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Case report

Polymicrobial arcanobacterium haemolyticum intracerebral abscess: A case report and review of the literature

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ABSTRACT

Objective: This article describes a case of polymicrobial *Arcanobacterium haemolyticum* pharyngitis and sinusitis complicated by intracranial complications and reviews similar cases in the literature

Case summary: A 21-year-old immunocompetent male presented with symptoms of sore throat, rhinorrhoea, lethargy, headache, and rash. Imaging demonstrated sinusitis, pre-septal sinusitis, peritonsillar abscess formation, subdural empyema and cerebritis. He was managed with endoscopic sinus surgery, craniotomy for evacuation of subdural empyema and antibiotics. Microbiological samples demonstrated growth of *A. haemolyticum*, *strep. anginosus*, and *fusobacterium necrophorum*. He subsequently developed a cerebral abscess requiring stereotactic needle drainage. After a prolonged course of antibiotics, the patient was discharge and made a good recovery.

Discussion: A. haemolyticum is an uncommon cause of non-streptococcal pharyngitis that may occur alongside other microorganisms and is rarely associated with severe intracranial complications. This organism and its antibiotic susceptibility patterns should be considered in complicated upper respiratory tract infections in immunocompetent hosts. Penicillins and macrolide antibiotics form the mainstay of therapy for *A. haemolyticum*.

Introduction

Arcanobacterium haemolyticum is an uncommon organism classically associated with pharyngitis in immunocompetent adolescents and young adults [1,2]. In rare cases, *A. haemolyticum* may result in severe local or systemic disease [3]. *A. haemolyticum may present alongside other organisms, including fusobacterium necrophorum, a respiratory commensal implicated in a minority of A. haemolyticum infections* [3]. This article presents an illustrative case of a 21-year-old immunocompetent male with a polymicrobial *A. haemolyticum* intracerebral abscess following an upper respiratory tract infection and reviews the literature regarding *A. haemolyticum* intracerebral complications.

Case

A 21-year-old previously well male presented to the emergency department with a four-week history of sore throat, rhinorrhoea, lethargy, and headache. He had previously presented to his primary care physician and received a 10-day course of oral amoxicillin which had reportedly improved his symptoms. However, in the 24 h prior to admission, he developed a diffuse, blanching, erythematous rash over his arms, legs, and torso, which led to his presentation to the emergency department. On arrival, he was noted to have right sided tonsillar enlargement and blood work demonstrated an elevated total white blood cell count (15 × 10^9/L). His chest x-ray was clear and a rapid serum test for EBV (monospot) and a nasal swab PCR for SARS-CoV-2, influenza and respiratory syncytial virus were negative. His presentation was thought to be consistent with a viral infection and he was discharged with advice to follow-up with his primary care physician.

This patient represented to the emergency department 5-days later with ongoing fevers, rash, reduced oral intake and erythema of the sclera of his left eye. On examination he was noted to be tachycardic to 152

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beats/minute, febrile to 39.5 °C and markedly dehydrated. Initial lab results demonstrated leukocytosis with neutrophilia, elevated CRP of 138 mg/L and a lactate of 3.9 mmol/L. Further investigations demonstrated a bland urinalysis, negative serology for CMV, hepatitis C, HIV and syphilis, evidence of prior infection with parvovirus B19 and EBV, and immunity to hepatitis B and measles. A CT scan demonstrated left sided pre-septal cellulitis with extension of inflammatory changes into the left temporalis muscle and left sided sinusitis predominantly involving the left maxillary antrum and ethmoid air cells (see Fig. 1). Additionally, an enhancing right sided peritonsillar collection was noted. He was commenced on ceftriaxone 2 g IV BD and flucloxacillin 2 g IV q6hourly empirically. His peritonsillar collection was drained under local anaesthesia in the emergency department. Unfortunately, the patient experienced further deterioration, with increasing tachycardia, diaphoresis, new delirium and decrease in level of consciousness. Due to concern for meningoencephalitis, a lumbar puncture was performed which demonstrated leukocytosis with a neutrophil predominance (189 white cells, 70 % neutrophils, 30 % monocytes), elevated protein (0.68 g/L) and normal glucose (4.1 mmol/L). An MRI brain demonstrated extensive left sided paranasal sinus disease, abscess formation within the temporalis muscle, subdural empyema and left frontal cerebritis (see Fig. 2). Antimicrobial therapy was modified to ceftriaxone 2 g IV BD, vancomycin IV and metronidazole 500 mg IV q8hourly.

