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Case Report

Fatal bacteremia by *neisseria cinerea* in a woman with myelodysplastic syndrome: a case report

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Abstract: *Neisseria cinerea* has been rarely found in blood cultures. In this study, we are reporting a case of a Myelodysplastic Syndrome (MDS) patient in whose blood *Neisseria cinerea* was found and led a fatal consequence. This case will call our attentions to the uncommon pathogens in the pathogenicity of end-stage patients.

Keywords: *Neisseria cinerea*, myelodysplastic syndrome, bacteremia

Introduction

Neisseria cinerea is a commensal Neisseria sp., characterizing by gram-negative, oxidase-positive and catalase-positive. It was first discovered in 1906 in Europe and was often misidentified in laboratories because of its resemblance to *Branhamella catarrhalis* and *Neisseria gonorrhoeae.* Although it is classified as non-pathogenic bacterium, cases of its causing lymphadenitis in an immunosuppressed youth, ophthalmia neonatorum in a newborn, bacterial peritonitis in a 38-year-old man with Chronic Ambulatory Peritoneal Dialysis(CAPD), proctitis in an 8-year-old boy and nosocomial pneumonia in a 25-year-old man have been reported [1-5]. In this report, we first present a case of a woman with myelodysplastic syndrome (MDS), in whose blood *Neisseria cinerea* which led a fatal consequence was isolated.

Case report

Starting in October 2013, a 59-year-old retired woman started to feel dizziness, lack of strength and appetite. Overtime, with worsening symptoms, she visited Nanjing Drum Tower Hospital for treatment. Blood examination showed that WBC 1.1×10^9/L, Hb 34 g/L, and PLT 27×10^9/L. Cytogenetic examination demonstrated a chromosome alteration of 20q- (6%), while -5/5 q-, -7/7 q-, and +8 alterations were not found. She was diagnosed with MDS-REAB-II after confirmation with the bone marrow examination. Immediately after, chemotherapy regimen as Decitabine + CAG was employed with the following dosage: Decitabine, 42 mg intravenously guttae (ivgtt), (d1, d3, d5); aclacinomycin, 20 mg ivgtt, (d1, d3, d5); cytarabine, 17 mg q12hih, (d1-d5). Reexamination of bone marrow suggested a favorable prognosis with decreasing in bone marrow hyperplasia and low ratio of primitive cells. Thus, since November 14, 2013, consolidation therapy with Decitabine + CAG as the same dosage as before was carried out. However, in this time, the chemotherapy caused patient to experience myelosuppression and pulmonary fungal infection. With following anti-infection therapy, the patient relieved. Two weeks before she was admitted again, the patient started to feel dizzy and weak again with occasional chest congestion. This time, she was admitted to the affiliated hospital of Nanjing University of Chinese Medicine on March 8, 2014. Soon after the administration, symptoms were worsening with high fever, coarse breath sounds, disseminated subcutaneous hemorrhage and sternal tenderness. Immediately, the patient was subjected to Imipenem and Cilastatin Sodium (0.5 mg ivgtt, q6h) for anti-infection therapy, as well as the recombinant human granulocyte colony stimu-

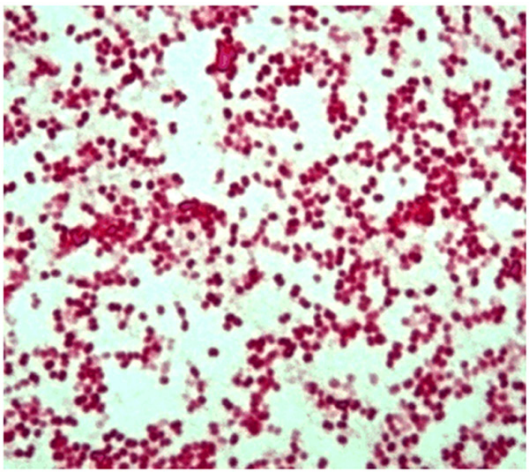

Figure 1. Gram-negative diplococcus were observed under microscope (1000×).

lating factor (G-CSF) for rising leukocytes. However, the results were not optimistic and the patient was threatened by infection and haemorrhage. Sustaining for 13 days, on the morning of March 21 2014, the patient arose symptoms of obnubilation, dilation of both pupils, loss of response to light and sound, and subsequent respiratory arrest. The patient was declared death after all rescue measures proved ineffectual. During hospitalization, all pathogen detection were negative except for a blood culture alarmed positive on March 20 2014 for *Neisseria cinerea,* which was believed to be the fatal pathogen leading death to the patient.

Microbiological investigation

Blood samples were referred to Department of Microbiology of the affiliated hospital of Nanjing University of Chinese Medicine. On March 20, 2014, one blood culture (BD FX system) alarmed positive and showed results with a growth of gram-negative diplococcus (**Figure 1**). The positive culture was inoculated to blood agars, chocolate agars and china blue agars for culture. 24 hours later, pale grey, translucent colonies grew well on both blood agars and chocolate agars (**Figure 2**). The isolate was an oxidase-positive, catalase positive, gram-negative diplococcus and then was confirmed as *Neisseria cinerea* with NH test kit and VITEKCOMPACT-2 identification system (No. 022-0400000, 99% identification confidence). The detailed identification results were shown in **Table 1**.

