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CASE REPORT

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Infective endocarditis due to nasal septal perforation during home oxygen therapy

Kyota Shinfuku[1] | Naoki Takasaka[1] | Ryutaro Ohashi[2] | Taiki Fukuda[3] | Makiko Takatsuka[1] | Ryo Sato[1] | Mitsuyoshi Mita[4] | Tsukasa Hasegawa[1] | Masami Yamada[1] | Yumie Yamanaka[1] | Yusuke Hosaka[1] | Kai Ryu[1] | Tokio Hoshina[5] | Hiroshi Takeda[1,6] | Takeo Ishikawa[1] | Jun Araya[7]

[1]Division of Respiratory Diseases, Department of Internal Medicine, The Jikei University Daisan Hospital, Tokyo, Japan

[2]Division of General Practice, Department of Internal Medicine, The Jikei University Daisan Hospital, Tokyo, Japan

[3]Department of Radiology, The Jikei University Daisan Hospital, Tokyo, Japan

[4]Division of Cardiology, Department of Internal Medicine, The Jikei University Daisan Hospital, Tokyo, Japan

[5]Department of Infectious Diseases and Infection Control, The Jikei University School of Medicine, Tokyo, Japan

[6]Department of Infectious Diseases and Infection Control, The Jikei University Daisan Hospital, Tokyo, Japan

[7]Division of Respiratory Diseases, Department of Internal Medicine, The Jikei University School of Medicine, Tokyo, Japan

Correspondence
Kyota Shinfuku, Division of Respiratory Diseases, Department of Internal Medicine, The Jikei University Daisan Hospital, 4-11-1 Izumihoncho Komae, Tokyo 201-8601, Japan.
Email: shinfuku.112@gmail.com

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Abstract

We report a case of infective endocarditis (IE) due to nasal septal perforation during Home oxygen therapy (HOT). A 64-year-old man with a history of interstitial pneumonia (IP) and on HOT was hospitalized for dyspnea. Methicillin-sensitive *Staphylococcus aureus* (MSSA) was repeatedly detected in blood cultures. Echocardiography revealed tricuspid valve vegetation and regurgitation. The patient was diagnosed with IE, according to the modified Duke criteria. A full-body examination revealed nasal septal perforation and MSSA was isolated from the nasal cavity. The patient was treated with cefazolin and clindamycin. However, he developed aspiration pneumonia and subsequently died. The portal of entry of MSSA was damaged nasal mucosa, caused by dryness and curettage of the dried nasal mucus during HOT. Nasal septal perforation, a potential complication of HOT, may cause severe bacterial infections. Consequently, diligent nasal care is crucial during HOT.

KEYWORDS
home oxygen therapy, infective endocarditis, nasal septal perforation

INTRODUCTION

Home oxygen therapy (HOT) uses dry oxygen, which causes nasal dryness and dried nasal mucus.

Humidification is provided with HOT in high-flow oxygen, but may not be provided at low flows.[1] In addition, there is little awareness regarding the serious complications associated with nasal dryness. We report a case of infective endocarditis (IE) due to nasal septal perforation caused by nasal dryness and curettage of dried nasal mucus during HOT.

CASE REPORT

A 64-year-old man who underwent HOT for interstitial pneumonia (IP) visited our hospital's emergency room because of dyspnea for 2 days and was admitted. We had been treating

him for idiopathic IP for 4 years. The patient also had a history of xeroderma. One year prior, he underwent HOT (nasal cannula, 0.5 L/min resting, and 4 L/min exercise). Thereafter, oxygenation worsened as IP progressed, and the patient was using home oxygen at a rate of 4 L/min for an increasing amount of time. Additionally, the patient repeatedly complained of nasal dryness and increased levels of dry nasal mucus.

The patient's vital signs on admission were as follows: SpO_2 89% (3 L/min); body temperature 38.6°C; blood pressure 118/92 mmHg; respiratory rate 26 /min; Glasgow coma scale 15 (E4V5M6). Blood tests revealed the following: neutrophil count 9282/μL (normal range, 1700–6300/μL); C-reactive protein 9.46 mg/dL (normal range, <0.14 mg/dL). Chest computed tomography (CT) revealed bilateral diffuse cystic lesions of unequal size immediately below the pleura and traction bronchiectasis. No new pneumonia was detected.

Although the cause of dyspnea was initially unclear, tazobactam/piperacillin (TAZ/PIPC) was initiated. Methicillin-sensitive *Staphylococcus aureus* (MSSA) was detected in two sets of blood cultures the following day. Transthoracic echocardiography revealed tricuspid valve vegetation and regurgitation (Figure 1). Additionally, the tricuspid regurgitation pressure gradient increased to 65 mmHg, suggesting pulmonary hypertension (PH). The patient was diagnosed with IE, according to the modified Duke criteria. To identify the entry portal of MSSA, a whole-body physical examination and an enhanced CT scan of the thorax and abdomen were performed, but no obvious findings were noted. Based on the patient's complaints, nasal endoscopy was performed. A 10-mm-sized perforation and surrounding redness were observed in the anterior and inferior cartilages of the nasal septum (Figure 2 A). MSSA was detected in wound cultures, and the antibiotic sensitivity and biochemical properties (Beckman Coulter Microscan) were consistent with those of the blood cultures. Nasal septal perforation was considered the entry portal for MSSA bacteremia. Head CT scan showed nasal septal perforation and deviation (Figure 2B). The nasal septal perforation was thought to have occurred because of nasal mucosal damage caused by nasal dryness and curettage of the dried nasal mucus.

