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REVIEW

Chromobacterium violaceum: A Review of an Unexpected Scourge

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Abstract: *Chromobacterium violaceum* is a common environmental bacterium that rarely causes disease in humans but has a high fatality rate if it does. Due to the rarity of the cases, clinicians are often unaware of the rapid progression of *C. violaceum* infection and its unexpected antibiotic resistance pattern, which contribute to the failure of patient management. Our review provides the clinical characteristics, possible sources of exposure, and comorbidities and determines factors associated with survival. We gathered information on 132 cases of *C. violaceum* causing disease in humans published between 1953 and 2020. Patients were predominantly male with a median age of 17.5, interquartile range (IQR) of 5.0–40.0 years, and a third of them were known to have immune deficiencies or comorbidities. Portals of entry were mainly through a wound in the leg and feet (28.0%), the torso (8.5%), or hands and arms (6.8%). It is not uncommon to acquire infection through unintended contact with contaminated water or dust through the mouth or inhalation. The median incubation period is 4.0 days (IQR 2.0–8.0 days) with a duration of clinical course of 17.5 days (IQR 8.0–30.8 days). The high rate of positive blood cultures (56.1%) and abscesses in internal organs (36.4%) shows the significant severity of this disease. Sepsis and Bacteremia were related to mortality with a risk ratio (RR) of 5.20 (95% CI, 0.831–32.58) and 2.14 (95% CI, 1.05–4.36), respectively. Appropriate antibiotic use prevented death at a RR 0.33 (95% CI, 0.21–0.52). Most patients who recovered and survived were treated with aminoglycosides, fluoroquinolones and carbapenems. This review shows the malignant nature of *C. violaceum* infection and the need for clinicians to be aware and provide prompt source management for patients. Appropriate empiric and targeted antibiotic regiment guided by susceptibility test results is of vital importance.

Keywords: bacteremia, antibiotic, sepsis, mortality

Introduction

Chromobacterium violaceum is a free-living, soil and freshwater gram-negative bacillus found in tropical and subtropical regions. Human infection with this organism is not common; therefore, its presence as a causative agent is mostly overlooked in clinical practice unless a patient presents with severe, rapidly deteriorating sepsis and a remarkable purplish lesion and colonies in culture.[1–3] *C. vioaleceum* was regarded as a saprophytic organism and non-pathogenic in humans. Wooley first reported its pathogenic characteristics in 1905, observing septicemia in water buffaloes caused by these bacteria.[4,5] Later, more observations reported high human mortality with fatal septicaemia and skin lesions as well as internal organ abscesses. These manifestations have been reported from various parts of the world in the subtropic and the tropics, including Indonesia.[2,4–12] The

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organism has been characterized as an opportunistic pathogen causing severe and fatal manifestations in immunocompromised hosts.[2,13–16] However, this notion is challenged by other cases reporting fatal and severe clinical manifestations in non-immunocompromised hosts as well.[5,17–19]

The earliest observation of the bacterium was made by French scientists Boisbaudran and Gasser in the 1870s, who found rice flower coloration.[20] Another independent observation was made by Bergonzini of Modena University, Italy, in 1880, who observed a contaminated flask in his experiments with a deep purple substance that after extraction was insoluble with many solvents. This bacterium was named *Chromobacterium violaceum* and later record it in the *Bergey's Manual of Systematic Bacteriology as C violaceum*.[20] In 1976, two strains of *C. violaceum* with different colors of white and violet were isolated in Brazil and it was hypothesized that the violacein protected the bacterium against the sun.[21] There are nine other species of Chromobacterium genus that have been recognized to date, *C. subtsugae, C. aquaticum, C. haemolyticum, C. piscinae, C. pseudoviolaceum, C. vaccinii, C. amazonense, C. alkanivorans*, and *C. rhizoryzae*.[22]

