Assess **14**: 83-114, 2010.

17. Capelozzi VL, Parra ER, Ximenes M, Bammann RH, Barbas CS, Duarte MI. Pathological and ultrastructural analysis of surgical lung biopsies in patients with swine-origin influenza type A/H1N1 and acute respiratory failure. Clinics (Sao Paulo) **65**: 1229-1237, 2010.

18. Abdo A, Alfonso C, Diaz G, Wilford M, Rocha M, Verdecia N. Fatal 2009 pandemic influenza A (H1N1) in a bone marrow transplant recipient. J Infect Dev Ctries **5**: 132-137, 2011.

19. Ru YX, Li YC, Zhao Y, et al. Multiple organ invasion by viruses: pathological characteristics in three fatal cases of the 2009 pandemic influenza A/H1N1. Ultrastruct Pathol **35**: 155-161, 2011.

20. Basu A, Shelke V, Chadha M, et al. Direct imaging of pH1N1 2009 influenza virus replication in alveolar pneumocytes in fatal cases by transmission electron microscopy. J Electron Microsc (Tokyo) **60**: 89-93, 2011.

21. Nakajima N, Sato Y, Katano H, et al. Histopathological and immunohistochemical findings of 20 autopsy cases with 2009 H1N1 virus infection. Mod Pathol **25**: 1-13, 2012.

22. Nin N, Sánchez-Rodríguez C, Ver LS, et al. Lung histopathological findings in fatal pandemic influenza A (H1N1). Med Intensiva **36**: 24-31, 2012.

23. Shelke VN, Kolhapure RM, Kadam D, et al. Pathologic study of pandemic influenza A (H1N1) 2009 cases from India. Pathol Int **62**: 36-42, 2012.

24. Bal A, Suri V, Mishra B, et al. Pathology and virology findings in cases of fatal influenza A H1N1 virus infection in 2009-2010. Histopathology **60**: 326-335, 2012.

25. Fujita J, Higa H, Azuma M, et al. Immunohistochemical findings of an autopsied lung specimen from a patient with pandemic influenza (A/H1N1pdm) virus infection. Intern Med **51**: 507-512, 2012.

26. Boulagnon C, Leveque N, Renois F, Andreoletti L, Fornes P. Influenza A/H1N1 (2009) infection as a cause of unexpected out-of-hospital death in the young. J Forensic Sci **57**: 1650-1665, 2012.

27. Skálová H, Povýšil C, Hofmanová J, et al. Histopathological autoptic findings in 8 patients with pandemic influenza A (H1N1) pneumonia. Cesk Patol **48**: 161-164, 2012.

28. Bhatnagar J, Jones T, Blau DM, et al. Localization of pandemic 2009 H1N1 influenza A virus RNA in lung and lymph nodes of fatal influenza cases by in situ hybridization: new insights on virus replication and pathogenesis. J Clin Virol **56**: 232-237, 2013.

29. Sun AP, Ohtsuki Y, Fujita J, Ishida T, Yoshinouchi T, Kohno N. Immunohistochemical characterisation of pulmonary hyaline membrane in various types of interstitial pneumonia. Pathology **35**: 120-124, 2003.

30. Kido H, Okumura Y, Takahashi E, et al. Role of host cellular proteases in the pathogenesis of influenza and influenza-induced multiple organ failure. Biochim Biophys Acta **1824**: 186-194, 2012.

Contents lists available at ScienceDirect

Respiratory Investigation

journal homepage: www.elsevier.com/locate/resinv

Case report

Implementation of bronchoalveolar lavage using a high-flow nasal cannula in five cases of acute respiratory failure

Kazuya Miyagi, Shusaku Haranaga, Futoshi Higa, Masao Tateyama, Jiro Fujita*

Department of Infectious, Respiratory, and Digestive Medicine, Control and Prevention of Infectious Diseases (First Department of Internal Medicine), Faculty of Medicine, University of the Ryukyus, 207 Uehara, Nishihara, Okinawa 903-0215, Japan

ARTICLE INFO

Article history:
Received 5 September 2013
Received in revised form
22 May 2014
Accepted 23 June 2014
Available online 25 July 2014

Keywords:
High-flow nasal cannula
Bronchoscopy
Bronchoalveolar lavage
Acute respiratory failure

ABSTRACT

In recent clinical practice, high-flow nasal cannula (HFNC) therapy has been used to improve oxygenation in adults with acute respiratory failure (ARF). However, bronchoscopy using HFNC in ARF has not yet been reported. Herein, we describe 5 cases of ARF where bronchoalveolar lavage (BAL) was employed successfully using an HFNC. We were able to discontinue or reduce the HFNC fraction of inspired oxygen (FiO_2) 30 min after completion of the bronchoscopy. Only 1 patient needed non-invasive positive pressure ventilation for 16 h after bronchoscopy. The HFNC may be a useful tool for ARF patients who require bronchoscopy.