The patient underwent operative management by both ENT and neurosurgery. A left sided anterior endoscopic sinus drainage procedure was performed, and his antrum and anterior ethmoids were opened, exposing the frontal recess. Intranasal pus swabs were sent, and these cavities were irrigated copiously with saline. A left sided pterional craniotomy, including drainage of the temporalis abscess, was performed for washout of the subdural empyema. An external ventricular drain was placed to monitor intracranial pressure and the patient was transferred to the intensive care unit where he remained intubated for several days. During this time, initial blood cultures flagged positive for mixed growth of *A. haemolyticum* and *Fusobacterium necrophorum*. Intraoperative cultures demonstrated growth of both *A. haemolyticum* and *Streptococcus anginosus* from the left temporal abscess, the subdural collection, and swabs of his ethmoid and maxillary sinuses. Azithromycin 500 mg IV daily was added to his antimicrobial therapy as empiric management of *A. haemolyticum*. After several days, he was successfully extubated. Ultrasonography demonstrated no thrombophlebitis of his internal jugular veins, excluding Lemierre syndrome His neurological deficits at this time included right sided weakness, which rapidly improved, and expressive speech disturbance. His antibiotic therapy was rationalised to ceftriaxone 2 g IV BD and metronidazole 500 mg IV q8hourly based on antibiotic susceptibility testing, and he was stepped down to the ward.

Progress MRI completed 2 weeks after the initial procedure demonstrated evolution of the cerebritis into a ring-enhancing complex collection with internal fluid levels, evidence of mass effect and possible early ventriculitis (see Fig. 3). Stereotactic needle drainage of the abscess was performed. The intraoperative sample demonstrated 4 + white cells, but no organisms were visualised on gram stain and the bacterial cultures were negative. The patient tolerated this procedure well with no new neurological deficits. He was discharged to inpatient rehabilitation on intravenous ceftriaxone and oral metronidazole, which were continued for 3 weeks after needle aspiration of his cerebral abscess (to complete 6 weeks of IV therapy) at which time he was changed to oral amoxicillin 1 g TDS. At his last clinical review, 3 months after initial presentation, he had progressed well neurologically and was continuing to take the oral amoxicillin. He had no residual motor deficit, was continuing to participate with speech therapy and had returned to work. A repeat MRI scan at this point, demonstrated resolution of the intracranial abscess (see Fig. 4).

Discussion

A. haemolyticum was first isolated in 1946 in the Pacific Islands among patients with pharyngeal and skin manifestations [4]. It was originally classified as a *Corynebacterium* however was reclassified to a separate genus in 1982 on the basis of chemical and phenetic data [5]. It is a gram-positive (or gram-variable), catalase-negative, non-motile, non-spore forming, facultative anaerobic bacillus [4]. *A. haemolyticum* grows well on blood, or serum-enriched medium and its growth and haemolytic action are enhanced by the addition of carbon dioxide [4]. Humans are the primary reservoir for infection, although animal infection has been described [6]. Biochemical and spectroscopic testing can