The purple pigment called violacein, which is expressed by the bacteria, has created great research interests. Violacein is thought to confer the virulence of the bacteria and also to have antibiotic-inhibiting properties.[23,24] The violet pigment of the bacteria is assumed to be the causative agent of septicemia and fatal infections in humans and animals.[25] Bacteria with a dark violet color are resistant to various antibiotics, including vancomycin, ampicillin, and linezolid, and susceptible to colistin, oxacillin, gentamicin, norfloxacin, chloramphenicol, and amikacin.[26] Besides violacein, the bacteria also express metabolites that affect the growth of many gram-positive and -negative bacteria as well as produce an immunomodulatory effect.[25] Interestingly, these characteristics have been utilized to produce antibiotic attreonam.[27] Microbiologically, *C. violaceum* has biochemical characteristics similar to those of *pseudomonas* and *aeromonas* species,[24] which are related to the primary multi-drug characteristics that are challenging in treatment.[3,22]

Any part of the human body can serve as an inoculation site, with a traumatic break in the skin serving as the most common port of entry for infection in many reported cases, followed by regional lymphadenopathy, widespread bacteremia and abscess development in the visceral organs.[3,5,11,17,28,29] The mortality rate of *C. violaceum* infection has been reported to be 60%–80% in disseminated infection, and it was difficult to eradicate in most cases. Our review aimed to describe the clinical characteristics, possible sources of exposure, and comorbidities and to determine the factors that are associated with survival.

Materials and Methods

We carried out a literature search of published case reports or case series using two journal databases: PubMed and Google Scholar. The following search terms were used for searching relevant observational literature: "*Chromobacterium violaceum*" AND "infection" AND "Case Report." Exclusion criteria were 1) the report did not contain reported cases, 2) the report was written in a language other than English, 3) suspected case of chromobacterium infection but no culture evidence, 4) study reporting chromobacterium culture other than *Chromobacterium violaceum*, and 5) no information regarding final outcome of the cases. All reviewers did article selection and assessment. The outcome of the searches is summarized in Figure 1. De Siqueira reported three cases, but we included only one case, and the other two cases were not confirmed by bacterial isolation.[30] From the 111 studies reviewed, we collected information on 132 cases (Figure 1). Most are single-case reports,[1,2,4,5,7–9,11,15–18,28,31–80] except Huffam provided four cases,[81] Hagiya,[38] Karniyarakkal,[82] Macher,[83] Ma,[84] Tee,[85] and Teoh[18] each reported three cases, Al Khalifa,[31] Banik,[86] Feldman,[36] Jitmuang,[28] Macher,[83] Ognibene,[87] Pant,[88] Ponte,[89] Simo,[90] Sorensen,[91] Starr,[92] Suarez,[93] and Ti,[94] each reported two cases. Of these reports, 28 have been reviewed by Yang et al.[95] Based on these reports we collected information about demographics, incubation period, predisposing factors, comorbidities, clinical presentations, laboratory and microbiologic data, antimicrobial therapy, clinical course and outcome for analysis.

Definition

Case report year referred to the time when the paper was published. Sepsis was considered if the patient fulfilled the sepsis definition according to Bone et al (1992) as having two or more of 1) temperature > 38 or < 36 °C, 2) heart rate > 90, 3) respiratory rate > 20 and PaCO2 < 32, and 4) white blood cell count > 12.000/mm3, or < 4000, or > 10% Immature (band) forms.[96] The location of the first suspected port of entry was classified as in legs and