1. Introduction

High-flow nasal cannula (HFNC) therapy is a technique that can deliver oxygen at a maximum flow of 60 L/min that is used in pediatrics [1]. Furthermore, HFNC can confirm the accurate mixing of high-flow air and oxygen by using an oxygen analyzer [2]. In conventional oxygen therapy, a limited amount of oxygen is supplied, because patients cannot tolerate insufficient heating and humidification of oxygen. In contrast, HFNC therapy is achieved through the integration of heated humidification. An HFNC is capable of the following: (1) providing accurate oxygen delivery (21–100%); (2) washing out anatomical dead space; and (3) providing low-level positive pressure [2,3]. Moreover,

Abbreviations: HFNC, high flow nasal cannula; ARF, acute respiratory failure; BAL, bronchoalveolar lavage; FiO_2, fraction of inspired oxygen; PaO_2, partial pressure of oxygen in arterial blood; SpO_2, oxygen saturation of peripheral artery; CT, computed tomography; WBC, white blood cell; ESR, erythrocyte sedimentation rate; GM-CSF, granulocyte-macrophage colony-stimulating factor; NT-proBNP, N-terminal pro-brain natriuretic peptide

*Corresponding author. Tel.: +81 98 895 1144; fax: +81 98 895 1414.
E-mail addresses: miyagi.kazuya@gmail.com (K. Miyagi), shar439@gmail.com (S. Haranaga), fhiga@med.u-ryukyu.ac.jp (F. Higa), tateyama@med.u-ryukyu.ac.jp (M. Tateyama), fujita@med.u-ryukyu.ac.jp (J. Fujita).

http://dx.doi.org/10.1016/j.resinv.2014.06.006
2212-5345/

HFNC use is generally well tolerated by patients. These characteristics induce improvements in both oxygenation and respiratory distress.

In recent clinical practice, many reports have described the utility of HFNC therapy in combating adult acute respiratory failure (ARF) [4,5]. As such, HFNC therapy may be a first-line choice for ARF among adults. Other ARF cases where an HFNC was used during bronchoscopy presented higher partial pressure of oxygen in arterial blood (PaO_2), PaO_2/FiO_2, and oxygen saturation of peripheral artery (SpO_2) values than conventional oxygen therapy at the end of the bronchoscopy [6]. However, both the efficacy and safety of HFNC use during bronchoscopy for ARF are not known. We report 5 cases of ARF patients where bronchoscopy was performed successfully using an HFNC.

2. Case report

2.1. *Case 1*

A man in his seventh decade with myelodysplastic syndrome was diagnosed with chronic active Epstein–Barr virus (EBV) infection with pulmonary disorder at his last admission. After 17 months, his dyspnea and cough had worsened and he was readmitted. He presented with tachypnea and hypoxia. During lung auscultation, we could hear fine crackles and rhonchi. The laboratory findings were as follows: white blood cell (WBC) count, $2.3 \times 10^4/\mu L$; hemoglobin, 12.8 g/dL; C-reactive protein (CRP), 1.6 mg/dL; erythrocyte sedimentation rate (ESR), 102 mm/h; lactate dehydrogenase, 567 IU/L; and KL-6, 8720 IU/mL. Arterial blood gas analysis showed the following (3 L nasal cannula with reservoir): PaO_2, 61 Torr; $PaCO_2$, 41 Torr; and HCO_3, 25.2 mEq/L.

