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Case Report

Dolosigranulum Pigrum Bacteremia in an Injecting Drug User Associated with Methicillin Resistant Staphylococcus Aureus Endocarditis and Fungal Endophthalmitis

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Abstract:

Dolosigranulum pigrum is a commensal bacterium of the upper respiratory tract. We present a case of D. Pigrum bacteremia, identified incidentally in a patient with a history of injecting drug use (IDU), chronic hepatitis C (HCV) and partially treated methicillin resistant staphylococcus aureus (MRSA) endocarditis, who presented with acute monocular vision loss. Workup was consistent with unilateral fungal endophthalmitis. The patient was initially treated with intravenous Voriconazole, Ceftriaxone and linezolid for 4 weeks with resolution of his symptoms. This case highlights the emerging role of D. Pigrum as a pathogen in the immune compromised and specifically in the IDU population. This case further illustrates that although identification of fungal growth in vitreous fluid may include culture and PCR analysis, fungal endophthalmitis remains a clinical diagnosis and that fungal cultures have lower sensitivity.

Key words: *dolosigranulum pigrum;* endocarditis; fungal endophthalmitis; immunocompromised; people who inject drugs

Introduction

Dolosigranulum Pigrum is a gram positive, catalase and oxidase negative, non-hemolytic facultative anaerobic *Firmicute* bacteria. Since identification in 1993 and characterization as a commensal organism of the naso-respiratory tract, it has become the focus of investigation as a therapeutic probiotic due to the noted inverse relationship between *D. Pigrum* nasopharyngeal colonization and the incidence and severity of respiratory diseases, as well as in vitro inhibition of respiratory pathogens such as *S. Aureus* and *S. Pneumoniae* [1–5]. Despite this putative role as a probiotic, with advanced microbial identification methods such as 16S rRNA and MALDI-TOF, it has increasingly been associated with disease and as such is regarded as an emerging pathogen. The preponderance of case reports documenting pathology have involved focal eye diseases such as conjunctivitis, keratitis and corneal abscess [1,6–10]. The pathological spectrum extends to other critical illnesses: synovitis, sinusitis, pneumonia, cholecystitis and septicemia, however it has only rarely been associated with pre-existing immunocompromised states per se (Cystic fibrosis, HIV, Rheumatoid arthritis, Interstitial lung disease, chronic disease, extremes of age) [5,11–16]. Here, we present the case of *D. Pigrum* bacteremia in an injecting drug user (IDU), in constellation with immunocompromise from comorbid chronic HCV, fungal endophthalmitis and MRSA endocarditis.

Case Presentation

A 34-year-old male experiencing homelessness, with a history of IDU, untreated chronic hepatitis C and incompletely treated MRSA endocarditis, presented with a 2 week history of progressively deteriorating visual acuity of his right eye, associated with conjunctival redness and foreign body sensation. One week after symptoms began he reported treatment for 'pink eye' with erythromycin ointment. There was no drainage or eye pain at that time, however his symptoms reportedly acutely worsened thereafter, and now involved headaches, eye pain on movement and only being able to distinguish between light and dark. He denied fevers, eye drainage, photophobia, neck stiffness, confusion, chest pain, use of contact lenses or glasses or ocular trauma.

Two years previously, he had been admitted for MRSA septicemia and mitral valve endocarditis (seen on transthoracic echocardiography) and started on Vancomycin. However, at that time he left against medical

advice and was given a prescription for trimethoprim-sulfamethoxazole which he subsequently lost. His last use of IV heroin and methamphetamine was 2 months prior to his index admission. He endorsed diluting his injected substances with bottled water, filtering through cotton buds and occasionally licking his needles. He denied sharing needles. He denied exchanging sex for drugs or money.