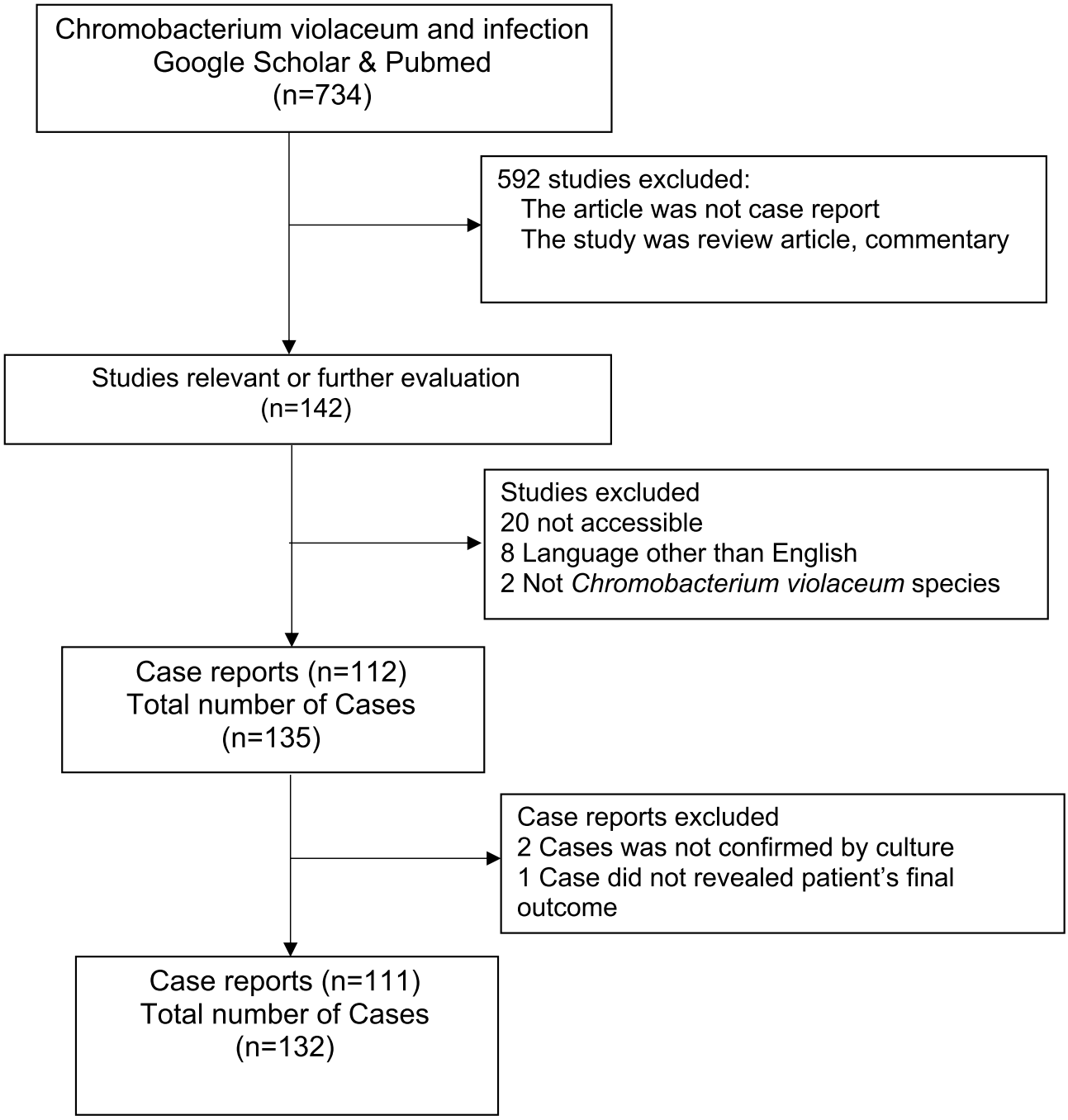

Figure 1 Enrolment and selection chart of the reviewed studies and cases.

feet, hands and arms, head and neck, neck and trunk, ingestion, inhalation, urinary tract. Skin abscess and cellulitis were defined as any skin infection in the form of pustules, vesicles, ecthyma, or ulcers including lymphadenitis. Comorbidity is defined as having a previously known disease, diagnosed during the course of *C. violaceum* infection or under other clinical care. Internal organ abscess was noted if reports contained information about the presence of at least an abscess in the liver, spleen, lung, brain or other organ based on imaging or post-mortem studies. The clinical course was defined as the interval from the onset of symptoms to death or discharge from hospital. A patient was considered to have a particular exposure if the author provided

information regarding the suspected way of acquiring the infection.

Antimicrobial susceptibility tests were obtained from the reviewed reports, if available. Interpretation of the results followed Clinical and Laboratory Standards Institute guidelines for non-Enterobacteriaceae gram-negatives).[97] An intermediate reaction was classified as non-susceptible. Antimicrobial therapy was considered appropriate if the treatment regimen included at least one antimicrobial agent described as an active agent against *C. violaceum* isolates in vitro. It was considered inappropriate if neither drug was sensitive against the isolated strain nor the patient did not receive the specified antimicrobial therapy. Healthcare-associated infection was defined if the infection occurred within hospital care or if the *C. violaceum* infection was related to a medical procedure. The year cut-off of 1990 was used as a -reference year because of the general availability of new antibacterial agents, including ciprofloxacin in 2010 because of a previous review made by Yang et al in 2011.[95]

Statistical Analysis

We presented data for dichotomous and categorical variables as frequencies and percentages. Continuous variables were presented as mean ± standard deviation and median and interquartile range (IQR). We used the Kolmogorov–Smirnov test to identify normal distributions. In the univariate analysis of variables associated with mortality, chi-square and Fisher's exact tests were used for binary variables and risk ratios were calculated. In multivariate analysis, binary logistics regression with backward method was employed to determine the factors most related to mortality outcome. The level of significance (α) was set at 0.05.