A chest radiograph showed bilateral ground-glass opacity, and chest computed tomography (CT) revealed ground-glass opacities with interlobular thickness (Fig. 1A). The patient underwent bronchoscopy and bronchoalveolar lavage (BAL) as diagnostic tools for respiratory failure. He required oxygen therapy to maintain his SpO_2; therefore, he received oxygen via HFNC (50 L/min, FiO_2=50%) during bronchoscopy. The patient's SpO_2 and heart rate were stable during the procedure (Table 1). Oxygenation did not worsen 30 min after bronchoscopy, and he was able to discontinue the HFNC. His BAL fluid was light-colored and milky in appearance. He was diagnosed with pulmonary alveolar proteinosis through BAL fluid analysis with an electron microscope and by the presence of anti-granulocyte-macrophage colony-stimulating factor (GM-CSF) autoantibodies in his serum.

2.2. *Case 2*

A man in his seventh decade who was being monitored for hypertrophic cardiomyopathy by the circulatory department at our hospital visited another hospital with a high fever. He was diagnosed with bacterial pneumonia and admitted to the hospital. After receiving antimicrobial treatment, his fever subsided. He was discharged from the hospital, but continued to suffer from dyspnea. After 1 week, he again became febrile and his dyspnea worsened. He visited our emergency room

Table 1 – Clinical status of 5 cases who received broncho-alveolar lavage using oxygenation with high-flow nasal cannula.

Case	Age	Sex	P/F before BF	Setting of HFNC				During BF		SpO_2 after BF (%)	$PaCO_2$ after BF (Torr)	Duration of HFNC	Diagnosis
				Before BF		30 min after BF							
				FiO_2 (%)	Flow (L/min)	FiO_2 (%)	Flow (L/min)	SpO_2 (%)	HR (/min)				
1	78	Male	150	50	50	Discontinued		93–98	79–97	90	42.6	30 min	Pulmonary alveolar proteinosis
2	69	Male	144	80	40	80	45	91–97	92–112	91	32.7	16 h[a]	Eosinophilic pneumonia
3	79	Female	181	60	35	40	35	98–100	76–103	92	nd	2 days	Diffuse alveolar hemorrhage
4	57	Male	221	95	30	55	30	99–100	90–111	94	nd	3 days	Drug-induced lung injury
5	63	Male	146	50	50	50	50	97–100	95–104	98	36	6 days	Drug-induced lung injury

P/F: PaO_2/FiO_2; BF: bronchoscopy; HFNC: high-flow nasal cannula; FiO_2: fraction of inspired oxygen; SpO_2: oxygen saturation of peripheral artery; HR: heart rate; $PaCO_2$: partial pressure of arterial carbon dioxide; nd: not determined.

[a] Followed by non-invasive positive pressure ventilation.

and was admitted after presenting with tachypnea and hypoxia. During lung auscultation, we could hear fine crackles in the lower regions of both lungs. The laboratory findings were as follows: WBC count, $9.2 \times 10^3/\mu L$ (neutrophils, 84.8%; lymphocytes, 6.0%; monocytes, 6.8%; eosinophils, 2.2%); hemoglobin, 10.4 g/dL; CRP, 13.3 mg/dL; lactate dehydrogenase, 243 IU/L; and N-terminal pro-brain natriuretic peptide (NT-proBNP), 566.8 pg/mL. An arterial blood gas analysis showed the following (40 L/min, FiO_2=80%): PaO_2, 115 Torr; PCO_2, 29 Torr; and HCO_3, 23.1 mEq/L. A chest radiograph showed peripherally predominant bilateral ground-glass opacity (the photographic negative of pulmonary edema), and chest CT revealed peripherally predominant ground-glass opacity and consolidation (Fig. 1B). At first, the patient was suspected of bacterial pneumonia and acute heart failure. He was therefore hospitalized in the cardiology department for administration of antibiotics and diuretics. However, after his dyspnea worsened, our department was consulted. To make the diagnosis, bronchoscopy and BAL were performed.

