Bacterial identification of acne vulgaris

Lovenia Sari*, Nelva Karmila Jusuf, Imam Budi Putra

ABSTRACT

Background: Acne vulgaris (AV) is a chronic inflammation of the pilosebaceous unit with clinical polymorphic lesion consist of non-inflammatory (open and closed comedones) and inflammatory lesions (papules, pustules, and nodules) with varying degree of inflammation and depth. Earlier studies showed that other bacteria might also found and played a role in acne pathogenesis besides Cutibacterium acnes (C. acnes).

Patients and Methods: This descriptive observational study used cross-sectional method. Samples were collected from 40 subjects with AV. We took the samples from non-inflammatory (closed comedones) and inflammatory lesions (pustule) in each subject, followed by Gram staining, aerobic and anaerobic bacterial culture, and bacterial identification. This research has been approved by the Ethical Committee, Faculty of Medicine, University of Sumatera Utara.

Results: There were 12 bacterial species that were identified from 80 samples. We identified Cutibacterium acnes (21,2%) in anaerobic culture. While in aerobic culture, we identified Staphylococcus epidermidis (47,5%), Staphylococcus hominis (17,5%), Staphylococcus aureus (8,7%), Staphylococcus haemolyticus (8,7%), Leuconostoc mesenteroides (6,2%), Micrococcus luteus (3,7%), Kocuria varians (2,5%), Staphylococcus vitulinus (1,2%), Staphylococcus cohnii (1,2%), Staphylococcus arlettae (1,2%) and Dermacoccus nishinomyaensis (1,2%).

Conclusion: The two most common bacteria in acne vulgaris are Staphylococcus epidermidis and Cutibacterium acnes

Keywords: acne vulgaris, bacteria, culture, identification

Cite this Article: Sari, L., Jusuf, N.K., Putra, I.B. 2020. Bacterial identification of acne vulgaris. Bali Medical Journal 9(3): 753-756. DOI: 10.15562/bmj.v9i3.1737

INTRODUCTION

Acne vulgaris (AV) is a chronic inflammation of the pilosebaceous unit with clinical polymorphic lesion consist of non-inflammatory (open and closed comedones) and inflammatory lesions (papules, pustules, and nodules) with varying degree of inflammation. AV commonly is self-limiting disease and often found in the adolescence period.1,2,3 Although AV is a self-limiting disease, it can cause sequels such as scar tissue and pigmentary changes that can persist for a lifetime and decrease the Patient’s quality of life and cause the psychological disorder.1,2,4,5

The pathogenesis of AV is multifactorial, consists of hyperproliferation of infundibulum, excess sebum production, inflammation, and colonisation of Propionibacterium acnes.1 Previous studies reported that other bacteria found and played a role in the pathogenesis of AV besides Propionibacterium acnes.5 Genomic and metagenomic investigations recently led to change in Propionibacterium acnes’ denomination to Cutibacterium acnes (C. acnes).7 Various studies demonstrated that other bacteria, namely Staphylococcus epidermidis, Staphylococcus aureus, Micrococcus spp. were related to the pathogenesis of AV other than Propionibacterium acnes.6 However, the role of these microorganisms in pathogenesis AV is still controversial.5,10

This study was undertaken to identify bacterial anaerobically and aerobically patterns from the non-inflammatory lesion (closed comedones) and the inflammatory lesion (pustule) of AV patients.

METHODS

This study has been approved from the Ethics Commission in Faculty of Medicine University of Sumatera Utara/Adam Malik Hospital number 807/TGL/KEPK FK USU-RSUP HAM/2019. This study was a descriptive cross-sectional study involving 40 acne vulgaris subjects of Dermatology and Venereology outpatient Clinic University of Sumatera Utara Hospital from October till December 2019. Acne vulgaris diagnosed based on history taking and physical examinations. Inclusion criteria were patients who have been diagnosed with acne vulgaris, age more than 18 years, and signed informed consent. The exclusion criteria included pregnancy and breastfeeding, received a systemic antibiotic in the last two weeks, and topical antibiotic in the previous one weeks.