Results

We have listed and examined case reports from almost all continents, including India and its neighboring countries (n=31), Southeast Asia (n=20), North America (n=31), South America (n=10), Australia and New Zealand (n=8), Africa (n=8), Middle East (n=3) and Europe (n=6). Before 1990, most reports came from North America, but over the years, more reports came from all other countries (Table 1), including areas with cooler climate, such as Japan,[64] Korea,[56] China, Switzerland, the Czech Republic and Poland.

Our *C. violaceum* patients were mostly males from all age groups with a median of 17.5 (IQR 5.0 to 40.0) years old. The highest number of patients was in the younger age group between 0–9 years old and 20–49 years old. The incubation period was mostly short (median of 4 days, ranging from 1 to 80 days). Most patients sought medical care because of rapidly developing fever after a localized wound infection. Skin lesions developed into dark or purple colored vesicle abscesses accompanied by necrosis (see Figure 2A). In more than 75% of cases, routine hematologic examinations showed a pattern of ongoing bacterial infection with a leucocyte count of more than 11.000/mm^3 (Table 1). As the disease progressed to sepsis, the leucocyte count gradually increased in parallel with decreasing haemoglobin and thrombocyte values.[98]

Following the initial infection, hematogenic spread occurred, as shown by the high proportion of positive blood culture in these patients (Table 1). The hematogenic dissemination also caused multiple small pustules to occur in other areas of the body.[99,100] Severe sepsis and septic shock were the most common complications found, often followed by lung involvement with respiratory distress, requiring intensive care intervention.[18,24] Some patients presented with developing abdominal pain, related abscess formation in the visceral organs, mainly the liver and spleen as shown by imaging studies. Abscess and necrosis also occurred in the lungs following disseminated infection. Skin and soft-tissue infection in the facial area also led to brain abscesses.[101] Culturing the specimen mainly reveals purple colonies of *C. violaceum* (Figure 2B).

Antibiotic usage was variable, and a large proportion of use was not appropriate as indicated by the antibiotic sensitivity pattern (Table 1). There was a wide range of clinical course duration with a median of 18 days (range 2–264 days). Surviving patients were also affected by relapse of *C. Violaceum* infection, especially those with underlying chronic granulomatous disease.[102] Elimination of existing visceral abscesses took several weeks to months.[81,103] As guided by inflammatory markers, antibiotics needed to be taken until abscesses were fully cured.[28,103–105]

These patients acquired *C. violaceum* infection mostly in the community because of environmental exposures. Injuries to the extremities were the most common source (Table 2), mainly due to recreational accidents,[33] puncture wounds,[106] or insect bites.[100] Surprisingly, ingestion or inhalation were not uncommon. Al Khalifa et al reported fatal cases in two boys who acquired infection from a contaminated water storage tank.[31] Recreational activity in a contaminated lake

Table 1 Demographic, Clinical Characteristics, Culture Result and Outcome of 132 *Chromobacterium violaceum* Infection Cases Reviewed