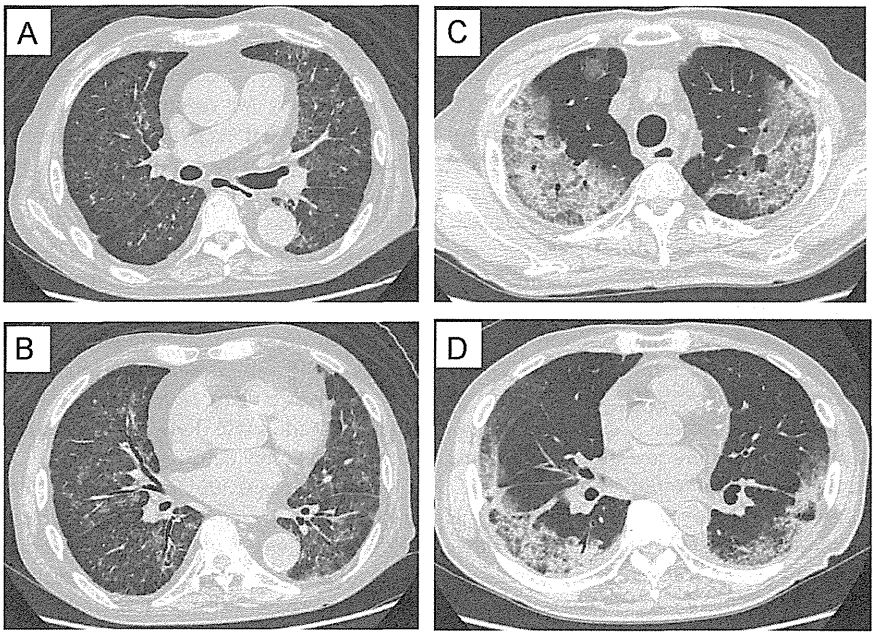


Fig. 1 – Chest CT imaging. Case 1: A set of chest CT images showing bilateral ground-glass opacities with interlobular thickness. (A) Subcarina level, (B) lower lobe level. Case 2: A set of chest CT images showing peripheral predominant ground-grass opacity and consolidation. (C) Upper lobe level, (D) lower lobe level. CT: computed tomography.

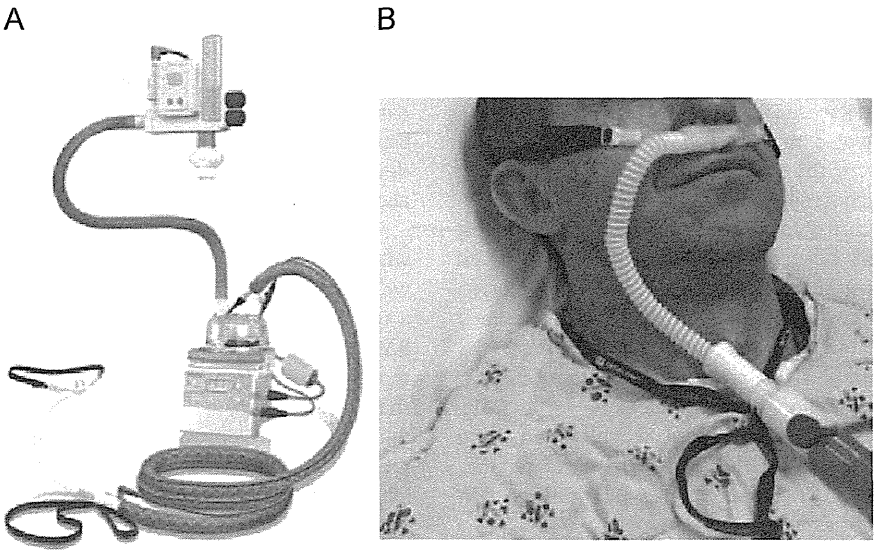


Fig. 2 – High flow nasal cannula system (from Ref. [2]). (A) Optiflow™ system (Fisher & Paykel, Auckland, New Zealand). (B) OptiFlow™ adult cannula on an adult patient. Additional sedative agent and insertion of the bronchoscope via the mouth was used when the patient tolerated the device.

The patient's oxygenation was poor; he therefore received oxygen via HFNC (40 L/min, FiO_2=80%) during bronchoscopy. His SpO_2 and heart rate were stable during the procedure (Table 1). Sixteen hours after BAL, he did not display thoraco-abdominal asynchrony, and his heart rate and respiratory rate had not increased; however, his PaO_2/FiO_2 ratio worsened from 144 to 80.3. To control his respiratory failure, we started continuous positive airway pressure (CPAP) (10 cm H_2O; FiO_2=60%). On the basis of BAL findings, a diagnosis of eosinophilic pneumonia was made, and corticosteroids were started. His respiratory failure improved with these treatments, and CPAP was discontinued. The duration of CPAP was 5 days.