After the diagnosis, the antimicrobial agent was replaced with cefazolin (CEZ). Oxygen therapy was adjusted by 1 L to maintain a minimum SpO2 90%, and oxygen was humidified even below 4 L/min. The blood culture was negative on the 5th day. Still, echocardiography on the 20th day showed enlargement of the vegetation to 20 × 20 × 12 mm. Exacerbation due to toxin production by MSSA was considered. Consequently, clindamycin (CLDM) was administered instead of gentamicin, due to the organism's susceptibility to clindamycin and resistance to gentamicin. Following the administration of clindamycin, the vegetation size reduced. Betamethasone valerate and petrolatum ointment were used at the site of perforation, with improvements in redness and crust formation. After confirming a negative blood culture, CEZ was administered for 6 weeks, and IE treatment was completed. Subsequently, the patient developed aspiration pneumonia, and TAZ/PIPC was resumed; however, the patient died on the 53rd day.

DISCUSSION

Because dry oxygen is used in HOT, the increased flow causes dryness of the nasal cavity. Therefore, adding humidification to inhaled oxygen is considered useful for symptomatic relief. The American Association for Respiratory Care guidelines

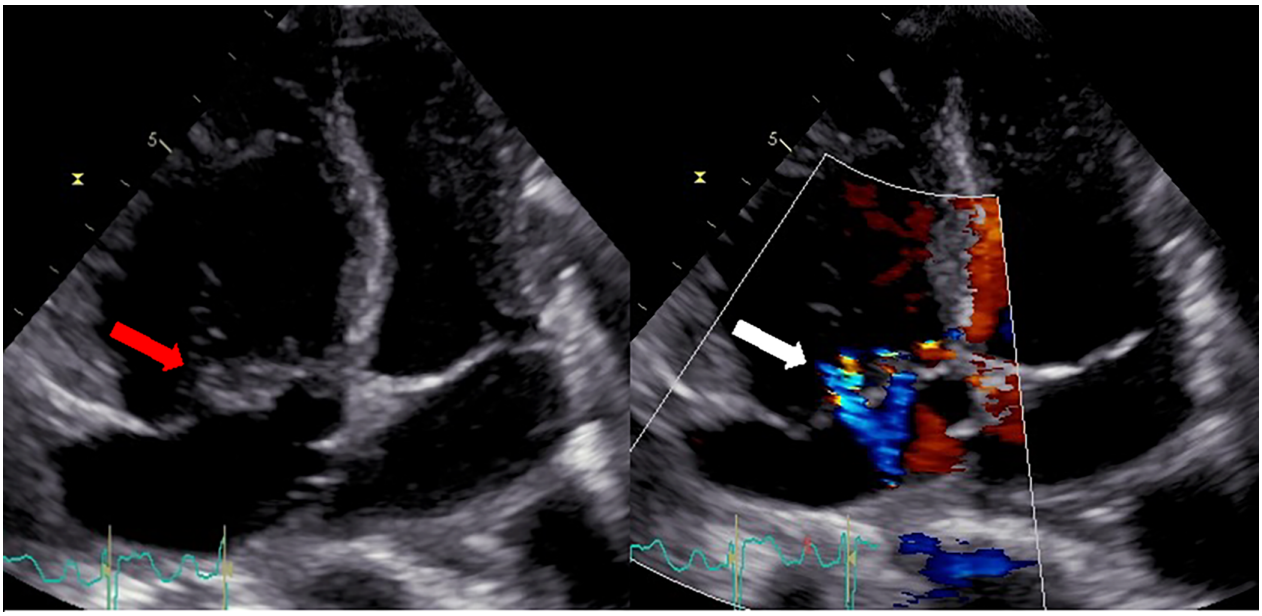

FIGURE 1 Transthoracic echocardiography shows tricuspid valve vegetation (red arrow) and regurgitation (white arrow). The tricuspid regurgitation pressure gradient increased to 65 mmHg.

