Breast abscess in a case of duct atresia caused by CO_2-auxotrophic small colony variants of *Staphylococcus aureus*: Case report and review of literature

Sayan Bhattacharyya, Nitesh Kumar Jaiswal, Manoj Kumar[1], Mohammad Aftab Alam Ansari, Asim Sarfraz

Departments of Microbiology and [1]General Surgery, All India Institute of Medical Sciences, Patna, Bihar, India

Address for correspondence:
Dr. Sayan Bhattacharyya, Department of Microbiology, All India Institute of Medical Sciences, Patna - 801 505, Bihar, India. E-mail: sayantheboss@yahoo.co.in

Abstract

Small colony variants (SCV) are persistent, intracellular morphotypes of *Staphylococcus aureus* causing indolent, subclinical infections. They are usually auxotrophic for Thymidine, menadione or carbon dioxide. Diagnosis is often misleading due to small colony type, delayed coagulase activity and lack of hemolysis. We hereby describe a case of CO_2-auxotrophic SCV of *S. aureus* causing left breast abscess in a young female with duct atresia of breast, detected by increased colony size in CO_2 rich environment. SCVs can often be missed and have to be identified and treated properly.

Key words: Auxotrophism, small colony variants, *Staphylococcus aureus*

INTRODUCTION

Small colony variants (SCV) originate from wild-type *Staphylococcus aureus* by mutation in metabolic genes, leading to the emergence of auxotrophic bacterial subpopulations.[1] *S. aureus* SCV are characterized as electron transport deficient bacteria because of their auxotrophism to hemin or menadione or are recognized as thymidine-dependent.[2] These variants produce very small, mostly nonpigmented and nonhemolytic colonies.[2] Due to deficient components of electron transport chain, aminoglycoside antibiotics, cannot be transported across their cell membrane, thus explaining resistance to this class of antibiotics.[3] These variants are usually recovered from chronic, persistent and relapsing infections.[4] Diagnosis is very difficult due to atypical phenotypic features like lack of hemolysin and slow growth.[4] We here describe isolation of Carbon dioxide auxotrophic SCV of *S. aureus* in a case of breast abscess.

CASE REPORT

KD, a 23-year-old female patient, resident of Patna, Bihar, presented to the General Surgery Out-Patient Department of the Institute with chief complaints of pain and lump in lower part of left breast with purulent nipple discharge, since last 1-year. She also gave a history of consumption of multiple antibiotics over last 6 months, prescribed by local practitioners, but could not specify the antibiotics. On examination, the lower part of left breast was tender and showed black discoloration. Transmammary ultrasonography showed duct atresia of the left breast. Under ultrasonographic guidance, about 10 ml of pus was drained from her left breast. Sample was sent to the microbiology lab for microbiological evaluation. Gram-stain of the sample showed copious leucocytes and Gram-positive cocci arranged in clusters. Culture on 5% Sheep blood agar and MacConkey agar showed nonhemolytic, low convex, opaque, lactose-fermenting colonies of 0.1 mm diameter after 24 h of aerobic incubation. Gram-stain of the colonies showed Gram-positive cocci of variable size, in grape-like clusters. Catalase was positive using 3% H_2O_2. Slide coagulase test was positive but delayed, and came positive only after addition of excess pooled human plasma. Antibiotic susceptibility was carried out by Kirby-Bauer disk diffusion method as per Clinical and Laboratory Standards Institute protocol.[5] The following antibiotic discs were used: Cefotaxime (30 μg), Amoxiclav (30 μg), Clindamycin (2 μg), Erythromycin(15 μg), Cefotaxime (30 μg) and Levofloxacin (5 μg). The isolate was susceptible to all of the antibiotics tested. Simultaneously, colonies were subcultured on 1 Mueller-Hinton agar (MHA) plate and grown inside a glass

candle jar (desiccator) containing a burning candle, at 37°C. Furthermore, lawn culture of the isolate was performed on 1 MHA plate and a disc containing 15 μg Vitamin K (menadione) was placed on it. Next day, the plate placed in the candle jar grew large (1.5 mm diameter) colonies. There was no increase in size around Menadione (Vitamin K) disc. Thus, the isolate was auxotrophic for CO_2.

DISCUSSION

Small colony variants of *S. aureus* can persist inside host cells and modulate host defences.[6] They form small, colorless, nonhemolytic colonies after 48 h of incubation, are only slowly coagulase positive, fail to ferment mannitol, and can revert to the parental wild phenotype.[6] SCVs are characterized by a strong reduction in growth rate, an atypical colony morphology, and unusual biochemical characteristics, which causes them to be commonly undetected or misidentified by standard clinical microbiology procedures.[7] Mostly SCVs are auxotrophic for menadione or hemin, but rarely for CO_2.[8] These types of SCVs have been recovered from cases of endocarditis, wound infections, respiratory infections and others.[8] Although the prevalence of SCVs of *S. aureus* in clinical specimens in a general microbiology laboratory has been estimated to be around 1%, SCVs are recovered more frequently from certain groups of patients, such as those with cystic fibrosis.[7,8] They also have reduced α-toxin gene expression and hence cause less damage to host cells, thus producing subclinical, persistent infections.[9] CO_2 auxotrophic SCVs are characterized by fastidious growth characteristics and an atypical, small-colony morphology (nonpigmented and nonhemolytic colonies) that may be entirely overlooked in mixed cultures.[7] When they are recognized, SCVs may still be misidentified as coagulase-negative *Staphylococcus* spp., and susceptibility test results can be difficult to interpret.[6] To reduce the potential of missing CO_2 auxotrophic SCV isolates, it is important to observe the microbiological characteristics of colonies on plates incubated in a CO_2 rich atmosphere and to extend conventional cultures to at least 72 h.[6] As far as we know, this is the first case of abscess caused by CO_2 auxotrophic SCVs in a female patient from this region.

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How to cite this article: Bhattacharyya S, Jaiswal NK, Kumar M, Ansari MA, Sarfraz A. Breast abscess in a case of duct atresia caused by CO_2 -auxotrophic small colony variants of *Staphylococcus aureus*: Case report and review of literature. J Nat Sc Biol Med 2015;6:475-6.

Source of Support: Nil. **Conflict of Interest:** None declared.

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Quick Response Code:	Website: www.jnsbm.org
	DOI: 10.4103/0976-9668.160046