Including these 2 cases, the results of 5 cases of ARF in which bronchoscopy and BAL were performed with HFNC are shown in Table 1. The 5 patients (1 woman and 4 men) ranged in age from 57 yr to 79 yr and had PaO_2/FiO_2 ratios ranging from 144 to 221. Each had a single comorbidity, including malignancy, interstitial pneumonia, cardiovascular disease, and a hematological disorder. Our HFNC was an OptiFlow™ RT050 device (Fisher & Paykel, Auckland, New Zealand) (Fig. 2). The main criterion for using an HFNC in bronchoscopy was respiratory failure that required oxygen therapy. When we performed the procedure using an HFNC, we inserted the bronchoscope by mouth. The HFNC settings were adjusted according to the patient's condition (flow, 30–50 L/min; FiO_2, 50%–95%). After the procedure, FiO_2 was gradually decreased until it returned to under 40%. We were able to discontinue the HFNC therapy in Case 1; in Cases 3 and 4, we were able to reduce the HFNC FiO_2 30 min after the completion of bronchoscopy. Case 2 required non-invasive positive pressure ventilation (NIPPV) 16 h after completion of the bronchoscopy. HFNC use was well tolerated in these cases. In 3 cases, definite diagnoses were made from BAL fluid analyses. In another 2 cases, BAL fluid analyses were useful in determining the treatment strategy for respiratory failure.

3. Discussion

A bronchoscope can decrease PaO_2 by 10–20 mmHg [7], and BAL is associated with worse oxygenation than when it is not performed [8]. Therefore, the American Thoracic Society recommends avoiding bronchoscopy and BAL in patients with hypoxemia that cannot be corrected to at least a PaO_2 of 75 mmHg or to an arterial oxygen saturation of 90% with supplemental oxygen [9]. In a recent study, compared to high-flow oxygen use alone, NIPPV improved oxygenation during bronchoscopy in patients with respiratory failure [10,11]; hence, there is the possibility that we can perform bronchoscopy without intubation and invasive ventilation, even in cases of severe hypoxia. However, NIPPV may not be indicated when patients are intolerant to NIPPV or have large quantities of respiratory secretions. Furthermore, NIPPV may cause gastric distention and increase the risk of aspiration [10].

In this report, we describe 5 cases of ARF where BAL was employed successfully using an HFNC. There were no interruptions in the bronchoscopy procedures because of hypoxia. Furthermore, the patients' oxygenation was well maintained for 30 min after bronchoscopy. In a previous report, the use of a CPAP of 7.5 cm H_2O during bronchoscopy resulted in minimal alterations in gas exchange and avoided subsequent respiratory failure [12]. HFNC can generate CPAP to a degree [13], which may be one of the reasons that HFNC can maintain the oxygenation of patients during and after bronchoscopy. However, Case 4 required NIPPV 16 h after the completion of bronchoscopy. The cause of worsening respiratory failure was exacerbation of the underlying disease, not as a result of the procedure itself. The patient was diagnosed with eosinophilic pneumonia from BAL fluid analysis and was able to discontinue NIPPV and oxygen therapy with steroid treatment.

Appropriate indications and settings for HFNC during bronchoscopy for respiratory failure were not determined. Because HFNC therapy is well tolerated and can be started or discontinued easily, we may use this system more readily for any patient who needs oxygen therapy before the procedure. Regarding the flow settings for HFNC, a flow rate of under 60 L/min produced better oxygenation than a 40 L/min flow rate during bronchoscopy in a healthy volunteer, and it is suggested that a 60 L/min flow rate is suitable for use with HFNC during bronchoscopy with respiratory dysfunction [6]. However, in our present analysis, we were able to perform the procedure using HFNC with flow settings of 30–50 L/min. Further studies will therefore be useful to determine the most appropriate flow settings.

In these cases, we did not assess the tolerance of patients to the device by using a visual analog scale or similar method. However, there was no interruption in any bronchoscopy procedure due to HFNC intolerance. According to a tolerance scale used for the assessment of comfort in a previous report [14], HFNC was well tolerated.

One of the most important points with such a technique is not to delay using another device if necessary, such as NIPPV or intubation. Even in the case of NIPPV, delayed intubation is associated with increased mortality in patients with respiratory failure [15]. Therefore, the intubation of patients should not be delayed if their respiratory failure progresses. Sztrymf et al. identified an early lack of decrease in respiratory rate and the persistence of thoraco-abdominal asynchrony as early and simple indicators of HFNC failure [16]. In such cases, it is necessary to stop the procedure or use another device if findings of hypoxia, tachycardia, or thoraco-abdominal asynchrony are present.