*Corresponding to: Lovenia Sari
Postgraduate Master of Clinical Medicine Department of Dermatology and Venereology, Faculty of Medicine University of Sumatera Utara, University of Sumatera Utara Hospital, Medan, Indonesia

lovenasari@yahoo.co.uk

Received: 2020-01-11
Accepted: 2020-09-20
Published: 2020-11-07
Patient demographic data included age and gender taken from medical records history. The researcher recorded patient demographic data included age, gender, phone number, and address. Then, the Patient signed informed consent. Sample collected from each non-inflammatory (closed comedones) and inflammatory lesions (pustule) in each subject. Specimens were taken using a sterilized comedones extractor. Then, using a sterilized swab moistened with nutrient broth. The specimens were immediately split into anaerobic and aerobic conditional treatments in jars containing blood agar and Brucella blood agar. Impression smears were taken on a clean slide for Grams staining. The samples were immediately inoculated on, then incubated both anaerobically and aerobically at 37°C for 24 – 48 hours. An AnaeroGen® Compact sachet was placed into each anaerobic jar for the isolation of anaerobic bacteria. No sachets were used for the isolation of aerobic bacteria. Bacterial identification performed with Vitek® 2 compact (Biomerieux, France).

Table 1. Demographic characteristics of subjects

| Characteristics | Frequency (n=40) |
|-----------------|-----------------|
| Gender          | number (n)      | percentage (%) |
| Male            | 14              | 35             |
| Female          | 26              | 65             |
| Age             | number (n)      | percentage (%) |
| 18-25 years old | 29              | 72,5           |
| 26-35 years old | 11              | 27,5           |
| Total           | 40              | 100            |

Table 2. Bacterial distribution of acne vulgaris

| Bacteria              | Frequency (n=80) |
|-----------------------|-----------------|
|                       | number (n)      | percentage (%) |
| Anaerobic bacteria    |                 |                |
| C. acnes              | 17              | 21,2           |
| Aerobic bacteria      |                 |                |
| S. epidermidis        | 38              | 47,5           |
| S. hominis            | 14              | 17,5           |
| S. aureus             | 7               | 8,7            |
| S. haemolyticus       | 7               | 8,7            |
| Leuconostoc mesenteries| 5               | 6,2             |
| Micrococcus luteus    | 3               | 3,7            |
| Kocuria varians       | 2               | 2,5            |
| S. vitulinus          | 1               | 1,2            |
| S. cohnii             | 1               | 1,2            |
| S. arlettae           | 1               | 1,2            |
| Dermacoccus nishinomyaensis | 1 | 1,2 |
| Mixed growth          |                 |                |
| C. acnes and S. epidermidis | 11 | 13,7   |
| C. acnes and S. hominis| 3              | 3,7            |
| C. acnes and S. aureus| 2               | 2,5            |
| C. acnes and S. haemolyticus | 1 | 1,2 |
| Total                 | 80              | 100            |

Data that has been collected then processed using statistical package for the social sciences (SPSS) version 23.0 with descriptive statistical analysis for age, gender, anaerobic and aerobic bacteria distribution.

RESULTS

The Patient’s demographic characteristics are shown in Table 1. Most patients were in the age group 18-25 years old with 29 subjects (72,5%), the age group 26-35 years old with 11 subjects (27,5%). Based on gender, the female gender was dominant (65%) compare to the male gender (35%).

The bacterial distribution of subjects describes in Table 2. Twelve bacterial species were identified from 80 samples. We identified Cutibacterium acnes in 17 samples (21,2%) in anaerobic culture. While in aerobic culture, we identified Staphylococcus epidermidis (S. epidermidis) in 38 samples (47,5%), Staphylococcus hominis (S. hominis) in 14 samples (17,5%), Staphylococcus aureus (S. aureus) in 7 samples (8,7%), Staphylococcus haemolyticus (S. haemolyticus) in 7 samples (8,7%), Leuconostoc mesenteries in 5 samples (6,2%), Micrococcus luteus in 3 samples (3,7%), Kocuria varians in 2 samples (2,5%), Staphylococcus vitulinus (S. vitulinus) in 1 sample (1,2%), Staphylococcus cohnii (S. cohnii) in 1 sample (1,2%), Staphylococcus arlettae (S. arlettae) in 1 sample (1,2%) and Dermacoccus nishinomyaensis in 1 sample (1,2%).

The most common bacteria found in anaerobic culture was Cutibacterium acnes (21,2%), wherein aerobic culture was Staphylococcus epidermidis (47,5%). The most common mixed growth bacteria found in AV lesions was Cutibacterium acnes concomitant with Staphylococcus epidermidis (13,7%).