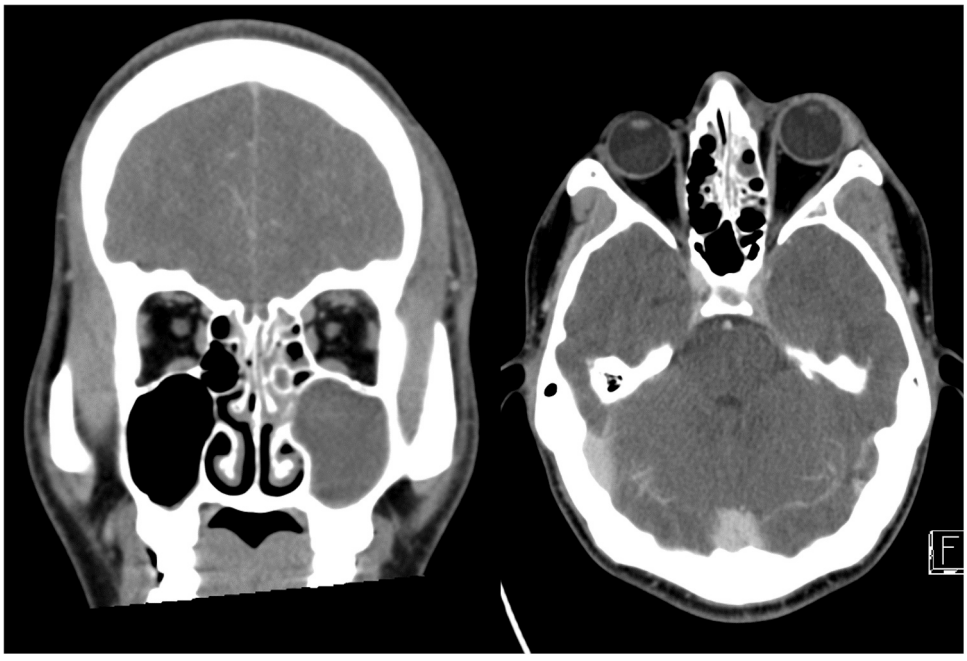

Fig. 1. Coronal and axial post-contrast CT images. CT imaging (coronal and axial post-contrast) demonstrating left sided pre-septal cellulitis with inflammatory changes of the left temporalis muscle with left maxillary and ethmoid sinuses.

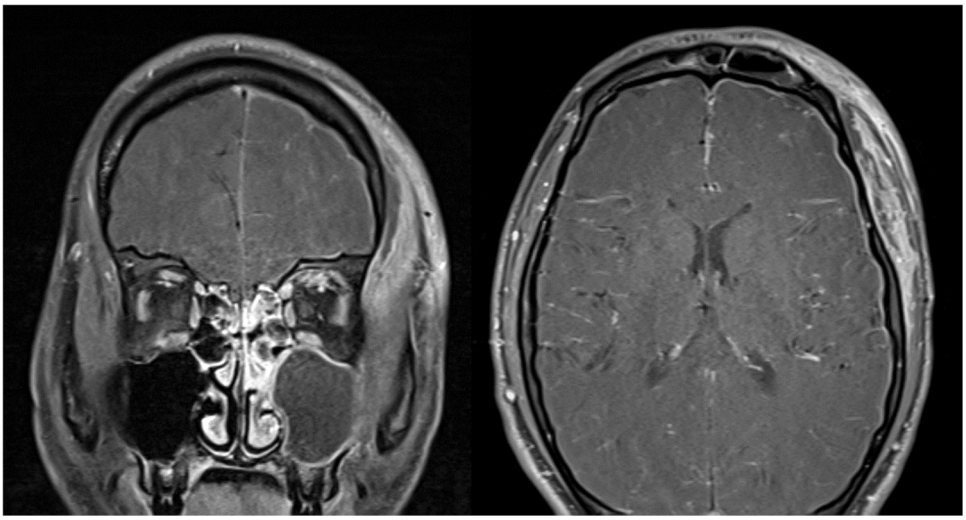

Fig. 2. *Coronal and axial T1 post-contrast MRI images.* MRI images (T1 weight, post-contrast coronal and axial) demonstrating left ethmoid and maxillary sinusitis, infection of the left temporalis muscle, left frontal cerebritis, and trace subdural empyema.