We reported 5 cases of ARF that were able to undergo BAL with the use of HFNC. Because HFNC therapy is effective and well-tolerated by patients with hypoxia and dyspnea, it may be a useful tool for ARF patients who require bronchoscopy and BAL. Furthermore, we can easily perform suction from the mouth during bronchoscopy. However, we cannot conclude that HFNC therapy is superior to conventional oxygen therapy during bronchoscopy, because our article only comprises case reports. Further investigation is necessary to confirm the efficacy and safety of HFNC use during bronchoscopy and BAL.

Conflict of interest

The authors have no conflicts of interest.

REFERENCES

[1] Shoemaker MT, Pierce MR, Yoder BA, et al. High flow nasal cannula versus nasal CPAP for neonatal respiratory disease: a retrospective study. J Perinatol 2007;27:85–91.

[2] Ward JJ. High-flow oxygen administration by nasal cannula for adult and perinatal patients. Respir Care 2013;58:98–122.

[3] Kernick J, Magarey J. What is the evidence for the use of high flow nasal cannula oxygen in adult patients admitted to critical care units? A systematic review. Aust Crit Care 2010;23:53–70.

[4] Sztrymf B, Messika J, Bertrand F, et al. Beneficial effects of humidified high flow nasal oxygen in critical care patients: a prospective pilot study. Intensive Care Med 2011;37:1780–6.

[5] Rello J, Pérez M, Roca O, et al. High-flow nasal therapy in adults with severe acute respiratory infection: a cohort study in patients with 2009 influenzaA/H1N1v. J Crit Care 2012;27:434–9.

[6] Lucangelo U, Vassallo FG, Marras E, et al. High-flow nasal interface improves oxygenation in patients undergoing bronchoscopy. Crit Care Res Pract 2012;10:1–6.

[7] Payne Jr CB, Goyal PC, Gupta SC. Effects of transoral and transnasal fiberoptic bronchoscopy on oxygenation and cardiac rhythm. Endoscopy 1986;18:1–3.

[8] Katz AS, Michelson EL, Stawicki J, et al. Cardiac arrhythmias. Frequency during fiberoptic bronchoscopy and correlation with hypoxemia. Arch Intern Med 1981;141:603–6.

[9] Goldstein RA, Rohatgi PK, Bergofsky EH, et al. Clinical role of bronchoalveolar lavage in adults with pulmonary disease. Am Rev Respir Dis 1990;142:481–6.

[10] Murgu SD, Pecson J, Colt HG. Bronchoscopy during noninvasive ventilation: indications and technique. Respir Care 2010;55:595–600.

[11] Antonelli M, Conti G, Rocco M, et al. Noninvasive positive-pressure ventilation versus conventional oxygen supplementation in hypoxemic patients undergoing diagnostic bronchoscopy. Chest 2002;121:1149–54.

[12] Maitre B, Jaber S, Maggiore SM, et al. Continuous positive airway pressure during fiberoptic bronchoscopy in hypoxemic patients. A randomized double-blind study using a new device. Am J Respir Crit Care Med 2000;162:1063–7.

[13] Groves N, Tobin A. High flow nasal oxygen generates positive airway pressure in adult volunteers. Aust Crit Care 2007;20:126–31.

[14] Tiruvoipati R, Lewis D, Haji K, et al. High-flow nasal oxygen vs high-flow face mask: a randomized crossover trial in extubated patients. J Crit Care 2010;25:463–8.

[15] Demoule A, Girou E, Richard JC, et al. Increased use of noninvasive ventilation in French intensive care units. Intensive Care Med 2006;32:1747–55.

[16] Sztrymf B, Messika J, Bertrand F, et al. Beneficial effects of humidified high flow nasal oxygen in critical care patients: a prospective pilot study. Intensive Care Med 2011;37:1780–6.