DISCUSSION

Total of 40 AV patients were involved in this study with a total of 80 samples. Acne vulgaris mostly found in the age group 18-25 years old, with primarily female gender affected. Skroza et al. also reported that 12 – 25 years old was the majority age group of AV patients. Eyaboglu et al. also reported in their study that females were more affected by AV than males.

In this study, we found 12 bacterial species from 80 samples. This study supported previous studies’ results because not only Cutibacterium acnes, but other bacteria were also found from AV lesions. The two most common bacteria are Staphylococcus epidermidis (47,5%) and Cutibacterium acnes (21,2%). Overall, we found Cutibacterium acnes, Staphylococcus epidermidis, Staphylococcus hominis,
Propionibacterium acnes provides favorable anaerobic conditions to grow innate host components defense and protects them against significant human inflammation. This similar finding conducted by Srikanth et al. also identified bacteria in comedones lesion, where the most frequent bacteria found was Propionibacterium acnes (37.2%), followed by Staphylococcus epidermidis (30.2%).

In this study, the two most common bacteria found were Cutibacterium acnes concomitant with Staphylococcus epidermidis. This similar finding also found in the study of Biswal et al. from all AV samples, 13.75% of samples showed mixed growth (aerobic and anaerobic growth), and the most common mixed growth was Propionibacterium acnes and Staphylococcus epidermidis.

Acne vulgaris is one of the most common skin diseases, predominantly seen in adolescence and also a multifactorial disease in which Cutibacterium acnes is thought to play an essential role in the pathogenesis of inflamed lesions. Propionibacterium acnes is a gram-positive and anaerobic bacteria that colonies in the human skin’s sebaceous glands and hair follicles. The pathogenesis of AV is based on multiple factors, such as increased sebum production, Propionibacterium acnes proliferation, and inflammation.

The role of bacteria other than Propionibacterium acnes in the pathogenesis of AV is still controversial. Lipase (geh1 gene) and delta hemolysin (hld gene) are two virulence factors produced by Staphylococcus epidermidis in AV development that impacts acne inflammation. Staphylococcus epidermidis also secreted exopolysaccharide intercellular adhesin (PIA), which is responsible for biofilm formation and protects them against significant human innate host components defense. This biofilm provides favorable anaerobic conditions to grow Propionibacterium acnes.

**CONCLUSION**

This study concluded that the two most common bacteria identified in acne vulgaris are Staphylococcus epidermidis and Cutibacterium acnes.

**ACKNOWLEDGMENTS**

We want to express thanks of gratitude to the Head of the Department of Dermatology and Venereology of Faculty of the Medicine University of Sumatera Utara and University of Sumatera Utara Hospital.

**ABBREVIATIONS**

AV, acne vulgaris; C. acnes, Cutibacterium acnes; geh1 gene, lipase; hld gene, hemolysin; P. acnes, Propionibacterium acnes; PIA, exopolysaccharide intercellular adhesin; S. aureus, Staphylococcus aureus; S. epidermidis, Staphylococcus epidermidis; S. haemolyticus, Staphylococcus haemolyticus; S. hominis, Staphylococcus hominis; SPSS, statistical package for the social sciences.

**AUTHOR CONTRIBUTION**

All authors have contributed to all processes in this research, including preparation, data gathering, and analysis, drafting, and approval for publication of this manuscript.

**FUNDING**

The authors are responsible for all of the study funding without grant or any external source of funding.

**CONFLICT OF INTEREST**

The authors declare no conflict of interest regarding the publication of this article.

**REFERENCES**

1. Goh C, Cheng C, Agak G, Zaenglein AL, Graber EM, Thiboutot DM, Kim J. Acneiform Disorder. In: Kang S, Amagai M, Bruckner AL, Enk AH, Margolis DJ, McMichael AJ, Orringer JS, editors. Fitzpatrick’s dermatology in general medicine. First Volume. 9th editions. New York: The McGraw Hill Companies; 2019:1391-1418.

2. Layton AM, Eady EA, Zouboulis CC. Acne. In: Griffith C, Barker J, Bleiker T, Chalmers R, Creamer D, editors. Rooks Textbook of Dermatology. Third Volume. 9th editions. United Kingdom: Wiley Blackwell; 2016: 90.1-67

3. Barratt H, Hamilton F, Car J, Lyons C, Layton A, Majeed A. Outcomes measures in acne vulgaris: systematic review. British Journal of Dermatology. 2009;160(3):132-6.