Description		Patients (n=132)	%
Case report year	Before 1990	22	16.7
	Between 1990 −2010	47	35.6
	After 2010	63	47.7
Age (years)	0–9	44	33.3
	10–19	27	20.5
	20–49	37	28.0
	50-	24	18.2
Gender (n=130)	Male	99	76.2
Incubation period (days)	Median, IQR (n=49)	4.0	2.0–8.0
Duration of illness (days)	Median, IQR (n=124)	5.0	2.0–7.0
Duration of illness	0–3 days	50	37.9
	4–7 days	44	33.3
	More than 7 days	30	22.7
	Unknown	8	6.1
Clinical Manifestation*	Skin abscess and cellulitis	66	50
	Sepsis	102	77.3
	Respiratory tract infection	34	25.8
	Prolonged fever	16	12.1
	Abdominal pain	24	18.2
	Dysuria	9	6.8
	Other	12	9.1
White blood cell count (count/mm3)	Median, IQR (n=112)	14,500	9,400–20,800
White blood cell count (count/mm3)	< 4000	16	14.3
	4000–11,000	21	18.8
	11,000–20.000	44	39.3
	> 20,000	31	27.7
Abscesses found in internal organ*	Liver	42	31.8
	Lung	22	16.7
	Spleen	12	9.1
	Brain	4	3.0
Culture collected from*	Blood	74	56.1
	Pus	49	37.1
	Biopsy	19	14.4
	Tracheal & sputum	9	6.8
	Urine	12	9.1
	Other	6	4.5
Colony color (n=80)	Purple	62	77.5
	Purple black/metalic	7	8.8
	Purple translucent	3	3.8
	Translucent/other	8	10
Appropriate antibiotic treatment	Appropriate	82	62.1
	No	36	27.3
	Unknown	14	10.6
Duration of clinical course	Within 7 days	26	19.7
	8 to 21 days	53	40.2
	More than 21 days	48	36.4
	Unknown	5	3.8
Final outcome	Survived	50	37.9
	Died	82	62.1

Notes: *Any report is calculated as 1, no information regarded as none, IQR. interquartile range.

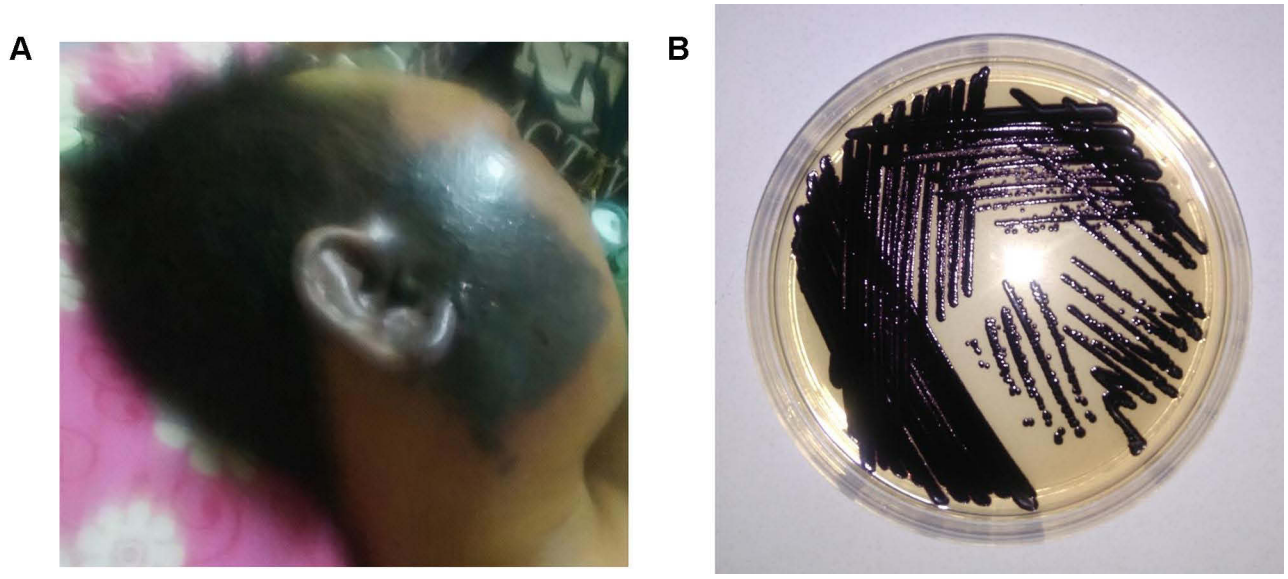

Figure 2 Picture of a patient with black purplish abscess in the right cheek extending to the right ear (**A**) and *Chromobacterium violaceum* shown on a culture plate (**B**).
Notes: Reproduced from Darmawan G, Kusumawardhani RY, Alisjahbana B, Fadjari TH. *Chromobacterium violaceum*: the Deadly Sepsis. *Acta Med Indones*. 2018;50(1):80–81.[2] Creative Commons Attribution 4.0 International License (https://creativecommons.org/licenses/by/4.0/legalcode).

