Review Article

Human *Erysipelothrix rhusiopathiae* Infection: Unsolved Issues and Possible Solutions

Prasit Upapan MD*

** Division of Infectious Diseases, Department of Medicine, Faculty of Medicine, Srinakharinwirot University, Nakhon Nayok, Thailand*

Erysipelothrix rhusiopathiae infection in humans may not be as rare as previously thought. In most cases, the disease is acquired from animals through work-related exposure. Human infection has been reported since the early 1900's up to the present. Unsolved issues associated with this organism include inadequate disease control in animals, difficulty in identification and isolation of the bacteria, diagnostic delay due to unawareness of this uncommon disease or unfamiliarity with the increasingly diverse clinical manifestations, and inappropriate antibiotic use due to misdiagnosis, as well as drug resistance. In this review, we attempt to address the unsolved issues related to human Erysipelothrix infection and suggest possible solutions.

Keywords: *Erysipelothrix rhusiopathiae, Erysipeloid, Bacteremia, Occupational disease*

J Med Assoc Thai 2015; 98 (Suppl. 9): S170-S176
Full text. e-Journal: *http://www.jmatonline.com*

Erysipelothrix rhusiopathiae belongs to the genus *Erysipelothrix*. There are two other species: *E. tonsillarum* and *E. inopinata*[1]. While *E. tonsillarum* infects only canines[2], *E. rhusiopathiae* primarily causes disease in humans. Koch first isolated *E. rhusiopathiae* as a gram-positive rod in 1878[3]. However, it was Rosenbach who determined the first human infection in 1909 by isolation of this organism from skin lesions[4]. Bacterial colonies are relatively small and may have a slow growth rate[5]. Thus, even with excellent laboratory facilities and expertise, low yields and delays in identification and isolation may occur.

Erysipelothrix rhusiopathiae has been recognized as a commensal or an important pathogen in a wide variety of vertebrate and invertebrate species including chickens, ducks, swine, sheep, cattle, horses, dogs, mice, wild rodents, and fresh- and saltwater fish[6], but it is also known as a serious pathogen in humans[7]. Human acquisition of the disease can occur from direct contact with infected animal secretions or products. People with the greatest risk for infection include butchers, meat cutters, fisherman, and housewives[8,9]. In Thailand, a large portion of the rural population work in agriculture, growing rice in the fields, and raising livestock. Because it is difficult to control *Erysipelothrix* infection among animals, subsequent human infection may occur, resulting in loss of revenue from animal sales and worker morbidity[10].

Traditionally, clinical syndromes of *E. rhusiopathiae* are comprised of three main forms that include a localized cutaneous form (erysipeloid) (this term is used to avoid confusion with the human streptococcal disease erysipelas), a diffuse cutaneous form, and a rare systemic form with septicemia and endocarditis[11]. After the year 2000, increasingly diverse clinical presentations have been reported, especially the systemic form of infection. In several reported cases from Thailand, *Erysipelothrix* infection occurred mainly in immunocompromised patients[12,13]. Unawareness of this uncommon disease or unfamiliarity with the increasingly atypical clinical manifestations may cause the diagnosis to be wrong or delayed, leading to inappropriate antibiotic use and increased morbidity and mortality.

Thus, there are several unsolved problems associated with human *Erysipelothrix* infection. These include inadequate disease control in animals, difficulty in identification and isolation of the bacteria, delayed diagnosis, improper antibiotic use due to misdiagnosis, as well as drug resistance. In this article, we attempt to address the unsolved issues related to human

Correspondence to:
Upapan P, Division of Infectious Diseases, Department of Medicine, Faculty of Medicine HRH Princess Maha Chakri Sirindhorn Medical Center, Srinakharinwirot University, Nakhon Nayok 26120, Thailand.
Phone: +66-2-6641000, Fax: +66-37-395085
E-mail: yokinnon@yahoo.co.th

Erysipelothrix infection and suggest possible solutions.