In the emergency department, he was in no acute distress, was afebrile and hemodynamically stable (blood pressure 132/86, heart rate 100 beats per minute, respirations 20 per minute, temperature 36.9 degrees Celsius). Gross examination of his right eye revealed a cloudy appearing eye with surrounding conjunctival injection but no drainage or pus. With his right eye, he was only able to distinguish between hand motion, light and dark whereas visual acuity was 20/400 in his left eye. Extraocular movement was intact but painful. Pain was reproduced on direct palpation of the globe but not the surrounding orbit. There was no relative afferent pupillary defect. No corneal abrasion was found on fluorescein staining. Ophthalmoscopy of his left eye was unremarkable but on his right eye, revealed an anterior chamber with flare and 2+ cells. Posterior synechiae but no nodules were noted in his iris. Clumps of white debris consistent with fungal balls were noted in his posterior chamber. Although his optic nerve was faintly visible without obvious edema, his optic disc, macular and peripheral retinal vessels were obscured. Intraocular pressure was elevated at 15 mmHg on tonometry. Otherwise his physical exam was remarkable for a 3/6 systolic murmur loudest at the apex without radiation. His neck was supple. He had multiple well healed tattoos but no cuts, bruises, lesions or skin rashes. EKG was notable for an incomplete right bundle branch, also seen on previous admissions.

Initial laboratory abnormalities included a microcytic anemia (Hb 11.3 g/dL, MCV 73.7 fL), mildly elevated aspartate transaminase 47 units/L and alanine transaminase 80 units/L but otherwise his complete blood count and comprehensive metabolic panels were unremarkable (white blood count 7.0 K/uL, platelets 336 K/uL, C-reactive protein < 0.40 mg/dL, erythrocyte sedimentation rate 5 mm/Hr). A limited infectious workup with blood cultures, human deficiency virus (*HIV*), hepatitis C virus (*HCV*), herpes simplex virus (*HSV*), varicella zoster virus (*VZV*), Toxoplasma, Quantiferon, Lyme, rapid plasma reagin (*RPR*), Bartonella and an immunological workup were undertaken at that time including rheumatoid factor, ANCA, antiSSA/SSB. No acute imaging was performed.

He was admitted due to concern for acute fungal panuveitis. In addition to topical eye treatments with prednisolone acetate 1% every 4 hours, atropine 1%, moxifloxacin 0.5%, he was started on empiric systemic therapy with vancomycin, ceftazidime and voriconazole. He subsequently underwent vitrectomy with vitreous biopsy and culture, with clinical findings again consistent with fungal endophthalmitis. After 1 month there was no fungal or bacterial growth on vitreous fluid. The sample was ultimately not sent for broad range PCR identification.

He was found to be HIV negative on 4th generation testing as well as HIV-1 RNA viral load. His HCV viral load was 555,000. His immunologic workup was unremarkable. After 4 days, *D. Pigrum* was identified on MALDI-TOF from pinpoint colonies growing on 1 out of 4 bottles plated on blood agar under ambient conditions. Due to lower confidence in the MALDI-TOF results, the organism was sent out to a CDC reference laboratory for identification and sensitivity testing. Although he had a clinical diagnosis of fungal uveitis, the lack of microbiological diagnosis made it difficult to rule out additional contribution from either *D. Pigrum* or his history of *MRSA.* Previously, case reports have discussed the management of *D. Pigrum* infections using MICs and breakpoints of Streptococcus Pneumoniae [17]. Using these reported sensitivities, during the period that the CDC identification was pending and in order to afford enhanced CNS penetration for the other possible contributing pathogens (fungal, *D. Pigrum*, MRSA), the antibiotic regimen was transitioned to Voriconazole 400mg IV Q12H, Linezolid 600mg PO Q12H and Ceftriaxone for a planned 6 week course. After 14 days on this regimen his right eye exam had improved with resolution of ocular pain, conjunctival injection, macroscopic haziness, and an increase in visual acuity to 20/50 (previously could distinguish hand movement only). However, his liver enzymes increased from baseline up to AST ~140s, ALT ~140s and voriconazole was subsequently dose reduced to 200mg IV Q12H.

Transesophageal echocardiography was remarkable for a 0.6 x 0.1cm mobile echo density attached to the ventricular surface of the aortic valve. The mitral valve echodensity seen on previous admissions was not visualized. The CDC reference laboratory verified identification of the *D. Pigrum* and the following MICs reports (after incubation for 22 hours on cation-adjusted Mueller-Hinton broth + 5% lysed horse blood, under ambient conditions at 35ºC; MIC in brackets in ug/mL): Ampicillin (<0.03), Ceftriaxone (0.25), Levofloxacin (0.12), Linezolid (1), Penicillin (<0.016), Vancomycin (<0.25). Therapy for his chronic hepatitis C infection was deferred. He declined further evaluation by Cardiothoracic surgery for the remainder of his admission. During the course of his treatment the patient left against medical advice multiple times and on several occasions after returning was found to have a positive urine drug screen. He ultimately decided to leave against medical advice permanently and was given Amoxicillin 1gm Q8H, Voriconazole 200mg PO Q12H, Linezolid 600mg PO Q12H in order to complete his course.