INTERNAL MEDICINE

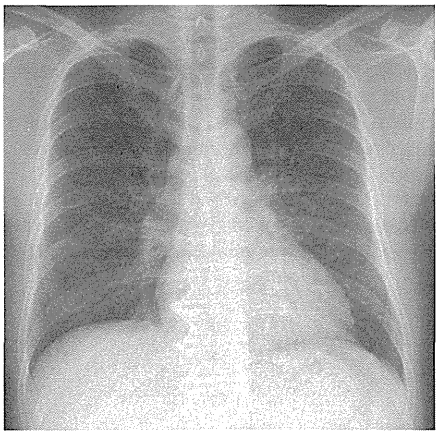
□ PICTURES IN CLINICAL MEDICINE □

Rhinovirus Pneumonia in a Patient Infected with HIV

Shusaku Haranaga, Takeshi Kinjo, Masao Tateyama and Jiro Fujita

Key words: human rhinovirus, pneumonia, human immunodeficiency virus

(Intern Med 53: 2027-2028, 2014)
(DOI: 10.2169/internalmedicine.53.2827)

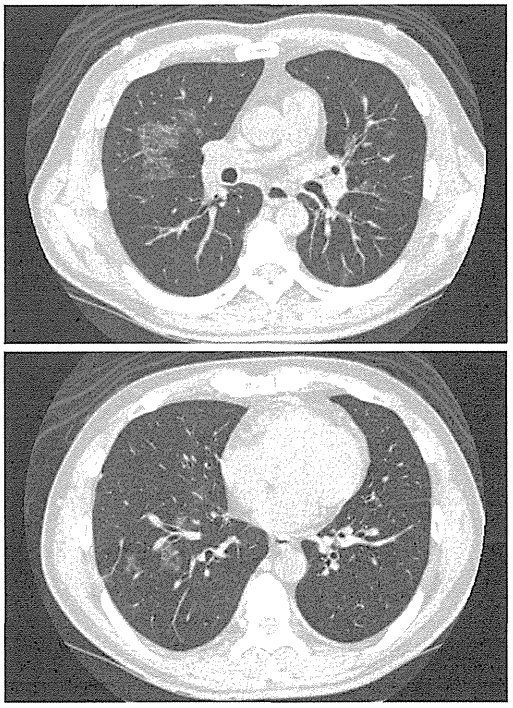


Picture 1.

A 51-year-old man on antiretroviral therapy for human immunodeficiency virus (HIV) infection (CD4 196/μL, viral load below 20 copies/mL) visited the emergency room complaining of fever and dry cough. There was no abnormal infiltration observed on his chest X-ray (Picture 1); however, chest computed tomography (CT) revealed multiple areas of patchy ground-glass opacity in both lungs (Picture 2).

He had no sputum, and a rapid antigen test for influenza and urinary antigen tests for *Streptococcus pneumoniae* and *Legionella pneumophila* were negative. Bronchoalveolar lavage (BAL) was performed, and his sample was cultured for bacteria and fungi, and used in a multiplex polymerase chain reaction (PCR) that can detect 15 respiratory viruses and six bacteria strains (Seeplex kit, Seegene, Seoul, Korea). The BAL sample was also tested with Grocott's stain for *Pneumocystis jirovecii*. Only rhinovirus was detected, and he was diagnosed as pure rhinovirus pneumonia.

Rhinovirus is a pathogen that can cause upper respiratory tract infections. However, Puro et al. reported that rhinovirus was detected in 60% of patients with pneumonia (1). Jacobs et al. described CT findings in patients with rhinovirus infections and found that the most common finding was patchy ground-glass opacities, as observed in the present patient (2).



Picture 2.

The authors state that they have no Conflict of Interest (COI).

References

1. Puro V, Minosse C, Cappiello G, Lauria FN, Capobianchi MR. Rhinovirus and lower respiratory tract infection in adults. Clin Infect Dis **40**: 1068-1069, 2005.
2. Jacobs SE, Soave R, Shore TB, et al. Human rhinovirus infections

Department of Infectious, Respiratory, and Digestive Medicine, Control and Prevention of Infectious Diseases (The First Department of Internal Medicine), Faculty of Medicine, University of the Ryukyus, Japan
Received for publication March 7, 2014; Accepted for publication April 2, 2014
Correspondence to Dr. Shusaku Haranaga, f014936@med.u-ryukyu.ac.jp

of the lower respiratory tract in hematopoietic stem cell transplant recipients. Transpl Infect Dis **15**: 474-486, 2013.