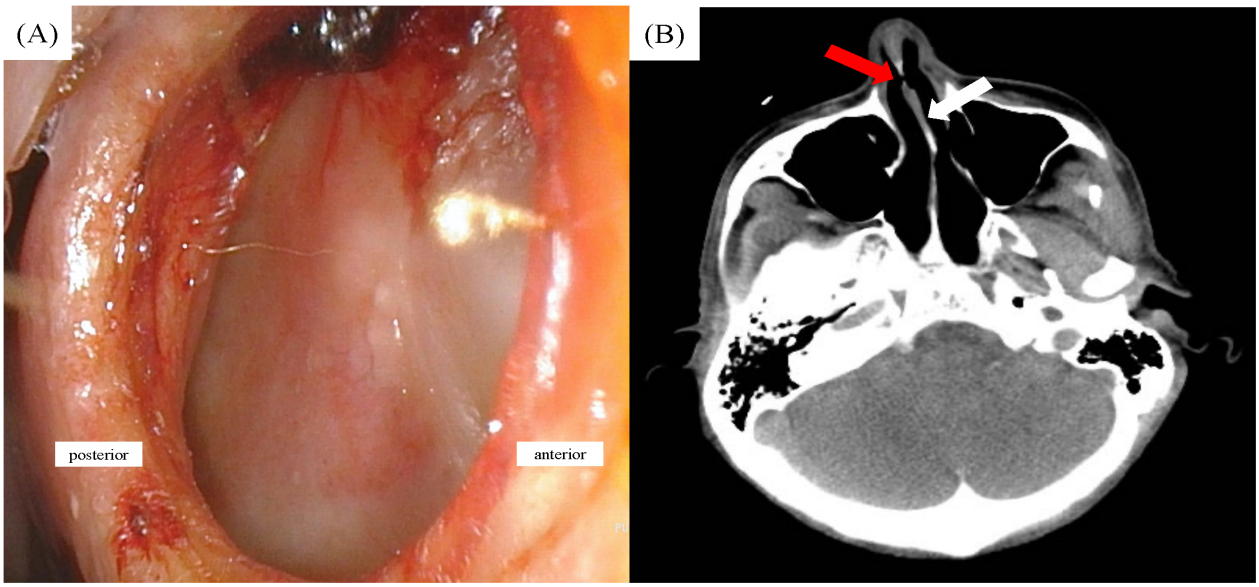

FIGURE 2 (A) Nasal endoscopy, taken from right nasal cavity, shows a 10-mm-sized perforation and surrounding redness in the anterior and inferior cartilage of the nasal septum. The anterior side indicates the external nostril and the posterior side indicates the nasopharyngeal side. (B) Axial plane head computed tomography image shows nasal septal perforation (red arrow) and nasal septal deviation (white arrow).

state that humidification is not necessary below 4 L/min.[1] However, there are reports that even a low flow rate of 3 L/min can cause mucosal damage within a short period. Humidification, even below 4 L/min, may be considered flexibly based on patient symptoms. In this case, perforation could have been avoided if the nasal environment had been cared for and treated beforehand. During HOT, listening to patients' complaints of nasal dryness and increased levels of dry nasal mucus was considered very important. Nasal dryness should be treated with HOT humidification or moisturization, and if symptoms are severe, nasal endoscopy may be considered to confirm nasal mucosal damage.

Possible causes of nasal septal perforation include (1) surgical injury, (2) chemical agents, (3) collagen and autoimmune diseases (mainly granulomatosis with polyangiitis), (4) hematologic diseases, (5) infectious diseases, and (6) trauma.[2] In this case, trauma due to nasal curettage was considered.

Although no previous nasal culture had been submitted, the patient had previously complained of nasal dryness and increased levels of dry nasal mucus, suggesting that he had become a nasal carrier of MSSA during repeated curettage.

IE of the right heart is rarer and accounts for 5%–12% of all cases.[3] Factors contributing to right-sided IE include cardiac devices such as pacemakers and defibrillators, repeated intravascular drug administration, and other factors.[4] In the present case, nasal septal perforation and other factors were considered.

In this case, CEZ caused an increase in vegetation when administered alone, possibly owing to toxin production by MSSA. In an in vitro study, CLDM, a protein synthesis inhibitor, effectively inhibited toxin production in staphylococci.[5] Although there is no established clinical evidence for concomitant use of CLDM in the treatment of IE with MSSA, CLDM was used in combination with CEZ due to the need for treatment. After the combination treatment, a significant size reduction of the vegetation was confirmed. This case suggests that CLDM is a clinical treatment option for refractory staphylococcal infections.

Thus, we report a case of IE due to nasal septal perforation caused by nasal dryness and curettage of the dried nasal mucus. Our case emphasizes that the care of the nasal environment during HOT is important to avoid complications.

AUTHOR CONTRIBUTIONS

Kyota Shinfuku contributed to the conception of the study, interpretation of the data, and drafting of the manuscript. Naoki Takasaka, Ohashi Ryutaro, Taiki Fukuda, Makiko Takatsuka, Ryo Sato, Mitsuyoshi Mita, Tsukasa Hasegawa, Masami Yamada, Yumie Yamanaka, Yusuke Hosaka, Kai Ryu, Tokio Hoshina, Hiroshi Takeda, Takeo Ishikawa, and Jun Araya contributed to the interpretation of data and revision of the manuscript. All the authors critically reviewed and approved the final version of this manuscript.

CONFLICT OF INTEREST STATEMENT

None declared.

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available from the corresponding author. The data are not publicly available due to privacy concerns.

ETHICS STATEMENT

The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying images.

ORCID

Kyota Shinfuku https://orcid.org/0000-0002-3463-9916

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