

Case Report

Streptococcus gallolyticus subspecies *pasteurianus* Meningitis in an Infant: A Case Report and Literature Review

Warunee Punpanich MD, PhD^{*,**},
Anurak Munsrichoom BSc, MSc^{***}, Surang Dejsirilert MSc^{****}

* Department of Pediatrics, Queen Sirikit National Institute of Child Health, Bangkok, Thailand

** College of Medicine, Rangsit University, Bangkok, Thailand

*** Division of Microbiology, Queen Sirikit National Institute of Child Health, Bangkok, Thailand

**** National Institute of Health, Department of Medical Sciences, Nonthaburi, Thailand

A case of 6-week-old male infant with meningitis and concurrent bacteremia caused by *Streptococcus gallolyticus* subspecies *pasteurianus* (*Streptococcus bovis* biotype II.2) is presented. The isolates were susceptible to all beta-lactam antibiotics. Nevertheless, delayed defervescence and lack of satisfactory clinical improvement after treatment with multiple beta-lactam antibiotics prompted the combination therapy of vancomycin and penicillin G. The patient completed a 2-week course of antibiotics and recovered uneventfully.

Keywords: *Streptococcus gallolyticus* subspecies *pasteurianus*, *S. bovis*, Meningitis, Infants, Case report

J Med Assoc Thai 2012; 95 (12): 1606-12

Full text. e-Journal: <http://jmat.mat.or.th>

Streptococcus gallolyticus subspecies *pasteurianus*, a non-enterococcal group D streptococcus formerly known as *Streptococcus bovis* biotype II.2, has been infrequently reported as a cause of invasive infections in young infants^(1,2) and adults^(3,4). As the biochemical and phenotypic characteristics of *S. bovis/gallolyticus* complex are not always clear-cut, the final differentiation of various subspecies requires the gene sequence analysis of 16S rRNA segment. Among adult populations, *S. bovis/gallolyticus* is commonly associated with underlying carcinoma of gastrointestinal tract. Among pediatric population, it is an uncommon cause of infection among infants and neonates. However, reports of invasive infection caused by this pathogen in children are rare including those reported by Cheung et al in 2000, Gavin et al in 2003, Onoyama et al in 2009, Floret et al in 2010, and Klatte et al in 2012 as a cause of neonatal sepsis and meningitis^(1,5-8). In the first two reports, the pathogen was identified as *S. bovis* biotype II.2, which is now known as *S. gallolyticus* subsp. *pasteurianus*. The objective of the present study was to provide a clinical description

and literature review of *Streptococcus gallolyticus* subspecies *pasteurianus* invasive infection in pediatric population. The study received approval from the Queen Sirikit National Institute of Child Health Ethics Committee (IRB No. 55-030).

Case Report

A 6-week-old male infant presented with acute fever and generalized tonic-clonic convulsion. He had a history of acute onset of high-grade fever for nine hours prior to the seizure requiring immediate medical attention. There was no report of concurrent respiratory or gastrointestinal symptoms. Up to the onset of seizure, the child had been well appearing without any noticeably decreased level of activity or consciousness. Prenatal, perinatal, and postnatal history were unremarkable with a birth weight of 3,600 grams. No family history of seizure or other neurological disorder was present. Neither intercurrent household illness nor pet contact was reported. He received both breast milk as well as formula. His developmental milestones were age-appropriate. Physical examination revealed a well-nourished male infant, relatively inactive, in mild discomfort but without apparent distress. His body temperature was 38.6° Celsius, blood pressure was 84/42, pulse rate was 140/minute, and respiratory rate was 46/minute.

Correspondence to:

Punpanich W, Department of Pediatrics, Queen Sirikit National Institute of Child Health, Bangkok 10400, Thailand.

