associated with poor outcomes, high mortality rates, and limited treatment options [29]. Nevertheless, there is very little information regarding carbapenem-resistant *P. agglomerans* (a member of the *Enterobacteriaceae* family). In the present study, 21.4% of the *P. agglomerans* isolates were carbapenem-resistant and caused a documented infection. However, interpretation of this result is limited by the small number of cases and the potential effects of the patients' underlying serious comorbidities and other concomitant pathogens.

There is also very little information regarding the origin of *P. agglomerans* in the healthcare setting. Cheng et al. have suggested that the transmission of *P. agglomerans* may involve gastrointestinal translocation through mucosal lesions in the gastrointestinal tract and/or low stomach acidity after the ingestion of plant products [1]. *P. agglomerans* can also be detected among the normal hand flora, and the household environment may be a source for both community- and hospital-acquired infections [30]. Furthermore, Gora et al. found that *P. agglomerans* was prevalent in air samples from cases of occupational exposure to organic dust [31]. Moreover, *P. agglomerans* may be capable of directly penetrating human skin through microtrauma sites and/or medical devices.

P. agglomerans is generally considered an opportunistic pathogen, although it has other features. For example, *P. agglomerans* can be used to synthesize antibiotics (e.g., pantocins,

herbicidins, microcins, and phenazines), and *P. agglomerans* Tx10 has also been used to treat *Staphylococcus aureus* infections [8,32]. Furthermore, *P. agglomerans* is used as a bio-pesticide because of its antifungal and antibacterial properties, as well as its safety in animals, and it can also be found in plant products [13]. Moreover, a lipopolysaccharide from *P. agglomerans* (IP-PA1, a macrophage-priming agent) is thought to improve immune protection against various diseases and prevent stress-related immunosuppression [33]. This lipopolysaccharide also improved survival and ameliorated chemotherapy-induced immunosuppression in a mouse model of melanoma [34,35].

In conclusion, *P. agglomerans* may cause serious morbidity and mortality, especially in young patients with underlying comorbidities, and pneumonia with *P. agglomerans* may be a life-threatening condition in children. Nevertheless, community-acquired and hospital-acquired cases of *P. agglomerans* infection may have different pathogenic and clinical features. Therefore, further large-scale studies are needed to investigate the clinical and pathogenic characteristics of drug-resistant *P. agglomerans*.

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Competing interests

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version. Additionally, there are no conflicts of interest in connection with this paper, and the material described is not under publication or consideration for publication elsewhere.

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