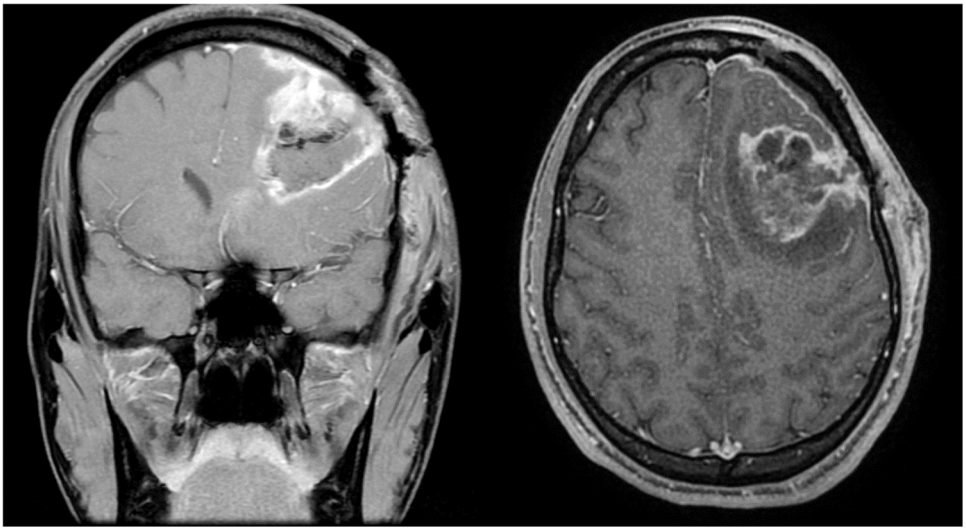

Fig. 3. *Coronal and axial T1 post-contrast MRI images.* MRI images (T1 weight, post-contrast coronal and axial) demonstrating progress of left frontal cerebritis into a complex abscess with internal fluid levels, peripheral enhancement and surrounding vasogenic oedema.

aid in the diagnosis of *A. haemolyticum*, including the reverse Christie-Atkins-Munch-Peterson (CAMP) (positive in *A. haemolyticum* infection) and matrix-assisted laser desorption ionisation time of flight mass spectrometry (MALDI-TOF MS) of the bacterium 16sRNA [7,8].

While *A. haemolyticum* has been implicated predominantly in non-streptococcal pharyngitis and wound infections [9], it has also been associated with severe sepsis and invasive disease including pulmonary abscesses, pleural empyemae, osteomyelitis, septic arthritis, spontaneous bacterial peritonitis and endocarditis [3,10–15]. *A. haemolyticum* is also associated with severe central nervous system infections; in one case series of patients with severe infection, CNS infections were present in 6 of 23 cases [9]. To date, 13 cases with central nervous system involvement have been reported in the literature (Table 1. Central nervous system involvement by local extension has been reported in relation to dental extraction [16], head trauma [17] and upper respiratory tract infection [9,18,19]. Although cases of isolated meningitis have been described [9,20], the natural history of most cases of central nervous system infection appears to reflect, as in this case, subdural empyema formation, cerebritis and progressive abscess formation necessitating surgical intervention [16–19]. In contrast, two cases have been reported of infective endocarditis with resultant septic emboli to the brain [10,21]. Both cases were complicated by intracerebral haemorrhage, possibly due to the formation of mycotic aneurysms. Central nervous system infection has also been described without a clear primary focus, particularly in immunocompromised patients such as those undergoing chemotherapy [20].