Phone & Fax: 0-2354-8345

E-mail: warunee@gmail.com

Anterior fontanel was flat and 2 x 2 cm in size. No pallor, jaundice, nor rashes were noted. Pharynx, tonsils, and tympanic membrane were not injected. Heart, lung, and abdominal examination were unremarkable. Extremities and spine showed no abnormality. Neurological examination was grossly intact with reactive papillary reflex; muscle tone was normal; neck stiffness and Brudzinski's Sign were absent. Ankle clonus was positive with dorsiflexion of Babinski's reflex on both sides. He and his family lived in an apartment in urban Bangkok. Laboratory data showed hematocrit of 34.8%, white blood cell count (WBC) of $28.4 \times 10^9/L$ (neutrophil 53%, lymphocyte 36%, monocyte 9%), platelet count of $290 \times 10^9/L$, sodium 136, potassium 5.1, chloride 103, and carbon dioxide 27%. Provisional diagnosis was bacterial meningitis and lumbar puncture was performed. Cerebrospinal fluid (CSF) was slightly turbid with WBC of $1,910 \text{ cell/mm}^3$ (polymorphonuclear cell 96%, mononuclear cell 4%), red blood cell (RBC) 70 cell/mm^3 , CSF protein = 351 mg/dL, CSF sugar 93 mg/dL, blood sugar 138 mg/dL, and CSF gram stain and latex agglutination were negative. Urine analysis was normal. Empirical antimicrobial treatment was initiated with ceftriaxone 50 mg/kg/day at the emergency room and was later increased to 100 mg/kg/day after the CSF analysis result was obtained a few hours later at an in-patient ward. After initial treatment, the patient's condition was stabilized. No new onset of seizure was identified. The next day (day 2) of admission, axillary temperature spiked to around 39.3° and 39.8° Celsius and his general well-being did not improve significantly. Physical examination did not reveal any other possible cause; lumbar puncture for CSF analysis was repeated. The second CSF analysis revealed WBC of $3,165 \text{ cell/mm}^3$ (polymorphonuclear cell 94%, mononuclear cell 6%, RBC 650 cell/mm^3), protein 279 mg/dL and sugar 35 mg/dL, blood sugar 121 mg/dL. Latex and gram stain remained negative. Amikacin was added to ceftriaxone to expand coverage against gram-negative enteric bacteria. At seventy-two hours of admission, the third lumbar puncture and CSF analysis was attempted due to the sustained fever, but failed due to traumatic tap. Ceftriaxone and amikacin were then replaced by meropenem to cover potential drug resistant gram-negative pathogens. The peak temperature went down to around 38° and 38.5° Celsius after 24 hours of meropenem initiation. However, the temperature went back up to 39° Celsius within 72 hours after meropenem therapy. As a result, meropenem was replaced by the combination of

vancomycin and ciprofloxacin to expand coverage against drug resistant pneumococcus and beta-lactam resistant salmonella, which are among the most common cause of bacterial meningitis in Thailand. Temperature began to decline within 24 hours after the initiation of vancomycin plus ciprofloxacin and remained seizure-free thereafter. On day 8 of admission, hemoculture and CSF culture revealed streptococcus group D (non-enterococci) sensitive to penicillin, vancomycin, ceftriaxone, and cefotaxime by disc diffusion method. The pathogens were detected after 72 hours of incubation in broth at 37°C automate BacT/Alert 3D system. Ciprofloxacin was then replaced by penicillin G sodium. The fever gradually disappeared and clinical well-being significantly improved. Cranial ultrasonogram was performed on day 10 of admission showing minimal bilateral frontal, subdural effusion, and 1-cm widening of intra-hemispheric junction. The head circumference was measured daily and with no clinical sign indicative of intracranial complication such as hydrocephalus or ventriculitis. In addition, an echocardiogram was conducted to rule out bacterial endocarditis commonly associated with streptococcus group D bacteremia. The results did not reveal any associated endocarditis. A subsequent lumbar puncture was repeated on day 13 of admission, to assess the treatment response. The CSF analysis showed the reduction of WBC counts to 135 cell/mm^3 , PMN 26% and mononuclear 74%, protein 154.5 mg/dL, sugar 32 mg/dL, and blood sugar 101 mg/dL. CSF culture was negative.