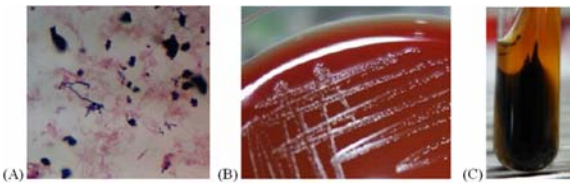
Microbiology

All species in the genus *Erysipelothrix* are facultative anaerobic, non-spore-forming, straight or slightly curved, thin gram-positive bacilli[14] (Fig. 1A). Growth develops at a temperature around 5 to 44°C (optimal temperature 30-37°C), and at a pH between 7.2 and 7.6 (range 6.8-8.2). For some strains, the growth is promoted by 5 to 10% carbon dioxide[3]. The organism may be shown as alpha-hemolytic, but has never been identified as beta-hemolytic on blood agar[14] (Fig. 1B). The colonial appearance of this organism can be classified into two main forms. After incubating for 24 hours at 37°C, colonies turn small, circular, and transparent, with a smooth surface and well-circumscribed, so-called smooth or S-forms. Larger and flatter colonies with a matte surface surrounded with fimbriated edge are rough colonies or R-forms. Both forms are usually light blue or sometimes green. The combination of S and R forms, the intermediate form (RS form), is the most common colonial conformation[3,11].

Many factors may affect the morphology formation of colonies. Such variations depend on the medium, blood pH, and temperature of incubation. Growth in acidic pH and temperature of 37°C favor R-forms[15], whereas incubation in alkaline pH (7.6 to 8.2) and temperature of 33°C favor S-forms[11]. It has been suggested that S-form colonies are found in acute disease, whereas R-forms are dominant in more chronic forms of the disease[16,17].

E. rhusiopathiae is catalase, oxidase, indole, motility, Voges-Proskauer and methyl red negative[18]. Hydrogen sulfide (H_2S) is synthesized by most strains. Some media may produce more H_2S than others. Triple sugar iron agar is preferred due to robust H_2S production, resulting in a blackened butt (Fig. 1C). The "test tube brush" growth pattern found in gelatin stab cultures is also very characteristic[7].

Biochemical testing can be utilized to differentiate *E. rhusiopathiae* from other organisms. One should be aware that *Erysipelothrix* might be misidentified as *Lactobacillus* or *Enterococcus* species[19,20]. However, gram staining can be used to differentiate *Enterococcus,* which appears as gram-positive cocci in pairs, compared to *Erysipelothrix and Lactobacillus,* which are both gram-positive rods. The H_2S test can be used to further distinguish *E. rhusiopathiae* from *Listeria monocytogenes*, *Lactobacillus*, and *Corynebacteria*. *E. rhusiopathiae* produces the H_2S gas, while the other three do not[7,19]. Moreover, *E. rhusiopathiae* can be differentiated from *E. tonsillarum* by the sugar fermentation testing, in which the latter, but not the former, ferments sucrose[21].



Courtesy of the Bacteriology Laboratory Unit, Department of Microbiology, Mahidol University, for providing microscopic pictures and biochemistry tests of *E. rhusiopathiae*

Fig. 1 Gram stain from blood culture broth demonstrated slightly curved, thin gram-positive bacilli (A). Blood agar showed small alpha-hemolytic colonies (B). Hydrogen sulfide on triple sugar iron media (C)

There have been efforts to develop faster and more accurate detection methods. Polymerase chain reaction (PCR) has been used for species-specific screening of this bacteria in animal tissue within five hours[22,23]. Limitations for using PCR include specimen contamination, high cost, and limited availability.

Difficulty in identification and isolation of *Erysipelothrix* may be due to slow-growing, tiny bacterial colonies, and heavily contaminated specimens. Although a variety of selective media may improve qualitative isolation of this organism, it may not be possible to inhibit all contaminants[5]. Thus, when there is clinical suspicion of this infection, notifying the laboratory directly may lead to more rapid and efficient identification. It may sometimes be necessary to send specimens to better-equipped laboratories with more advanced methods of detection.