As noted in the cases presented in this report, *A. haemolyticum* infection may occur alongside other micro-organisms. Although *Fusobacterium necrophorum* and *strep. Anginosus* were identified in this patient's samples, given the burden of arcanobacterium in surgical and blood cultures, and the classic initial presentation, *A. haemolyticum* was thought to represent the primary pathogen. In line with this case, polymicrobial infection may occur in a significant minority of cases, as highlighted by a recent review of cases of *A. haemolyticum,* resulting in severe sepsis or complicated bacteraemia, which reports polymicrobial infection in 6 out of 20 cases [3]. In 5 of these cases, *Fusobacterium necrophorum* was also identified, as noted in the case presented. *Fusobacterium necrophorum* is considered part of the normal upper respiratory tract flora, although it may also cause local infections [27,28]. The factors that underly co-infection between these two organisms, and the

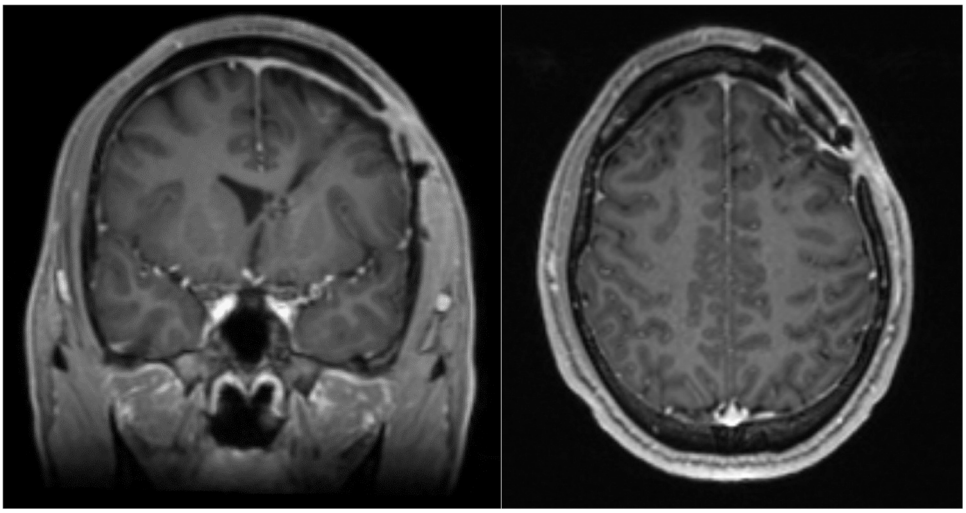

Fig. 4. *Coronal and axial T1 post-contrast MRI images.* MRI images (T1 weight, post-contrast coronal and axial) demonstrating significant improvement in the appearances of the left frontal cerebral abscess and resolution of mass effect.

Table 1
Cases of central nervous system *A. Haemolyticum* infection.

Case	Gender	Age	Organisms	Focus of infection	Intra-cranial complication	Antibiotic treatment	Duration of treatment	Ref.
1	M	24	*A. Haemolyticum, propionibacterium avidum*	Pharyngitis, sinusitis	Cerebral abscess	Ceftriaxone, metronidazole	7 weeks	Adams [18]
2	F	16	*A. Haemolyticum;* Bacteroides species, anaerococcus tetradius, dialister micraerophilus, erysipelotrichaceae species, Propionibacterium acnes	Head trauma	Subdural empyema, cerebral abscess	Vancomycin, ceftriaxone, metronidazole; amoxicillin/ clavulanate	6 weeks	Cortes-Penfield [17]
3	M	18	*A. Haemolyticum*	Dental extraction	Cerebral abscess	Penicillin G	4 weeks	Vargas [16]
4	M	20	*A. Haemolyticum, Epstein-Barr Virus*	Sinusitis	Subdural empyema, cerebral abscess	Ceftriaxone, metronidazole	9 weeks	Poplin [19]
5	F	21	*A. Haemolyticum*	Infective endocarditis	Cerebral abscess, intracerebral haemorrhage	Ceftriaxone, gentamicin	6 weeks	Wong [10]
6	M	15	*A. Haemolyticum, fusobacterium necrophorum*	Pharyngitis, tonsillitis	Meningitis, abducent palsy	Cefotaxime, metronidazole	Unclear	Skov [9]
7	M	58	*A. Haemolyticum*	Unclear, immunocompromised	Meningitis	Teicoplanin, penicillin, ceftazidime	16 days (terminated due to discharge against medical advice)	Minárik [20]
8	M	50	*A. Haemolyticum*	Infective endocarditis	Cerebritis, cerebral abscess, intracerebral haemorrhage	Penicillin G	7 days (terminated due to fatal intracerebral haemorrhage)	Chandrasekar [21]
9	M	24	*A. haemolyticum*	Sinusitis	Meningitis, abducent palsy	Gentamicin, ampicillin, metronidazole, probenecid	Unclear	Cook [22]
11	M	11	*A. haemolyticum, bacteroides melanogenicus*	Unclear	Cerebral abscess	Penicillin G, metronidazole	Unclear	Chhang [23]
12	M	17	*A. Haemolyticum, fusobacterium necrophorum*	Unclear	Meningitis, cerebral abscess, abducent palsy	Cephalothin, gentamicin, chloramphenicol	2 days (terminated due to fatal cerebral abscess)	Washington [24]
13	M	16	*A. haemolyticum*	Unclear	Cerebral abscess	Penicillin G	Unclear	Altmann [25]
14	M	65	*A. Haemolyticum, Bacteroides fragilis*	Unclear	Meningits, encephalitis	Penicillin, cloxacillin	Unclear	Ben- Yaacob [26]