The patient did not receive any additional investigation or work-up for detecting immuno deficiency. He continued to improve throughout his hospital stay and was discharged after completing a 2-week course of intravenous vancomycin and penicillin G sodium. The results from Department of Medical Sciences, National Institute of Health, Thailand, was later reported back to QSNICH. The pathogen identification was established by phenotypic and genotypic characterization. The isolates identified were gram-positive cocci, non-hemolytic, with positive Lancefield's D antigen and esculin hydrolysis. The isolates phenotypic characteristics⁽⁹⁾ were compatible with those of *Streptococcus bovis* biotype II as shown in Table 1 below. The genotypic characterization was performed using the in-house multiplex polymerase chain reaction (PCR), being developed at the Miscellaneous Bacteriology Laboratory, Department of Medical Science, Ministry of Public Health, Thailand, using primers specific to *S. agalactiae*⁽¹⁰⁾,

Table 1. Biochemical characteristics of streptococcus group D nonenterococci isolated from blood and cerebrospinal fluid

Characteristics	Result
Haemolysis	non-beta
Optochin	-
Bile solubility	-
Bile esculin	+
Growth in 6.5% NaCl broth	-
PYR	-
Esculin	+
VP	+
Mannitol	-
Melibiose	+
Sorbitol	-
Trehalose	+

PYR = pyrrolidonyl aminopeptidase; VP = Vogues Proskauer

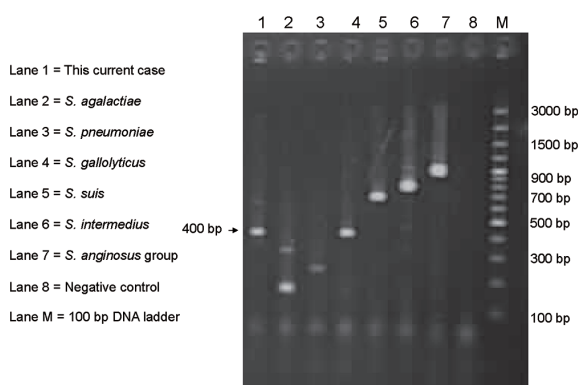


Fig. 1 Genotypic characterization by multiplex polymerase chain reaction for *S. gallolyticus* subsp. *pasteurianus*

S. pneumoniae⁽¹¹⁾, *S. gallolyticus*⁽¹²⁾, *S. suis*⁽¹³⁾, *S. intermedius*⁽¹⁴⁾ and *S. anginosus*⁽¹⁵⁾. The result of multiplex PCR, which was applied directly to bacterial colony, confirmed the identity of the organism as being *Streptococcus bovis* subspecies *pasteurianus* (Fig. 1).

The patient's last follow-up at 4 years of age, he appeared to have a normal developmental status with no hearing impairment or long term neurological sequelae.

Discussion

Invasive infection caused by *S. bovis/gallolyticus* among the adult population in many

Western countries is commonly known to be associated with carcinoma of the gastrointestinal tract especially colonic neoplasia⁽¹⁶⁻¹⁹⁾. In contrast, one report from Hong Kong found that the most common underlying condition found among adults with *S. bovis/gallolyticus* bacteremia was a biliary tract disease such as acute cholangitis and/or cholecystitis⁽²⁰⁾. Similarly, the first report of *S. bovis* biotype II infection in Thailand indicates that liver cirrhosis was an underlying disease of all seven adult cases presenting with spontaneous bacterial peritonitis⁽²¹⁾. Among the pediatric population, *S. bovis/gallolyticus* is considered an uncommon cause of invasive infection affecting mainly neonates and young infants⁽²²⁾. Nevertheless, the true burden of invasive infection caused by *S. bovis/gallolyticus* may be under estimated due to several reasons such as 1) the limited availability of technical resource to correctly identify this pathogen in many clinical settings, 2) the high chance of being misidentified as enterococci or other viridians streptococci due to their similar phenotypic characteristics⁽²³⁻²⁵⁾, and 3) the delay in obtaining the final report from another (and more specialized) microbiological laboratory center makes it unlikely for the patient's medical record and/or hospital database to be updated with the final result. Furthermore, there is more than one variant of *S. bovis/gallolyticus* that requires further detailed specification such as expanded biochemical tests^(23,25,26). For example, *S. bovis* can be identified as biotype II by its ability to hydrolyze starch and ferment mannitol^(23,26).