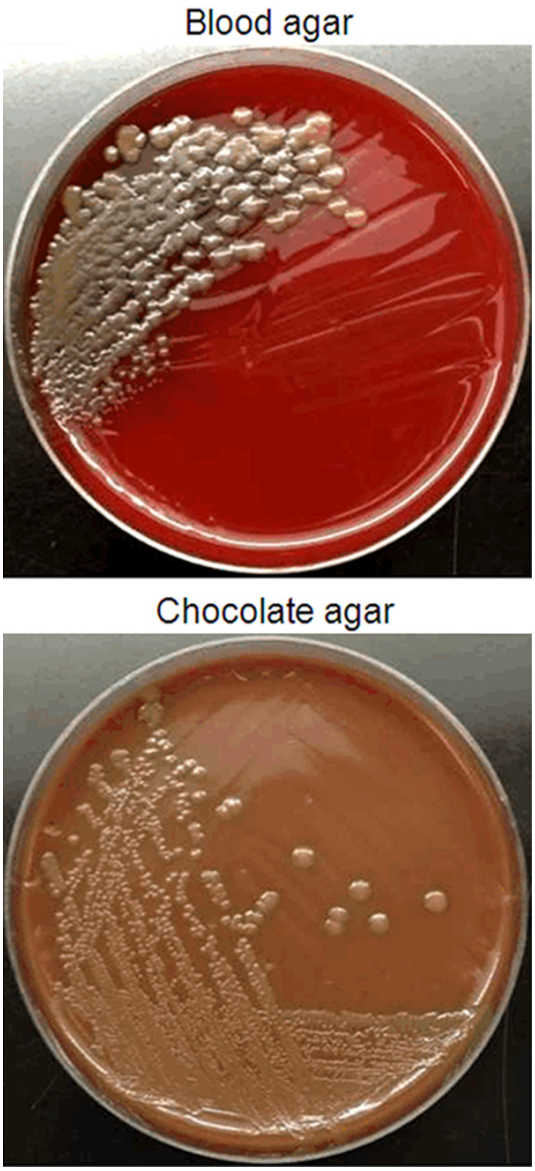

Figure 2. Glistening gray colonies with smooth margins on agars.

Discussion

Neisseria cinerea normally colonizies the upper respiratory tract of humans [6]. Although, associating single case reports with lymphadenitis [1], proctitis [4], conjunctivitis [7], and ophthalmia neonatorum [2], this organism is commonly recognized as normal flora in oropharynx rather than a cause of disease. Bacteremia due to *Neisseria cinerea is rarely* found except for three cases reported previously. The first was in a 2 1/2-year-old boy with otitis media and pneumonia [8]; the second was a 17-year-old boy with meningitis accompanied by septice-

Table 1. Biochemical details

1	ArgA*	-	2	GGT	-	3	LysA	-	4	dGAL	-	5	LeuA	+	6	ELLM	-
7	PheA	-	8	ProA	+	9	PyrA	-	10	TyrA	-	11	APPA	-	12	dGLU	-
13	GLYG	-	14	dMNE	-	15	dMAL	+	16	SAC	-	17	NAG	-	18	URE	-
19	BGALi	-	20	ODC	-	21	AARA	-	22	PVATE	-	23	PHC	-	24	dMLT	-
25	MTE	-	26	IGUM	-	27	PHOS	-	28	DRiB2	-	29	OPS	-	30	dXYL	-

*ArgA, Arginine Arylamidase; GGT, Gamma Glutamyl Transferase; LysA, L-Lysine Arylamidase; dGAL, d-Galactose; LeuA, Leucine Arylamidase; ELLM, ELLMAN; PheA, Phenylalanine Arylamidase; ProA, L-Proline Arylamidase; PyrA, L-Pyrrolydonyl Arylamidase; TyrA, Tyrosine Arylamidase; APPA, Ala-Phe-Pro Arylamidase; dGLU, d-Glucose; GLYG, Glycogene; dMNE, d-Mannose; dMAL, d-Maltose; SAC, Saccharose/Sucrose; NAG, N-Acetyl-d-Glucosamine; URE, Urease; BGALi, Beta-Galactopranosidase Indoxyl; ODC, Ornithine Decarboxylase; AARA, Alpha-Arabinosidase; PVATE, Pyruvate; PHC, Phosphoryl Choline; dMLT, d-Malate; MTE, Maltotriose; IGLM, L-Glutamine; PHOS, Phosphatase; DRIB2, d-Ribose 2; OPS, Phenyl phosphonate; dXYL, d-Xylose.

mia [9]; and the last was a 47-year-old man with a long history of alcohol abuse who was followed by death [8]. No any else was reported until today, in this report, we described what we believed to be the first case of a MDS patient in whose blood we isolated *Neisseria cinerea*. How did the microbe enter the blood is still a question? One possibility is that, at final stage of her disease, with enervating immune system, *Neisseria cinerea* may penetrated from respiratory tract to the blood stream, proliferating, and led bacteremia. However, the underlying mechanism by which *Neisseria cinerea* penetrated into blood is still need further investigation. Additionally, works must be done to exclude other reasons and determine *Neisseria cinerea* bacteremia as the main cause of the patient's death.

Because of the rarity of *Neisseria cinerea*'s isolation in clinical laboratories, care must be taken to distinguish this organism from *N. gonorrhoeae*, and other asaccharolytic gram-negative diplococci such as *N. flavescens* or *B. cat arrhalis*. Laboratory microbiologists must be familiar with this organism's colonial characteristics and variable biochemical reactions, especially when they are isolated from non-oropharynx sites.

Disclosure of conflict of interest

None.

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