INTERNAL MEDICINE

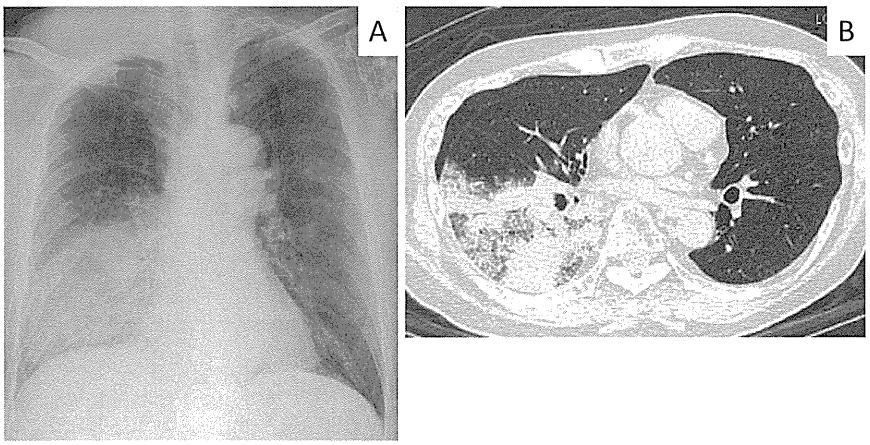
□ PICTURES IN CLINICAL MEDICINE □

Orange Sputum in a Patient with *Legionella pneumophila* Pneumonia

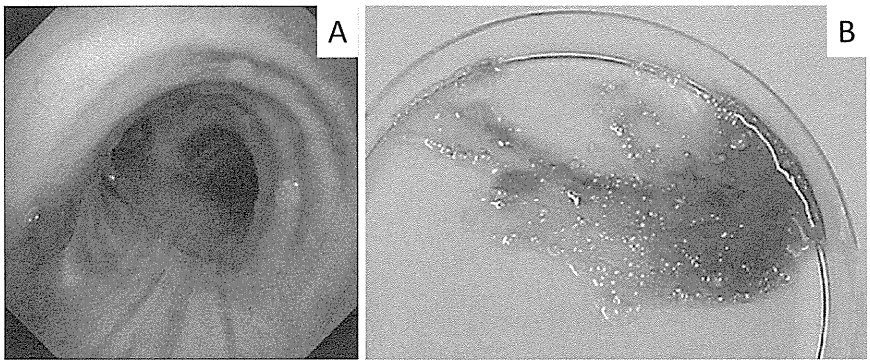
Takeshi Kinjo, Daijiro Nabeya, Futoshi Higa and Jiro Fujita

Key words: *Legionella pneumophila*, orange sputum, pneumonia

(Intern Med 53: 2029-2030, 2014)
(DOI: 10.2169/internalmedicine.53.2897)



Picture 1.



Picture 2.

A 56-year-old man with multiple myeloma and neurilemmoma of the neck was transported to our hospital due to cough, fever, diarrhea, and anorexia. A chest x-ray showed massive consolidation of the right lower lobe, and chest computed tomography revealed consolidation with an air bronchogram accompanied by ground-glass infiltrations (Picture 1A, B). He was diagnosed with *Legionella pneumophlila* pneumonia based on his positive urinary antigen

Department of Infectious, Respiratory, and Digestive Medicine, Control and Prevention of Infectious Diseases (The First Department of Internal Medicine), Faculty of Medicine, University of the Ryukyus, Japan
Received for publication March 16, 2014; Accepted for publication April 2, 2014
Correspondence to Dr. Takeshi Kinjo, kumanomis6@yahoo.co.jp

test and gradually recovered following treatment with intravenous levofloxacin. *L. pneumophila* serogroup 1 was isolated from his sputum, and the color of which had been orange for several weeks after admission. Orange sputum was also observed in the patient's trachea (Picture 2A, B).

A previous report described orange sputum in patients with *L. pneumophila* pneumonia (1). *L. pneumophila* changes the color of tyrosine-containing medium to brown-orange, and epithelial lining fluids contain tyrosine; these facts suggest that *L. pneumophila* causes orange sputum by affecting tyrosine in the body (1, 2).

The authors state that they have no Conflict of Interest (COI).

References

1. Fujita J, Touyama M, Chibana K, et al. Mechanism of formation of the orange-colored sputum in pneumonia caused by *Legionella pneumophila*. Intern Med **46**: 1931-1934, 2007.
2. Vickers RM, Yu VL. Clinical laboratory differentiation of *Legionellaceae* family members with pigment production and fluorescence on media supplemented with aromatic substrates. J Clin Microbiol **19**: 583-587, 1984.