Infections caused by *S. bovis/gallolyticus* among neonates are rather indistinguishable from those caused by group B streptococcus⁽⁷⁾. Proper identification of the pathogen may have an important implication for the antimicrobial selection. For example, penicillin is generally considered to be adequate treatment for *S. bovis/gallolyticus* infection. On the other hand, those caused by enterococci and some viridan streptococcus may require the addition of vancomycin therapy due to their reduced susceptibility for penicillin and other beta-lactam antibiotics⁽²⁷⁻³⁴⁾. For this particular patient, however, delayed defervescence, despite the in vitro susceptibility, prompted the addition of vancomycin to the treatment regimen resulting in rapid defervescence within 24 hours. *S. bovis/gallolyticus* was reported to be isolated from both human and animal specimens such as pigeons or duckling⁽³⁵⁻⁴⁰⁾. A recent report in Thailand showed that *S. bovis*, along with other lactic acid bacteria, is commonly isolated from Pla-som, which is a Thai fermented fish product. This particular dish in which whole fish or fish fillets

are fermented with cooked rice or steamed sticky rice, salt and garlic, is a local food staple commonly consumed by many Thais⁽⁴¹⁾. Besides one case series report of invasive *S. bovis* infection from adults in Thailand⁽²¹⁾, the authors were unable to locate any previous report of *Streptococcus gallolyticus* subspecies *pasteurianus* causing invasive infection in Thai children published in Thai and English literature.

Despite the availability of the molecular genetics method, the identification and taxonomic classification of *S. bovis/gallolyticus* remains a challenge and rather confusing not only for general pediatricians or internists but also clinical microbiologists and infectious disease physicians⁽⁴²⁾. Recently, advances in molecular techniques have led to further clarification and the update of the current taxonomy of the so-called '*S. bovis/equinus* complex'^(22,43). The new method, based on DNA-DNA reassociation experiments by which four different DNA clusters are identified, has reclassified what was termed *S. bovis*. Group II contains three different *S. gallolyticus* subspecies⁽⁴⁴⁾, one of which is isolated from our patients *i.e.*, *Streptococcus gallolyticus* subspecies *gallolyticus*. However, it is essential that attending physicians be notified of its former name "*S. bovis*" along with the updated recent nomenclature when reporting the result. This is essential since the lack of awareness of this change can lead to delayed investigation that may lead to a diagnosis of serious underlying condition especially carcinomatous change of colon⁽⁴⁵⁾. Despite the availability of information on the new classification system⁽⁴⁴⁾, most microbiological laboratories are currently neither able to accurately identify the member of *S. bovis* group nor adopt the new and complicated nomenclature which remains subject for debate⁽⁴⁶⁾.