also affected all three children in a family but without skin injury.[30] Dust from contaminated ground was also suspected to cause primary pulmonary infection with *C violaceum*.[1,107] In fact, five reported successful isolation of *C. violaceum* in the suspected lake, stream and soil sources. A more obscured mode of transmission was through the urinary tract in 12-year-old and 2-month-old children.[82,108]

C. violaceum healthcare-associated infections were also reported (Table 2). These patients were in hospital because of other illnesses or for a routine medical procedure. After a cervical spine surgery, one patient who remained febrile was shown to have *C. violaceum* infection in the femoral venous line.[18] Cases of urinary tract infection because of recurrent use of a catheter in a chronic kidney disease patients have also been reported.[88,109] Two cases of infection were probably acquired through the use of a humidified Venturi mask while in transfer to or from an intensive care ward.[38]

Antibiotic susceptibility testing revealed high rate of sensitivity to carbapenems, aminoglycosides, chloramphenicol, quinolones, tetracyclines and trimethoprim-sulfamethoxazole. Most first-line treatments for suspected gram-positive contamination of skin injuries, such as penicillin, beta-lactams, and clindamycin, were inactive against *C. violaceum* (Table 3).

Our serial cases showed that mortality decreased in the latter years (Table 4). It was almost 54.5% in the years before 1990 and 46.8% between 1990 and 2010, declining thereafter to 25.4%. Comparison of surviving patients versus fatal cases revealed that five variables were significant for the outcome: case report year, sepsis, bacteremia, appropriate antibiotic use, and duration of clinical course. We include all of these variables except the duration of the clinical course in an adjusted logistic regression model. Different categories of case report year were not associated with higher risk of death, while the other three variables remained important to note. Sepsis and bacteremia were associated with mortality with risk ratio of 5.20 (95% CI 0.83–32.58) and 2.14 (95% CI, 1.05–4.36), respectively. Appropriate antibiotic use prevented death at risk ratio 0.33 (95% CI, 0.21–0.52).

Discussion

We reviewed cases of *C. violaceum* reported from five continents, showing the universal distribution of this successful species. It is known that *C. violaceum* are distributed in warmer climates of the world between 35° latitude in the north and south.[29] The earliest human case reports came from Malaysia,[110] followed by cases in the southeastern United States among patients with chronic granulomatous disease and in healthy individuals.[83,89] However, various new cases have been reported from colder regions, such as Japan and Europe.[38]

Table 2 Location of Suspected Port of Entry and Comorbidity in Community Acquired and Health Care Associated *C. violaceum* Infection

	Community Acquired		Health Care Associated	
Suspected port d'entrée				
Legs and Feet lesion	33	28.0	2	14.3
Lesion in the torso	10	8.5	1	7.1
Head & Neck lesion	9	7.6	0	-
Ingestion	8	6.8	1	7.1
Arms & hand lesion	8	6.8	1	7.1
Inhalation/Lungs	6	5.1	3	21.4
Urinary tract	4	3.4	3	21.4
Ear	3	2.5	1	7.1
Eye	2	1.7	0	-
Unknown	35	29.7	2	14.3
Total	118	100.0	14	100.0
Comorbidity				
None	29	24.6	1	7.1
Chronic Granulomatous Disease	14	11.9	0	-
History of recurrent infection	7	5.9	0	-
Diabetes Mellitus	4	3.4	1	7.1
Hypertension, heart failure/other	3	2.5	1	7.1
G6PD	3	2.5	0	-
Systemic lupus erythematosus	1	0.8	0	-
Chronic liver disease	1	0.8	0	-
Chronic lung disease	0	-	2	14.3
Hematologic malignancy	0	-	2	14.3
Post-surgical intervention	0	–	2	14.3
Chronic kidney and urinary tract disease	0	–	2	14.3
No information	56	42.4	3	21.4
Total	118	100.0	14	100.0