Reference	Age, Sex*	Source/Entity of D. Pigrum infection	Comorbidity	Coinfecting pathogen
Sherret et al. [3]	61, M	Sepsis	OM, ESRD, CHF	-
Venkateswaran et al. [6]	2, F	Keratoconjunctivitis	-	-
Sampo et al. [7]	70, M	Corneal abscess	-	-
Sampo et al. [7]	78, F	Keratitis	-	-
Sampo et al. [7]	85, M	Keratitis	-	-
Sampo et al. [7]	71, F	Keratitis	RA	-
Sampo et al. [7]	?	Conjunctivitis	-	-
Monera-Lucas et al. [9]	49, F	Keratitis	HIV	
Laclaire et al. [10]	43, M	Eye		
Laclaire et al. [10]	?	PNA		
Laclaire et al. [10]	74, M	Blepharitis		
Laclaire et al. [10]	80, F	Blood		
Laclaire et al. [10]	Adult	Sepsis		
Laclaire et al. [10]	76, F	Eye		
Laclaire et al. [10]	1, F	Sepsis		
Laclaire et al. [10]	3, M	Sinusitis		

Laclaire et al. [10]	?	Blood		
Laclaire et al. [10]	85, M	UTI		
Laclaire et al. [10]	50, F	Eye		
Laclaire et al. [10]	83, F	Eye		
Laclaire et al. [10]	66, F	Sepsis		
Laclaire et al. [10]	63, M	Blood		
Laclaire et al. [10]	78, M	Blood		
Laclaire et al. [10]	?	Blood		
Laclaire et al. [10]	?	Nasopharyngeal		
Laclaire et al. [10]	2mo, M	Sepsis		
Laclaire et al. [10]	11, M	Blood		
Laclaire et al. [10]	?	Nasopharyngeal		
Laclaire et al. [10]	2mo, F	Eye		
Laclaire et al. [10]	2mo, M	Sepsis		
Laclaire et al. [10]	79, F	Gastric		
Laclaire et al. [10]	1.8, F	Blood		
Laclaire et al. [10]	?	Nasopharygneal		
Laclaire et al. [10]	?	Nasopharyngeal		
Mukhopadhyay et al. [11]	82, M	ILD	ILD	-
Lin et al. [12]	76, M	Cholecystitis	-	-
Lecuyer et al. [13]	73, M	HAP, sepsis	COPD, thrombocytopenia, hydrocarbamide	S. Aureus
Hoedemaeker et al. [15]	51, M	VAP	-	-
Hall et al. [16]	64, M	Synovitis	RA, MTX, Pred	
Hall et al. [16]	64, M	Synovitis	-	-
This report	34, M	Bacteremia	Fungal endophthalmitis, endocarditis, HCV	Fungal

CHF, congestive heart failure; COPD, chronic obstructive pulmonary disorder; ESRD, end stage renal disease; HAP, hospital acquired pneumonia; HCV, hepatitis C virus; HIV, human immunodeficiency virus; MTX, methotrexate; OM, osteomyelitis; Pred, prednisone; RA, rheumatoid arthritis; VAP, ventilator acquired pneumonia.

Table 1: Summary of case reports detailing disease (location/organ involvement, comorbidity) with Dolosigranulum Pigrum.

Summary	Number (%)
Total	**41 (100%)**
Total with bacteremia or septic	14 (34%)
Total with eye infections	13 (31%)
Total > 65 yo	15 (36%)
Total > 65 yo or immunosuppressed*	18 (43%)

* Immunosuppressed: includes systemic illness (eg. chronic obstructive pulmonary disorder/emphysema, End stage renal disease, HIV, Hepatitis C, Rheumatoid arthritis), on systemic steroids, chemo or immunotherapy (eg. methotrexate).

Table 2: Summary of characteristics of reported Dolosigranulum Pigrum infections.