Studies in adults in many western countries indicate that infection by *Streptococcus gallolyticus* subsp. *gallolyticus* (formerly known as *S. bovis* biotype I) is associated with colonic neoplasia^(18,24,47) whereas *S. bovis* biotype II appears to be associated with meningitis^(48,49). This might be due to a unique interaction between the biotype of pathogens and host as proposed by Ruoff and colleagues⁽²⁴⁾. On the other hand, among reports on invasive *S. bovis/gallolyticus* infection in neonates, detailed specification, and biotyping of causative strains were generally not reported. However, in a few case series studies with nine cases of neonatal *S. bovis* infection where biotype data were available, all of them were caused by *S. bovis* biotype II^(6,7,50,51). Therefore, the identification of

the biotype may be clinically relevant. For example, as mentioned earlier, *S. bovis/gallolyticus* (biotype I) septicemia in adults is associated with underlying colonic neoplasia and concomitant endocarditis. In contrast, *S. bovis/gallolyticus* variant (particularly biotype II) appears to be related to meningitis and/or septicemia affecting mainly young infants or neonates although reported cases in adults also exist. Similarly, a recent report on bloodstream infections caused by new species included in the old *S. bovis* group illustrated the association between causative species and clinical manifestations. For example, *Streptococcus gallolyticus* subsp. *gallolyticus* blood stream infection is a surrogate for endocarditis and/or intestinal pathology, whereas *Streptococcus gallolyticus* subsp. *pasteurianus* blood stream infection is suggestive of an underlying hepato-biliary disease⁽⁵²⁾.

Further investigations are required to examine the underlying mechanisms responsible for these apparent associations between clinical syndromes and the bacteria's biotype.

Conclusion

The present report describes a case of invasive infection in a young infant due to *Streptococcus gallolyticus* subspecies *pasteurianus*. The public health significance of this pathogen as a cause of invasive infection in infants and children remains to be elucidated.

Acknowledgement

The authors wish to thank Vipa Treeratweeraphong, Chief of Microbiology Division and all staff of the Division of Microbiology, Department of Pathology, Queen Sirikit National Institute of Child Health for their kind assistance and support in providing important laboratory data and enabling us to complete this work.

Potential conflicts of interest

None.

References

1. Klatte JM, Clarridge JE III, Bratcher D, Selvarangan R. A longitudinal case series description of meningitis due to *Streptococcus gallolyticus* subsp. *pasteurianus* in infants. *J Clin Microbiol* 2012; 50: 57-60.
2. Nagamatsu M, Takagi T, Ohyanagi T, Yamazaki S, Nobuoka S, Takemura H, et al. Neonatal meningitis caused by *Streptococcus gallolyticus*