Pathogenesis and virulence factors

There is limited knowledge about the pathogenesis and virulence factors of human *Erysipelothrix* infection, as most of the data are from in vitro and animal studies. According to current knowledge, the capsule of *E. rhusiopathiae* is an important virulence factor, helping it to escape phagocytosis. In the case it is phagocytized, it has the ability to survive and replicate intracellularly[24]. Other virulence factors that contribute to the pathogenicity of this organism include the production of the enzymes hyaluronidase and neuraminidase. Neuraminidase plays

a key role in bacterial attachment and invasion into the host cell. The role of hyaluronidase has not yet been elucidated[25].

Clinical manifestation and diagnosis

Diseases caused by *E. rhusiopathiae* share similar clinical manifestations in animals and humans, but there are some differences. In animals, erysipelas and polyarthritis are typical forms of infection, whereas erysipeloid of Rosenbach, a local skin infection or cellulitis, is a common presentation of diseases seen in humans. The second and third clinical categories of human diseases are the generalized cutaneous and septicemic form, respectively. The septicemic form is often associated with endocarditis[11].

Localized cutaneous form or erysipeloid of Rosenbach

The most common clinical manifestation of human *E. rhusiopathiae* infection is called erysipeloid. The erysipeloid skin lesion is usually seen as a sharply demarcated violaceous plaque[26]. This severely painful localized cellulitis is frequently found on fingers and hands with pre-existing wounds. The occupation of a butcher increases risk for exposure, and there is usually a one to two weeks (5-7 days or maximum 2 weeks)[26] prior history of direct contact with infected fish, pork, or other animal meat.

A common differential diagnosis of such a painful skin infection is staphylococcal or streptococcal infection. Useful clinical clues include the violet hue, the exquisite pain that seems out-of-proportion to the lesion, and the lack of edema and suppuration, all of which favor *E. rhusiopathiae* infection. Systemic manifestations are not common, only about one in 10 patients have a fever and joint pains, while one-third of patients may have associated lymphangitis and lymphadenopathy[27].

Diagnosis of erysipeloid is confirmed by aspiration or full-thickness skin biopsy at the edge of the lesion[3]. The specimen must be placed immediately in an infusion broth of 1% glucose[28], and may be handled at room temperature or refrigerated before being sent to the laboratory[3].

This localized skin infection may resolve even without treatment within three to four weeks[3]. However, if left untreated, relapses may occur.

Diffuse cutaneous form

The rare diffuse cutaneous form is characterized by either a proximal extension from the initial site of local infection or by distant skin lesions[11,29]. Bullous lesions may also be found. Features that may differentiate this diffuse form from the local form of *E. rhusiopathiae* infection include more pronounced, systemic symptoms and of a longer duration of infection. Furthermore, the rate of recurrence is higher[30]. Nonetheless, blood cultures are usually negative.

Disseminated form

Systemic infection with bacteremia with or without endocarditis

E. rhusiopathiae systemic infection is an uncommon, yet serious condition that usually presents subacutely. However, among the rare cases of *E. rhusiopathiae* septicemia, there is a high frequency of endocarditis (90% of case reports)[7]. Prior history of local skin infection or concomitant erysipeloid skin lesions were found in 36% of patients[7]. In most case reports, patients were immunocompetent, although about one-third had a history of alcohol abuse. Native cardiac valves were involved, except for one case that involved a Starr-Edwards prosthetic aortic valve[31].

Clinical findings such as fever, peripheral skin lesions of endocarditis, splenomegaly, hematuria, and mycotic aneurysm were similar between subjects with endocarditis caused by *E. rhusiopathiae* or other bacteria. However, in *E. rhusiopathiae* endocarditis, men were more frequently affected than women, death rates were higher (mortality rate of 38%), pre-existing cardiac diseases were more common, and the aortic valve was more predominantly involved[7,32].