4. Kraft J, Freiman A. Management of acne. Can Med Assoc J. 2011; 183(7):430-5.
5. Thiboutot D, Gollnick H, Bettoli V, Dréno B, Kang S, Leyden JJ, et al. New insights into the management of acne: an update from the global alliance to improve outcomes in acne group. J Am Acad Dermatol. 2009;60(5 Suppl):1–50.
6. Beylot C, Auffret N, Poli F, Claudel JP, Leccia MT, Del Giudice P, dkk. Propionibacterium acnes: An update on its role in the pathogenesis of acne. J Eur Acad Dermatol Venereol. 2014;28(3):271-8.
7. Dreno B, Pecastaings S, Corver S, Veraldi S, Khammari A, Roques C. Cutibacterium acnes (Propionibacterium acnes) and acne vulgaris: a brief look at the latest updates. Journal of the European Academy of Dermatology and Venereology. 2018; 32(2):5-14
8. Hassanzadeh P, Bahmani M, Mehrabani D. Bacterial resistance to antibiotics in acne vulgaris: An in vitro study. Indian J Dermatol. 2008;53(3):122.
9. Rocha M, Bagatin E. Skin barrier and microbiome in acne. Arch Dermatol Res. 2017:1-5.
10. Kumar B, Pathak R, Mary PB, Jha D, Sardana K, Gautam HK. New insights into acne pathogenesis: Exploring the role of acne-associated microbial populations. Derm Sinica. 2016;34(2):67-73.
11. Skroza N, Tulino E, Mambrin A, Zuber S, Balduzzi V, et al. Adult Acne Versus Adolescent Acne: A Retrospective Study of 1.167 Patients. J Clin Aesthet Dermatol. 2018;11(1): 21-25.
12. Eyuboglu M, Kalay I, Eyuboglu D. Evaluation of Adolescents Diagnosed with Acne Vulgaris for Qualityof Life and Psychosocial Challenges. Indian J Dermatol. 2018;63(2): 131-35.
13. Moon SH, Roh HS, Kim YH, Kim JE, Ko JY, Ro YS. Antibiotic resistance of microbial strains isolated from Korean acne patients. J Dermatol. 2012;39(10): 833-7.
14. Sylvia L, Kusnandar E, Lestari S. Hubungan antara Jenis Mikroorganisme yang ditemukan pada Lesi Akne dengan Jenis Lesi Akne. [thesis]. Padang: Universitas Andalas; 2010.
15. Srikanth M, Kalyani CS, Mohan N, Sridhar K, Padmaja JI. Bacteriology of acne. Journal of Evolutions of Medical and Dental Sciences. 2015;4(19):3267-74.
16. Syahrial MA, Nasution D, Jusuf NK, Lubis SE, Pola Resisten Propionibacterium acnes Terhadap Antibiotika Oral Pada Pasien Akne Vulgaris di RSUP H. Adam Malik Medan. [thesis], Medan: Universitas Sumatera Utara; 2009.
17. Biswal I, Gaind R, Kumar N, Mohanty S, Manchanda V, Khuenger N, dkk. In vitro antimicrobial susceptibility patterns of Propionibacterium acnes isolated from patients with acne vulgaris. J Infect Dev Ctries. 2016;10(10):1140–5.
18. Süer K, Güvenir M. Propionibacterium acnes (Cutibacterium acnes) and acne vulgaris: The latest updates of antimicrobial activity. Turk J Dermatol. 2019;13:57-9.
19. Leyden JJ. A review of the use of combination therapies for the treatment of acne vulgaris. J Am Acad Dermatol. 2003;49:S200–10.
20. Zhang YQ, Ren SX, Li HL, Wang YX, Fu G et al. Genome-based analysis of virulence genes in a non-biofilm-forming Staphylococcus epidermidis strain (ATCC 12228). Mol Microbiol. 2003;49(6):1577-93.
21. Heilmann C, Schweitze O, Gerke C, Vanittanakom N, Mack D, Goetz F. Molecular basis of intercellular adhesion in the biofilm-forming Staphylococcus epidermidis. Mol Microbiol. 1996;20:1083-91.
22. Vuong C, Voyich JM, Fischer ER, et al. Polysaccharide intercellular adhesion (PIA) protects Staphylococcus epidermidis against major components of the human innate immune system. Cell Microbiol. 2004;6:269-75.
23. Fey PD, Olson ME. Current concepts in biofilm formation of Staphylococcus epidermidis. Future Microbiol. 2010;5:917-33.