- subsp. *pasteurianus*. J Infect Chemother 2012; 18: 265-8.
3. Akahane T, Takahashi K, Matsumoto T, Kawakami Y. A case of peritonitis due to *Streptococcus gallolyticus* subsp. *pasteurianus*. Kansenshogaku Zasshi 2009; 83: 56-9.
 4. Sturt AS, Yang L, Sandhu K, Pei Z, Cassai N, Blaser MJ. *Streptococcus gallolyticus* subspecies *pasteurianus* (biotype II/2), a newly reported cause of adult meningitis. J Clin Microbiol 2010; 48: 2247-9.
 5. Floret N, Bailly P, Thouverez M, Blanchot C, Alez-Martin D, Menget A, et al. A cluster of bloodstream infections caused by *Streptococcus gallolyticus* subspecies *pasteurianus* that involved 5 preterm neonates in a university hospital during a 2-month period. Infect Control Hosp Epidemiol 2010; 31: 194-6.
 6. Cheung M, Pelot M, Nadarajah R, Kohl S. Neonate with late onset *Streptococcus bovis* meningitis: case report and review of the literature. Pediatr Infect Dis J 2000; 19: 891-3.
 7. Gavin PJ, Thomson RB, Jr., Horng SJ, Yogev R. Neonatal sepsis caused by *Streptococcus bovis* variant (biotype II/2): report of a case and review. J Clin Microbiol 2003; 41: 3433-5.
 8. Onoyama S, Ogata R, Wada A, Saito M, Okada K, Harada T. Neonatal bacterial meningitis caused by *Streptococcus gallolyticus* subsp. *pasteurianus*. J Med Microbiol 2009; 58: 1252-4.
 9. Facklam R. What happened to the streptococci: overview of taxonomic and nomenclature changes. Clin Microbiol Rev 2002; 15: 613-30.
 10. Dmitriev A, Bhide M, Mikula I. cpn60 Gene based multiplex-PCR assay for simultaneous identification of Streptococcal species. Acta Veterinaria Brno 2006; 75: 235-40.
 11. Stralin K, Backman A, Holmberg H, Fredlund H, Olcen P. Design of a multiplex PCR for *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Mycoplasma pneumoniae* and *Chlamydia pneumoniae* to be used on sputum samples. APMIS 2005; 113: 99-111.
 12. Sasaki E, Osawa R, Nishitani Y, Whiley RA. Development of a diagnostic PCR assay targeting the Mn-dependent superoxide dismutase gene (*sodA*) for identification of *Streptococcus gallolyticus*. J Clin Microbiol 2004; 42: 1360-2.
 13. Okwumabua O, O'Connor M, Shull E. A polymerase chain reaction (PCR) assay specific for *Streptococcus suis* based on the gene encoding the glutamate dehydrogenase. FEMS Microbiol Lett 2003; 218: 79-84.
 14. Takao A, Nagamune H, Maeda N. Identification of the anginosus group within the genus *Streptococcus* using polymerase chain reaction. FEMS Microbiol Lett 2004; 233: 83-9.
 15. Reissmann S, Friedrichs C, Rajkumari R, Itzek A, Fulde M, Rodloff AC, et al. Contribution of *Streptococcus anginosus* to infections caused by groups C and G streptococci, southern India. Emerg Infect Dis 2010; 16: 656-63.
 16. Abdulmir AS, Hafidh RR, Abu BF. The association of *Streptococcus bovis/gallolyticus* with colorectal tumors: the nature and the underlying mechanisms of its etiological role. J Exp Clin Cancer Res 2011; 30: 11.
 17. Klein RS, Recco RA, Catalano MT, Edberg SC, Casey JJ, Steigbigel NH. Association of *Streptococcus bovis* with carcinoma of the colon. N Engl J Med 1977; 297: 800-2.
 18. Murray HW, Roberts RB. *Streptococcus bovis* bacteremia and underlying gastrointestinal disease. Arch Intern Med 1978; 138: 1097-9.
 19. Shanan S, Gumaa SA, Sandstrom G, Abd H. Significant Association of *Streptococcus bovis* with Malignant Gastrointestinal Diseases. Int J Microbiol 2011; 2011: 792019.
 20. Lee RA, Woo PC, To AP, Lau SK, Wong SS, Yuen KY. Geographical difference of disease association in *Streptococcus bovis* bacteraemia. J Med Microbiol 2003; 52: 903-8.
 21. Vilaichone RK, Mahachai V, Kullavanijaya P, Nunthapisud P. Spontaneous bacterial peritonitis caused by *Streptococcus bovis*: case series and review of the literature. Am J Gastroenterol 2002; 97: 1476-9.
 22. Siegel JD, McCracken GH Jr. Group D streptococcal infections. J Pediatr 1978; 93: 542-3.
 23. Ruoff KL, Ferraro MJ, Holden J, Kunz LJ. Identification of *Streptococcus bovis* and *Streptococcus salivarius* in clinical laboratories. J Clin Microbiol 1984; 20: 223-6.
 24. Ruoff KL, Miller SI, Garner CV, Ferraro MJ, Calderwood SB. Bacteremia with *Streptococcus bovis* and *Streptococcus salivarius*: clinical correlates of more accurate identification of isolates. J Clin Microbiol 1989; 27: 305-8.
 25. Facklam RR. Physiological differentiation of viridans streptococci. J Clin Microbiol 1977; 5: 184-201.
 26. Facklam RR. Recognition of group D streptococcal