Complications of *E. rhusiopathiae* endocarditis include congestive heart failure (80%)[11], myocardial abscess formation, and aortic valve perforation[33-36], with over one-third of patients requiring valve replacement[7]. Other reported complications of *E. rhusiopathiae* septicemia include diffuse glomerular nephritis and meningitis[37,38], as well as septic shock[39].

Erysipelothrix bacteremia without endocarditis was previously believed to be rare. However, in the past three decades there have been several case reports of this atypical occurrence, the most recent being a patient from Thailand in 2014[19,39-42]. Moreover, there seemed to be an emergence of *Erysipelothrix* bacteremia among immunocompromised patients, particularly those that were receiving steroids or cytotoxic drugs[39-41]. In suspected cases of *E. rhusiopathiae* septicemia or endocarditis, routine blood culture techniques are usually adequate for

diagnosis.

Other manifestations

In addition to the previously mentioned clinical forms, after the year 2000 other atypical manifestations of the disease involving various organs have been reported, such as osteomyelitis[43,44], septic arthritis[45], necrotizing fasciitis[46], peritoneal dialysis-related peritonitis[47], chronic meningitis[48], endophthalmitis[49], prosthetic knee joint infection[50], intra-abdominal infection(abscess)[51], pneumonia[52] and paravertebral abscesses[19,53].

Clinicians should be aware of both the typical forms and possible variations in clinical presentations of *Erysipelothrix* infection, as this may have important treatment-related implications. It is notable this gram-positive bacteria is resistant to vancomycin. Thus, if initial blood cultures yield a gram-positive organism, and the patient has a history of direct contact with animal meat, additional appropriate antibiotic coverage should be strongly considered, especially if there is no clinical improvement. Therefore, it is crucial that physicians think of this disease in at-risk patients, leading to the correct diagnosis and prompt treatment.

Treatment

To date, data concerning *E. rhusiopathiae* susceptibility are limited, but the results from in vitro studies have shown that the organism is most susceptible to penicillin, cephalosporins and imipenem, subsequently followed by piperacillin, ciprofloxacin, and clindamycin. However, it should be noted that in 2015, macrolide resistance was reported for the first time in a porcine isolate. There was an increase in the minimum inhibitory concentration (MIC) of clindamycin and erythromycin to *E. rhusiopathiae* by at least 128-fold[54]. Thus, patterns of resistance to macrolides should be further observed. Vancomycin, daptomycin, teicoplanin, trimethoprim-sulfamethoxazole, netilmicin, and gentamicin should not be used because of poor or absent activity[55,56]. Hence, *E. rhusiopathiae* disease should be suspected in cutaneous or systemic gram-positive infections that fail to respond to vancomycin. In overall, the antibiotic of choice is penicillin[57]. Oral penicillin V should be prescribed for seven days in patients with localized skin disease. Erysipeloid will be resolved after 48 hours of oral penicillin treatment while intravenous penicillin is recommended in a more serious *E. rhusiopathiae* infection. Ciprofloxacin or clindamycin is alternative in cases of penicillin allergy. Intravenous penicillin G should be administered in patients with diffuse skin infection or septicemia, which can be substituted with ceftriaxone, imipenem, or fluoroquinolones[3,19]. A longer period (4-6 weeks) of antibiotic treatment is required for patients who have bacteremia with endocarditis[3,39]. Due to lack of data from clinical trials, duration of antibiotic use should be based on clinical response.

Prevention

To prevent human transmission, disease among animals must be eliminated. However, this may be difficult to achieve due the enormous cost and numerous animal reservoirs. Targeting livestock, which contribute to the economy, will help to reduce human infection and prevent loss or revenue.

Gloves, frequent hand washing by using disinfectant soap, and prompt treatment for any minor injuries are primarily the disease prevention methods. In addition, avoidance of alcohol drinking may reduce a chance of serious infections. Disinfection and removal of contaminated organ may be required to control organism spreading. The commercial vaccine is not available in humans[58]. The relapse of infection may result from reduced or compromised immunity. The quarantine of reservoir hosts in various animals to limit organism widespread is impractical[14].