Table 3 Antibiotic Susceptibility Pattern

Antibiotic	Tested (N=120)	Sensitive (n)	Sensitive %
Penicillin	9	0	0.0
Ampicillin	52	1	1.9
Amoxicillin/clavulanate	19	5	26.3
Ampicillin/sulbactam	8	0	0.0
Carbenicillin	7	4	57.1
Ticarcillin	8	2	25.0
Piperacillin	10	6	60.0
Mezlocillin	2	1	50.0
Tetracycline	**9**	**8**	**88.9**
Ticarcillin/clavulanate	8	2	25.0
Piperacillin/tazobactam	41	25	60.1
Cephalothin (or cephalexin/ cefazolin)	33	0	0.0
Cefuroxime	26	2	7.7
Cefoxitin	11	1	9.1
Cefotaxime	31	4	12.9
Ceftriaxone	38	5	13.2
Ceftazidime	54	10	18.5
Cefepime	36	16	44.4
Aztreonam	20	7	35.0
Imipenem	**57**	**48**	**84,2**
Meropenem	**34**	**32**	**94.1**
Ertapenem	9	5	55.6
Gentamicin	**92**	**79**	**85,9**
Netilmicin	**16**	**15**	**93.8**
Amikacin	65	51	78,5
Tobramycin	29	23	79,3
Kanamycin	13	10	76.9
Isepamicin	**7**	**6**	**85.7**
Clindamycin	5	4	80.0
Ciprofloxacin	**77**	**75**	**97.4**
Levofloxacin	**22**	**22**	**100**
Ofloxacin	**11**	**11**	**100**
Norfloxacine	**5**	**5**	**100**
Chloramphenicol	**46**	**44**	**95,7**
Tetracycline	**21**	**19**	**90,5**
Trimethoprim/sulfamethoxazole	**79**	**72**	**91.1**
Polymyxin B (or colistin)	9	2	22.2

Note: Figures marked in bold indicate a high proportion (>80%) of *C. violaceum* susceptible to the antibiotic.

Our *C. violaceum* infection cases are predominantly in young, male patients. This represents a group of people who are active outdoors and prone to injury. This pattern did not change much compared to case reviews by Yang et al in 2011[95] and the earlier reports in the United States, either among patients with chronic granulomatous disease[83] or non-immunocompromised individual.[89,111]

Infection with *C. violaceum* often occurred in a recreation or work environment. Contact with water through skin injury or ingestion is one of the most common routes to infection. In the water where *C. violaceum* was found, it was continuously detected through most of the observation time irrespective of human fecal contaminants as represented by *E. coli*.[112] The soil is also a natural habitat where *C. violaceum* can be isolated in abundant numbers but not in vegetables or other foods.[113] It is therefore reasonable to suspect that inhalation of dust outdoors could cause primary pulmonary infection with *C. violaceum*. It would also be reasonable to assume that *C. violaceum* may find an optimal environment in hospital equipment, such as catheters[88] and Venturi masks.[38]

Chronic granulomatous disease is a well-known predisposing factor to infection by *C. violaceum*.[83] Being immunocompromised may underlie other cases, as some reported history of recurrent infection and skin abscess[2,81,82] and diabetes mellitus.[114] Chronic granulomatous disease patients are susceptible to infection caused by catalase-producing bacteria such as