- species of human origin by biochemical and physiological tests. *Appl Microbiol* 1972; 23: 1131-9.
27. Chlebicki MP, Kurup A. Vancomycin-resistant enterococcus: a review from a Singapore perspective. *Ann Acad Med Singapore* 2008; 37: 861-9.
 28. Werner G, Coque TM, Hammerum AM, Hope R, Hryniewicz W, Johnson A, et al. Emergence and spread of vancomycin resistance among enterococci in Europe. *Euro Surveill* 2008; 13: 19046.
 29. Sood S, Malhotra M, Das BK, Kapil A. Enterococcal infections & antimicrobial resistance. *Indian J Med Res* 2008; 128: 111-21.
 30. Arias CA, Murray BE. Emergence and management of drug-resistant enterococcal infections. *Expert Rev Anti Infect Ther* 2008; 6: 637-55.
 31. Tacconelli E, Cataldo MA. Vancomycin-resistant enterococci (VRE): transmission and control. *Int J Antimicrob Agents* 2008; 31: 99-106.
 32. Bruckner L, Gigliotti F. Viridans group streptococcal infections among children with cancer and the importance of emerging antibiotic resistance. *Semin Pediatr Infect Dis* 2006; 17: 153-60.
 33. Yaniv LG, Potasman I. Iatrogenic meningitis: an increasing role for resistant viridans streptococci? Case report and review of the last 20 years. *Scand J Infect Dis* 2000; 32: 693-6.
 34. Levy CS, Kogulan P, Gill VJ, Croxton MB, Kane JG, Lucey DR. Endocarditis caused by penicillin-resistant viridans streptococci: 2 cases and controversies in therapy. *Clin Infect Dis* 2001; 33: 577-9.
 35. Hogg R, Pearson A. *Streptococcus gallolyticus* subspecies *gallolyticus* infection in ducklings. *Vet Rec* 2009; 165: 297-8.
 36. van der Toorn F, Lumeij JT. *Streptococcus gallolyticus* infections in racing pigeons, a literature review. *Tijdschr Diergeneeskd* 2001; 126: 66-71.
 37. Chadfield MS, Christensen JP, Decostere A, Christensen H, Bisgaard M. Geno- and phenotypic diversity of avian isolates of *Streptococcus gallolyticus* subsp. *gallolyticus* (*Streptococcus bovis*) and associated diagnostic problems. *J Clin Microbiol* 2007; 45: 822-7.
 38. Vanrobaeys M, De Herdt P, Charlier G, Ducatelle R, Haesebrouck F. Ultrastructure of surface components of *Streptococcus gallolyticus* (*S. bovis*) strains of differing virulence isolated from pigeons. *Microbiology* 1999; 145 (Pt 2): 335-42.
 39. Devriese LA, Vandamme P, Pot B, Vanrobaeys M, Kersters K, Haesebrouck F. Differentiation between *Streptococcus gallolyticus* strains of human clinical and veterinary origins and *Streptococcus bovis* strains from the intestinal tracts of ruminants. *J Clin Microbiol* 1998; 36: 3520-3.
 40. Vanrobaeys M, De Herdt P, Haesebrouck F, Ducatelle R, Devriese LA. Secreted antigens as virulence associated markers in *Streptococcus bovis* strains from pigeons. *Vet Microbiol* 1996; 53: 339-48.
 41. Kopermsub P, Yunchalard S. Identification of lactic acid bacteria associated with the production of plaa-som, a traditional fermented fish product of Thailand. *Int J Food Microbiol* 2010; 138: 200-4.
 42. Beck M, Frodl R, Funke G. Comprehensive study of strains previously designated *Streptococcus bovis* consecutively isolated from human blood cultures and emended description of *Streptococcus gallolyticus* and *Streptococcus infantarius* subsp. *coli*. *J Clin Microbiol* 2008; 46: 2966-72.
 43. Poyart C, Quesne G, Trieu-Cuot P. Taxonomic dissection of the *Streptococcus bovis* group by analysis of manganese-dependent superoxide dismutase gene (*sodA*) sequences: reclassification of '*Streptococcus infantarius* subsp. *coli*' as *Streptococcus lutetiensis* sp. nov. and of *Streptococcus bovis* biotype 11.2 as *Streptococcus pasteurianus* sp. nov. *Int J Syst Evol Microbiol* 2002; 52: 1247-55.
 44. Schlegel L, Grimont F, Ageron E, Grimont PA, Bouvet A. Reappraisal of the taxonomy of the *Streptococcus bovis*/*Streptococcus equinus* complex and related species: description of *Streptococcus gallolyticus* subsp. *gallolyticus* subsp. nov., *S. gallolyticus* subsp. *macedonicus* subsp. nov. and *S. gallolyticus* subsp. *pasteurianus* subsp. nov. *Int J Syst Evol Microbiol* 2003; 53: 631-45.
 45. van't Wout JW, Bijlmer HA. Bacteremia due to *Streptococcus gallolyticus*, or the perils of revised nomenclature in bacteriology. *Clin Infect Dis* 2005; 40: 1070-1.
 46. Romero B, Morosini MI, Loza E, Rodriguez-Banos M, Navas E, Canton R, et al. Reidentification of *Streptococcus bovis* isolates causing bacteremia according to the new taxonomy criteria: still an issue? *J Clin Microbiol* 2011; 49: 3228-33.