Conclusion

The article reviewed the knowledge regarding clinical manifestations and treatment of *Erysipelothrix*. Uncommon presentations, those that have been found recently, were further explored for possible mechanisms. Potential solution for disease control is suggested.

What is already known on this topic ?

Human *Erysipelothrix rhusiopathiae* infection was first reported over a century ago. The disease is transmitted primarily through work-related exposure to animal reservoirs. It typically presents as a local skin infection called erysipeloid, while systemic disease is rare. Treatment with penicillin results in favorable outcomes but the disease is resistant to vancomycin. Given that much of the conventional knowledge has been previously known, it is surprising that *Erysipelothrix* infection has not been eliminated. Indeed, it continues to occur with increasingly diverse and atypical manifestations, such septicemic forms in immunocompromised patients.

What this study adds ?

In this review, we identify the unsolved issues

and suggest possible solutions regarding the continuing occurrence of human *Erysipelothrix* infection. Firstly, disease control in animals is difficult. To reduce infection in animals, good farming practices and safe work procedures should be strictly implemented. Secondly, laboratory identification and isolation may have low yields from due to minute, slow-growing bacterial colonies and specimen contamination. In addition to conventional techniques, PCR methods, although not widely available, may be used to rapidly detect *Erysipelothrix* spp. in animal tissues. Importantly, the laboratory should be informed if this organism is suspected on clinical grounds. Thirdly, the diagnosis may be delayed due to unawareness of this disease or unfamiliarity with atypical presentations, especially in non-immunocompetent hosts. Physicians should have a high index of clinical suspicion, especially in patients with work-related exposure. Finally, due to misdiagnosis, antibiotic coverage may not be adequate. Penicillin, the drug of choice, or cephalosporins should be initiated as soon as possible.

Potential conflicts of interest

None.