Discussion

In this case of an IDU presenting with acute monocular vision loss consistent with fungal endophthalmitis, bacteremia with *D. Pigrum* was discovered incidentally but comorbid with aortic valve endocarditis and chronic hepatitis C. The main questions that arose for us from this scenario were - 'How did he contract *D. Pigrum* bacteremia ?'; 'What if any, was the pathogenic role played by *D. Pigrum* ?'; 'Does immunosuppression play any role in infections with *D. Pigrum* ?'.

Most bacterial infections in IDUs are commensal oral or skin flora, such as *Staphylococcus Aureus*, *Viridans Streptococci*, *Fusobacterium* spp., *Prevotella* spp. and the HACEK organisms [18,19]. Numerous practices surrounding the act of injection that result in poor hygiene can exacerbate the risk of contracting an infection from commensal bacteria. Examples include not cleaning the skin prior to injection; injection into sites heavily colonized with commensal bacteria; transmission of oral microbiota by licking their skin or needles, blowing out needles to clear clots, crushing capsules/tablets in their mouth during preparation; sharing or reusing needles. In one North American study from 2008, 32.5% of IDUs interviewed reported habitually licking needles prior to injection [19]. It seems likely that in the case of our patient, he contracted *D. Pigrum* bacteremia from previously licking his needles. Although now regarded as a commensal of the upper respiratory tract, *D. Pigrum* bacteremia has not previously been reported in the IDU population. In the IDU population, the prevalence of chronic diseases regarded as contributing to immune suppression is elevated compared to the general population [19]. As discussed further below, it is likely that together with wider access to advanced bacterial identification methods, the increased comorbidity associated with IDU, means that it is likely that *D. Pigrum* will be identified more frequently amongst the IDU population.

Pathologies associated with *D. Pigrum*, range from systemic phenomena including sepsis to focal diseases of the lungs and joints, with an apparent predilection for the eyes. The scarcity of reporting on *D. Pigrum* as a pathogen may be attributed to at least 3 factors: 1. Difficulty of

identification; 2. Putative role as a commensal and probiotic; and 3. Lack of virulence and role as an opportunistic pathogen.

D. Pigrum is a gram positive, catalase and oxidase negative, non-hemolytic facultative anaerobic bacterium that grows slowly on blood agar as grayish dome-shaped, cocci in pairs, tetrads and clusters (Figure 1). Prior to the widespread availability of 16S rRNA PCR and MALDI-TOF, identification was based on cumbersome and non-specific biochemical identification methods [13,15]. Given the similar appearance of *D. Pigrum* to either *Gemella* spp. or *Viridans Streptococci* on the plate and depending on the biochemical tests employed, it is possible that *D. Pigrum* has previously been mis-identified.

Multiple studies have emphasized that in contrast to being a pathogen, *D. Pigrum* is not only a commensal of the nasopharyngeal microbiome but might also serve as a possible probiotic aiding to reduce the incidence and severity of upper respiratory disease[[2–5]. In addition to colonizing the nasopharynx, in at least one study *D. Pigrum* was identified in the cerebrospinal fluid of multiple patients with multiple sclerosis (MS) without active disease.[18] In other studies, the population intensity of *D. Pigrum* in the upper respiratory system has been found to be inversely proportional to pathogens such as *S. Aureus* and *S. Pneumoniae* in disease states.[19] Mouse studies have also shown that *D. Pigrum* (together with *Corynebacterium* spp.) modulate the immune response to respiratory disease with Respiratory Syncytial Virus and SARS-CoV-2, with increased expression of antiviral and anti-inflammatory cytokines[[4]. Brugger et al demonstrated in vitro that *S. Aureus* and *S. Pneumoniae* growth was inhibited to varying degrees (and in the latter case in concert with *Corynebacterium* spp.) when added to plates pre-colonized with *D. Pigrum*[[2]. Although the mechanism of this inhibition has not been precisely elucidated, it is thought to be due to competitive consumption of auxotrophic nutrients. Interestingly, this inhibition was not observed when *D. Pigrum* was simultaneously co-plated with either *S. Aureus* and *S. Pneumoniae* as reflected by the current case as well as a previous report of coinfections with these organisms[13].