47. Herrero IA, Rouse MS, Piper KE, Alyaseen SA, Steckelberg JM, Patel R. Reevaluation of *Streptococcus bovis* endocarditis cases from 1975 to 1985 by 16S ribosomal DNA sequence analysis. J Clin Microbiol 2002; 40: 3848-50.
48. Clarridge JE III, Attorri SM, Zhang Q, Bartell J. 16S ribosomal DNA sequence analysis distinguishes biotypes of *Streptococcus bovis*: *Streptococcus bovis* Biotype II/2 is a separate genospecies and the predominant clinical isolate in adult males. J Clin Microbiol 2001; 39: 1549-52.
49. Coret FF, Vilchez Padilla JJ, Igual AR, Ferrando GJ. *Streptococcus bovis* meningitis: no association with colonic malignancy. Clin Infect Dis 1993; 17: 527-8.
50. Grant RJ, Whitehead TR, Orr JE. *Streptococcus bovis* meningitis in an infant. J Clin Microbiol 2000; 38: 462-3.
51. Parker MT, Ball LC. Streptococci and aerococci associated with systemic infection in man. J Med Microbiol 1976; 9: 275-302.
52. Gomez-Garces JL, Gil Y, Burillo A, Wilhelmi I, Palomo M. Diseases associated with bloodstream infections caused by the new species included in the old *Streptococcus bovis* group. Enferm Infecc Microbiol Clin 2012; 30: 175-9.

**เยื่อหุ้มสมองอักเสบจากเชื้อ *Streptococcus gallolyticus subspecies pasteurianus* ในเด็กทารก:
รายงานผู้ป่วย 1 ราย**

วารุณี พรรณพานิช, อนุรักษ์ มั่นศรีชุม, สุรางค์ เดชศิริเลิศ

รายงานผู้ป่วยทารกเพศชาย อายุ 6 สัปดาห์ มีอาการเยื่อหุ้มสมองอักเสบและติดเชื้อในกระแสเลือดจากเชื้อ *Streptococcus gallolyticus subspecies pasteurianus* เชื้อไวต่อยาปฏิชีวนะชนิด เบต้า-แลคแตมทุกชนิด อย่างไรก็ตาม หลังการรักษาด้วยยาในกลุ่มเบต้า-แลคแตมหลายขนาน อาการทางคลินิกไม่ดีขึ้น จึงได้ให้การรักษาด้วย vancomycin ร่วมกับ penicillin ผู้ป่วยได้รับการรักษาจนครบ 2 สัปดาห์ อาการดีขึ้นจนกลับสู่ภาวะปกติโดยไม่มีภาวะแทรกซ้อน