References

1. Verbarg S, Rheims H, Emus S, Fruhling A, Kroppenstedt RM, Stackebrandt E, et al. *Erysipelothrix inopinata* sp. nov., isolated in the course of sterile filtration of vegetable peptone broth, and description of Erysipelotrichaceae fam. nov. Int J Syst Evol Microbiol 2004; 54: 221-5.
2. Takahashi T, Tamura Y, Yoshimura H, Nagamine N, Kijima M, Nakamura M, et al. *Erysipelothrix tonsillarum* isolated from dogs with endocarditis in Belgium. Res Vet Sci 1993; 54: 264-5.
3. Reboli AC, Farrar WE. *Erysipelothrix rhusiopathiae*: an occupational pathogen. Clin Microbiol Rev 1989; 2: 354-9.
4. Adamson HG. Erythema serpens of Morrant Baker, or Erysipeloid of Rosenbach. Proc R Soc Med 1909; 2: 4-5.
5. Fidalgo SG, Riley TV. Detection of *Erysipelothrix rhusiopathiae* in clinical and environmental samples. Methods Mol Biol 2004; 268: 199-205.
6. Wang Q, Fidalgo S, Chang BJ, Mee BJ, Riley TV. The detection and recovery of *Erysipelothrix* spp. in meat and abattoir samples in Western Australia. J Appl Microbiol 2002; 92: 844-50.
7. Gorby GL, Peacock JE Jr. *Erysipelothrix rhusiopathiae* endocarditis: microbiologic, epidemiologic, and clinical features of an occupational disease. Rev Infect Dis 1988; 10: 317-25.
8. Veraldi S, Girgenti V, Dassoni F, Gianotti R. Erysipeloid: a review. Clin Exp Dermatol 2009; 34: 859-62.
9. Wang Q, Chang BJ, Riley TV. *Erysipelothrix rhusiopathiae*. Vet Microbiol 2010; 140: 405-17.
10. Wood RL. Swine erysipelas-a review of prevalence and research. J Am Vet Med Assoc 1984; 184: 944-9.
11. Grieco MH, Sheldon C. *Erysipelothrix rhusiopathiae*. Ann N Y Acad Sci 1970; 174: 523-32.
12. Mahavanakul W, Limmathurotsakul D, Teerawattanasuk N, Peacock SJ. Invasive *Erysipelothrix rhusiopathiae* infection in northeast Thailand. Southeast Asian J Trop Med Public Health 2007; 38: 478-81.
13. Kositapantawong N. *Erysipelothrix rhusiopathiae* bacteremia with rare manifestation of diffused cutaneous skin lesions. J Infect Dis Antimicrob Agents 2011; 28: 59-62.
14. Brooke CJ, Riley TV. *Erysipelothrix rhusiopathiae*: bacteriology, epidemiology and clinical manifestations of an occupational pathogen. J Med Microbiol 1999; 48: 789-99.
15. Ozawa M, Yamamoto K, Kojima A, Takagi M, Takahashi T. Etiological and biological characteristics of *Erysipelothrix rhusiopathiae* isolated between 1994 and 2001 from pigs with swine erysipelas in Japan. J Vet Med Sci 2009; 71: 697-702.
16. Starr MP, Stolp H, Triiper HG, Balows A, Schlegel HG, editors. The prokaryotes: a handbook on habitats, isolation, and identification of bacteria. New York: Springer-Verlag; 1981.
17. Markey B, Leonard F, Archambault M, Cullinane A, Maquire D.Clinical veterinary microbiology. 2nd ed. Edinburgh: Elsevier; 2013.
18. Sneath PH, Abbott JD, Cunliffe AC. The bacteriology of erysipeloid. Br Med J 1951; 2: 1063-6.
19. Upapan P, Chayakulkeeree M. *Erysipelothrix rhusiopathiae* bacteremia without endocarditis associated with psoas abscess: the first case report in Thailand. J Med Assoc Thai 2014; 97 (Suppl 3): S232-6.
20. Dunbar SA, Clarridge JE 3rd. Potential errors in recognition of *Erysipelothrix rhusiopathiae*. J Clin Microbiol 2000; 38: 1302-4.
21. Takahashi T, Fujisawa T, Tamura Y, Suzuki S,