factors that contribute to severe infection in some patients but not others have not yet been defined. *A. haemolyticum* has also been associated with Epstein-Barr virus infection, although evidence of EBV was not present in our case. In addition, some have suggested EBV, or its treatment with steroid therapy, might enhance the invasiveness of *A. haemolyticum* by impairing host immune defences [19,29]. *A. haemolyticum and fusobacterium necrophorum* have been implicated in Lemierre syndrome [30, 31] – a rare complication of oropharyngeal infection characterised by thrombophlebitis of the internal jugular vein, with metastatic infective foci. This was excluded in this case by negative ultrasonography of the internal jugular veins.

There is no consensus for the optimal antibiotic treatment of *A. haemolyticum*. A report of antimicrobial susceptibility testing of 138 isolates of *A. haemolyticum* found that all were susceptible to

phenoxymethylpenicillin, cephalosporins, erythromycin, azithromycin, clindamycin, doxycycline and ciprofloxacin, but were resistant to trimethoprim-sulfamethoxazole [32]. However, bactericidal tests suggest that some isolates of *A. haemolyticum* are tolerant to penicillins [33] and failure of microbiological eradication despite penicillin treatment has been reported (34. This may explain why our patient experienced initial improvement on amoxicillin, but subsequently represented with worsening symptoms several days later. In contrast, microbiological eradication was achieved in 87 % of patients with erythromycin, leading some sources to suggest macrolides as the primary treatment regimen [34,35]. For the patient presented in this report, azithromycin was added after identification of *A. haemolyticum*, until further management could be rationalised with susceptibility results.

Conclusion

A. haemolyticum should be considered in cases of severe infection complicating pharyngitis or sinusitis that may result from local extension or haematogenous spread. Our case demonstrates polymicrobial infection involving *A. haemolyticum* that progressed to subdural empyema, cerebritis and cerebral abscess requiring multiple neurosurgical intervention to attain source control. Because of these adverse and potentially life-threatening complications of *A. haemolyticum*, knowledge surrounding this organism and its effective treatment is crucial.

Ethical approval

Permission was received from the Chairman of the Human Research Ethics Committee for publication of this case report. A letter outlining this permission has been submitted.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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CRediT authorship contribution statement

Johnny Wong: Writing – review & editing, Supervision, Conceptualization. **Nila Dharan:** Writing – review & editing, Supervision, Conceptualization. **Michael Elliott:** Writing – review & editing, Supervision, Conceptualization. **Amshuman Rao:** Writing – review & editing, Writing – original draft. **Kate Lennard:** Writing – review & editing, Writing – original draft, Project administration, Data curation. **Nicholas Andrew Chang:** Writing – review & editing, Writing – original draft, Project administration.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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