Table 4 Association Risk Factor with Mortality Outcome

Description	n	Dead (n=50)	Recovered (n=82)	p-value	RR (95% CI RR)
Case report time					
Before 1990	22	12 (54.5)	10 (45.5)		1 (ref)
From 1990 −2010	47	22 (46.8)	25 (53.2)	0.539	0.86 (0.53–1.40)
After 2010	63	16 (25.4)	47 (74.6)	**0.009**	0.47 (0.26–0.82)
Age (years)					
0–9	44	19 (43.2)	25 (56.8)		1 (ref)
10–19	27	9 (33.3)	18 (66.7)	0.422	0.77 (0.41–1.45)
20–49	37	15 (40.5)	22 (59.5)	0.811	0.94 (0.56–1.57)
50-	24	7 (29.2)	17 (70.8)	0.278	0.68 (0.33–1.37)
Gender (Female)*		n=49	n=81		
Female	31	12 (38.7)	19 (61.3)		1 (ref)
Male	99	37 (37.4)	62 (62.6)	0.965	0.97 (0.58–1.61)
Comorbidity*		n=21	n=52		
No	30	8 (26.7)	22 (73.3)		1 (ref)
Yes	43	13 (30.2)	30 (69.8)	0.742	1.13 (0.54–2.39)
Community or healthcare associated					
Health care assoc.	14	4 (28.6)	10 (71.4)		1 (ref)
Community	118	46 (39.0)	72 (61)	0.478	1.36 (0.58–3.22)
Sepsis					
No	30	2 (6.7)	28 (93.3)		1 (ref)
Yes	102	48 (47.1)	54 (52.9)	**0.005**	7.06 (1.82–27.35)
Bacteraemia					
No	58	7 (12.1)	51 (87.9)		1 (ref)
Yes	74	43 (58.1)	31 (41.9)	**<0.001**	4.82 (2.34–9.90)
Abscesses found in internal organ*		n=45	n=73		
No	84	31 (36.9)	53 (63.1)		
Yes	48	19 (39.6)	29 (60.4)	0.759	1.07 (0.69–1.68)
Appropriate Antibiotic*		n=45	n=73		
No	36	30 (83.3)	6 (16.7)		1 (reff)
Yes	82	15 (18.3)	67 (81.7)	**<0.001**	0.22 (0.14–0.35)
Duration of illness*		n=49	n=75		
0–3 days	50	21 (42.0)	29 (58.8)		1 (ref)
4–7 days	44	17 (38.6)	27 (61.4)	0.741	0.92 (0.56–1.51)
More than 7 days	30	11 (36.7)	19 (63.3)	0.642	0.87 (0.49–1.55)
Duration of clinical course*		n=50	n=77		
Less 7 days	26	21 (80.8)	5 (19.2)		1 (ref)
8–21 days	53	16 (30.2)	37 (69.8)	**<0.001**	0.37 (0.24–0.59)
More than 21 days	48	13 (27.1)	35 (72.9)	**<0.001**	0.34 (0.20–0.55)

Note: Rows marked with * indicate lower number (n) subject observed. Figures marked in bold indicate a statistically significant difference (p<0.05).
Abbreviations: RR, risk ratios; Ref, reference group to calculate risk ratios.

C. violaceum because of their inability to carry out adequate oxidative metabolism during phagocytosis.[115] However, epidemiologically, we still cannot determine the strength of this association because of the small number of reported cases, and many reports did not confirm the immune status of their patients.[1,33,37] Other than the host immune condition, significant exposure to the bacteria seems to play a role as well. There are at

least two reports where several healthy children acquired the infection from a shared community source.[30,31]

Once infection is established, *C. violaceum* rapidly spreads locally and through the bloodstream to internal organs, causing multiple abscesses and sepsis.[22,110] If the mode of entry is inhalation, the patients rapidly develop upper respiratory tract infection followed by severe pneumonia[31,35] Diarrhea and abdominal pain could be manifestations if *C. violaceum* were transmitted through ingestion[116,117] The virulence of these bacteria is shown as well by the high proportion of positive blood cultures in the case reports. The success of the invasion by these bacteria partly results from an immunomodulatory effect of secreted substances[20] as well as a type III secretion system-mediated effect that damages host tissues, including hepatocytes.[22]

The most critical factors for the bacterium's high fatality rate are its severity, unawareness of the clinical characteristics of infection, and inappropriate antibiotic use. With the increasing knowledge of the disease and more antibiotic alternatives, we and others have shown that mortality has decreased.[22] Initial empirical antibiotic choice is important as *C. violaceum* are intrinsically resistant to many first-line antibiotics, mainly the penicillin group and beta-lactams.[1,23,32,37] Although less common, some strains have also been reported to be resistant to imipenem[29] and aminoglycosides.[7,9,16,107] Most patients who recovered and survived were treated with aminoglycoside, fluoroquinolone, and carbapenem antibiotic regimens as guided by culture and susceptibility test results.[10,14,15,32]

Conclusion

C. violaceum is a natural inhabitant of the water and soil and rarely causes disease. However, special care needs to be taken in dealing with patients with ulcers and abscesses and a history of environmental exposure, especially those with the disproportionately rapid development of sepsis, respiratory distress, and visceral pain. The organism shows high rate of susceptibility to aminoglycosides, carbapenems, quinolones, chloramphenicol, tetracyclines, and trimethoprim sulfamethoxazole. Early source control and prompt provision of the appropriate antibiotic regimen guided by culture and susceptibility test results are important in the management of this disease. Proper long-term follow-up care aimed at eliminating remaining bacteria is also vital to prevent relapses.

Disclosure

The authors report no conflicts of interest for this work.

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