Muramatsu M, Sawada T, et al. DNA relatedness among *Erysipelothrix rhusiopathiae* strains representing all twenty-three serovars and *Erysipelothrix tonsillarum*. Int J Syst Bacteriol 1992; 42: 469-73.
22. Takeshi K, Makino S, Ikeda T, Takada N, Nakashiro A, Nakanishi K, et al. Direct and rapid detection by PCR of *Erysipelothrix* sp. DNAs prepared from bacterial strains and animal tissues. J Clin Microbiol 1999; 37: 4093-8.
23. To H, Koyama T, Nagai S, Tuchiya K, Nunoya T. Development of quantitative real-time polymerase chain reaction for detection of and discrimination between *Erysipelothrix rhusiopathiae* and other *Erysipelothrix* species. J Vet Diagn Invest 2009; 21: 701-6.
24. Shimoji Y. Pathogenicity of *Erysipelothrix rhusiopathiae*: virulence factors and protective immunity. Microbes Infect 2000; 2: 965-72.
25. Abrashev I, Orozova P. *Erysipelothrix rhusiopathiae* neuraminidase and its role in pathogenicity. Z Naturforsch C 2006; 61: 434-8.
26. King PF. Erysipeloid. Lancet 1946; 2: 196-8.
27. Nelson E. Five hundred cases of erysipeloid. Rocky Mt Med J 1955; 52: 40-2.
28. Feist H, Flossmann KD, Erler W. Nutrient demand of *Erysipelothrix* bacteria. Arch ExpVeterinarmed 1976; 30: 49-57.
29. Ehrlich JC. *Erysipelothrix rhusiopathiae* infection in man; report of a case with cutaneous bullae, in which cure was achieved with penicillin. Arch Intern Med (Chic) 1946; 78: 565-77.
30. Klauder JV. Terramycin, aureomycin, ilotycin in the treatment of experimental erysipeloid infection of the mouse. Arch Klin Exp Dermatol 1955; 200: 346-9.
31. Gransden WR, Eykyn SJ. *Erysipelothrix rhusiopathiae* endocarditis. Rev Infect Dis 1988; 10: 1228.
32. Kaye D. Infective endocarditis: Baltimore: Univerity Park Press; 1976.
33. Kramer MR, Gombert ME, Corrado ML, Ergin MA, Burnett V, Ganguly J. *Erysipelothrix rhusiopathiae* endocarditis. South Med J 1982; 75: 892.
34. Mandal BN, Malloch JA. Endocarditis due to *Erysipelothrix rhusiopathiae*. N Z Med J 1971; 73: 355-7.
35. Morris CA, Schwabacher H, Lynch PG, Cross CD, Dada TO. Two fatal cases of septicaemia due to *Erysipelothrix insidiosa*. J Clin Pathol 1965; 18: 614-7.
36. Fowler NO, Hamburger MH, Bove KE. Aortic valve perforation. Trans Am Clin Climatol Assoc 1967; 78: 218-29.
37. Rahal JJ. Jr, Simberkoff MS, Hyams PJ. Pseudomonas cepacia tricuspid endocarditis: treatment with trimethoprim, sulfonamide, and polymyxin B. J Infect Dis 1973; 128 (Suppl): 762-7.
38. Silberstein EB. *Erysipelothrix* endocarditis: report of a case with cerebral manifestations. JAMA 1965; 191: 862-4.
39. Ognibene FP, Cunnion RE, Gill V, Ambrus J, Fauci AS, Parrillo JE. *Erysipelothrix rhusiopathiae* bacteremia presenting as septic shock. Am J Med 1985; 78: 861-4.
40. Berge F, Dupon M, Morel D, Merville P. Photo quiz. Diagnosis: swine erysipelas (erysipeloid). Clin Infect Dis 1996; 22: 21, 129.
41. Garcia-Restoy E, Espejo E, Bella F, Llebot J. Bacteremia due to *Erysipelothrix rhusiopathiae* in immunocompromised hosts without endocarditis. Rev Infect Dis 1991; 13: 1252-3.
42. Jones N, Khoosal M. *Erysipelothrix rhusiopathiae* septicemia in a neonate. Clin Infect Dis 1997; 24: 511.
43. Romney M, Cheung S, Montessori V. *Erysipelothrix rhusiopathiae* endocarditis and presumed osteomyelitis. Can J Infect Dis 2001; 12: 254-6.
44. Denes E, Camilleri Y, Fiorenza F, Martin C. First case of osteomyelitis due to *Erysipelothrix rhusiopathiae*: pubic osteomyelitis in a gored farmer. Int J Infect Dis 2015; 30: 133-4.
45. Ruiz ME, Richards JS, Kerr GS, Kan VL. *Erysipelothrix rhusiopathiae* septic arthritis. Arthritis Rheum 2003; 48: 1156-7.
46. Simionescu R, Grover S, Shekar R, West BC. Necrotizing fasciitis caused by *Erysipelothrix rhusiopathiae*. South Med J 2003; 96: 937-9.
47. Hardman SC, Carr SJ, Swann RA. Peritoneal dialysis-related peritonitis with bacteraemia due to *Erysipelothrix rhusiopathiae*. Nephrol Dial Transplant 2004; 19: 1340-1.
48. Kim SR, Kwon MJ, Lee JH, Lee NY. Chronic meningitis caused by *Erysipelothrix rhusiopathiae*. J Med Microbiol 2007; 56: 1405-6.
49. Elvy J, Hanspal I, Simcock P. A case of *Erysipelothrix rhusiopathiae* causing bilateral endogenous endophthalmitis. J Clin Pathol 2008; 61: 1223-4.
50. Hocqueloux L, Poisson DM, Sunder S, Guilbert S, Prazuck T. Septic arthritis caused by *Erysipelothrix*