The low incidence of pathogenicity and putative role of *D. Pigrum* as a probiotic provide circumstantial evidence for the low virulence of this organism. Jorge and others identified 'asymptomatic' colonization of *D. Pigrum* in the CSF of multiple MS patients without identification of any specific associated pathogenic toxins or antibodies[18]. Additionally, in their elegant study Flores Ramos et al evaluated *in silico*, 28 available strains of *D. Pigrum* genomically and were not able to identify any virulence factors that were analogous to *S. Aureus* or *Enterococcus* spp., albeit it was noted that no strains from eye disease were evaluated[8]. Of those 28 strains analyzed, all were sensitive to beta-lactam, although about half were resistant to erythromycin. Although the patient in this report was administered topical erythromycin ointment for pink-eye, it is unlikely that this played any significant role in inducing either the fungal endophthalmitis or *D. Pigrum* bacteremia with which he was subsequently diagnosed.

Given this low apparent virulence, it would make sense that in disease *D. Pigrum* acts as an opportunistic agent in the setting of dysbiosis due to chronic illness, immune suppression or aging. Eighteen out of the 41 (~44%) of the reported cases that detail disease with *D. Pigrum* describes either pre-existing immunosuppression with either chronic illness, chemotherapy or age greater than 65 years of age (See appendix). The population aged greater than 65 years of age represented 15 (~36%) of those cases.

For the patient detailed in this report it is likely that the combination of licking his needles, chronic disease and immune suppression (HCV, untreated endocarditis) with acute illness (fungal endophthalmitis) put him at risk for *D. Pigrum* bacteremia. Given the paucity of microbiological growth/identification in either the vitreous fluid or cardiac valvular tissue, it was difficult to rule out *D. Pigrum* colonization in those tissues and contributing to those pathologies. This is perhaps especially true in the case of his endocarditis, wherein he had previously been septic with MRSA and had mitral valve vegetations but on re-presentation had an aortic valve vegetation, without MRSA identified any culture or DNA probe. His lack of septic response reflected his lower level bacteremia (1 of 4 blood culture bottles).

Although broad range PCR testing was not performed on vitreal fluid in this case, fungal endophthalmitis remains a clinical diagnosis. Although vitreal fungal cultures were negative, it is noted they have lower sensitivity (~13% compared to ~69% for PCR, in one study) whereas bacteria in vitreous fluid frequently grow well and those cultures have higher specificity[20]. This suggests again that bacterial infection, and specifically with *D. Pigrum* was unlikely to have contributed to his endophthalmitis. The lack of definitive lab findings is therefore disappointing but not unexpected.

Conclusions

D. Pigrum is a commensal organism of the upper respiratory tract that can cause disease, predominantly in populations of the immune suppressed or age greater than 65 years of age. Although it is under investigation as a probiotic, its role as an opportunistic infection will limit its application. To date, no specific virulence factors have been identified but most cases have either involved bacteremia/sepsis or eye disease in immunocompromised hosts. As such, as shown in this case, injection practices as well as the increased prevalence of chronic disease amongst the IDU population place them at increased risk of infection with *D. Pigrum* and it is likely that with wider access to advanced bacterial identification tools that this bacterium will be identified more frequently amongst this population. Genomic evaluation and other studies of these specific pathogenic strains may reveal virulence and represent an opportunity for ongoing research. Whilst prescribed breakpoints for sensitivities have yet to be specifically established or validated, given the evidence for treatment efficacy with beta-lactams, it seems prudent to continue using substituted values for *Streptococcus Pneumonia*e. This case further illustrates that although identification of fungal growth in vitreous fluid may include culture and PCR analysis, fungal endophthalmitis remains a clinical diagnosis and therefore no growth on fungal culture should be considered as a false-negative result.

Ethical Approval

Patient consent was obtained for the preparation of this manuscript. All identifying information has been omitted and IRB review was not necessary for this case report.

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Consent

The patient provided verbal and written consent for publication. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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All cited authors contributed equally to: care of the patient, writing, editing, submitting, and revising the manuscript.

Author Statement

All authors have seen and approved the final version of the manuscript being submitted. They warrant that the article is the authors' original work, hasn't received prior publication and isn't under consideration for publication elsewhere.

Declaration Of Competing Interest

The authors report no declarations of interest.

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