rhusiopathiae in a prosthetic knee joint. J Clin Microbiol 2010; 48: 333-5.
51. Feasi M, Bacigalupo L, Cappato S, Pontali E, Usiglio D, Rollandi GA, et al. *Erysipelothrix rhusiopathiae* intra-abdominal abscess. Int J Infect Dis 2010; 14: e81-e83.
52. Meric M, Ozcan SK. *Erysipelothrix rhusiopathiae* pneumonia in an immunocompetent patient. J Med Microbiol 2012; 61: 450-1.
53. Andrychowski J, Jasielski P, Netczuk T, Czernicki Z. Empyema in spinal canal in thoracic region, abscesses in paravertebral space, spondylitis: in clinical course of zoonosis *Erysipelothrix rhusiopathiae*. Eur Spine J 2012; 21 Suppl 4: S557-S563.
54. Xu CW, Zhang AY, Yang CM, Pan Y, Guan ZB, Lei CW, et al. First report of macrolide resistance gene erm(T) harbored by a novel small plasmid from *Erysipelothrix rhusiopathiae*. Antimicrob Agents Chemother 2015; 59: 2462-5.
55. Venditti M, Gelfusa V, Castelli F, Brandimarte C, Serra P. *Erysipelothrix rhusiopathiae* endocarditis. Eur J Clin Microbiol Infect Dis 1990; 9: 50-2.
56. Venditti M, Gelfusa V, Tarasi A, Brandimarte C, Serra P. Antimicrobial susceptibilities of *Erysipelothrix rhusiopathiae*. Antimicrob Agents Chemother 1990; 34: 2038-40.
57. Fidalgo SG, Longbottom CJ, Rjley TV. Susceptibility of *Erysipelothrix rhusiopathiae* to antimicrobial agents and home disinfectants. Pathology 2002; 34: 462-5.
58. Groschup MH, Timoney JF. Modified Feist broth as a serum-free alternative for enhanced production of protective antigen of *Erysipelothrix rhusiopathiae*. J Clin Microbiol 1990; 28: 2573-5.

การติดเชื้ออีริซิเพโลทริกซ์ในมนุษย์: ประเด็นที่ยังไม่ได้แก้และแนวทางการแก้ไข

ประสิทธิ์ อุพาพรรณ

การติดเชื้ออีริซิเพโลทริกซ์ในมนุษย์ไม่ได้พบได้ยากดังเช่นที่ผ่านมา ผู้เป็นโรคส่วนใหญ่ติดเชื้อจากสัตว์ ซึ่งมักสัมพันธ์กับการประกอบอาชีพที่มีโอกาสสัมผัสเชื้อนี้ การติดเชื้อในมนุษย์มีรายงานตั้งแต่ต้นปี พ.ศ. 2443 จนถึงปัจจุบัน ประเด็นที่ไม่ได้รับการแก้ไขของการติดเชื้อนี้ เนื่องจากการควบคุมโรคนี้ในสัตว์ยังไม่ดีเพียงพอ ความยากในการเพาะและระบุตัวเชื้อแบคทีเรียชนิดนี้ รวมถึงการวินิจฉัยที่ล่าช้า เนื่องจากโรคนี้พบได้ไม่บ่อยหรือไม่คุ้นชินกับโรค ร่วมกับโรคมีอาการแสดงได้หลากหลายมากขึ้น และการใช้ยาปฏิชีวนะที่ไม่เหมาะสมต่อโรคอันเนื่องจากการวินิจฉัยที่ผิดพลาดหรืออาจให้ยาที่เชื้อนี้ดื้อ ในบทความนี้ผู้นิพนธ์ได้รวบรวมประเด็นต่างๆ ที่มีในการติดเชื้ออีริซิเพโลทริกซ์ในมนุษย์และแนวทางในการแก้ไข