Occurrence of Staphylococcus Aureus in Food Stuffs

Sarvesh Gautam¹, Priya Srivastava², Dr. Anju Tyagi³
¹, ²Institute of Applied Medicines and Research, Ghaziabad
³Assistant Professor, Institute of Applied Medicines and Research, Ghaziabad

Abstract: Methicillin-resistant Staphylococcus aureus (MRSA) infection is one of the leading causes of hospital-acquired infections and is commonly associated with significant morbidity, mortality, length of stay, and cost burden. Although Staphylococcus is a common bacterium that resides on skin and in nasal passages, it can cause infections if it enters the body through a cut in the skin, and these infections can be serious. MRSA infections can occur among hospitalized patients who undergo surgery or who have suppressed immunity; however, they also increasingly occur among non-hospitalized patients who are otherwise healthy. Although identification and prevention techniques have improved, MRSA remains a major healthcare issue. MRSA bacteria can be challenging to manage, especially in patients at high risk of complications or in those with toxigenic or multidrug-resistant strains. Early identification of MRSA is an important step toward timely implementation of appropriate treatment. The development of new molecular and immune-chromatographic testing technologies has the potential to dramatically shorten delays to diagnosis and treatment. In addition, novel antibiotic therapies are becoming available to provide effective alternatives for strains that have acquired resistance to existing drugs.

Keywords: Staphylococcus aureus; Methicillin-resistant; infection; Antibiotics; Treatment

I. INTRODUCTION

Staphylococcus aureus is Gram-positive, spherical bacteria have a diameter of 1µm - 1.3µm. on microscopic examination, the organisms seem in clusters, similarly bunches of grapes. Toxins are produced by way of a few strains while growing in meals. These pollutants can cause gastrointestinal disorder normally called Staphylococcal food poisoning. The enterotoxin produced by using S. Aureus is a heat-solid protein that resists heating at one hundred oC for 30-70 min. S. aureus is also liable for food-borne infections [1]. Staphylococcal food poisoning is usually characterized by self-limiting gastrointestinal symptoms, but sometimes the infection can be more serious or indeed deadly [2]. S. aureus is ubiquitous within the environment and it is also one of the major causes of bovine mastitis [3]. In this manner, crude milk and crude milk dairy products may be contaminated with S. aureus, due to the shedding of large segments of the life form into milk [4, 5]. Besides, cheese-makers may carry enterotoxin-producing S. aureus in their noses or on their hands and the need of proper sterile measures during food preparing increments the probability of contamination with S. aureus, particularly in small-scale artisanal dairies [6]. Without a doubt, dairy items are among the foods most commonly involved in Staphylococcal food poisoning outbreaks [2, 7]. 23 different Staphylococcal Enterotoxins have been depicted and many S. aureus strains harbour more than one SEs gene. SEs can be separated into typical types (i.e. A to E) and new variants classified at present as SEs or SEs-like (SEls) based on their capability to cause emesis. SEs are synthesised when S. aureus cell density reaches 105 - 108 cfu g-1. However, all of these toxins are heat-stable and can therefore be still present in the food even when the microorganism is inactivate or the contamination level is reduced by processing [2]. Among S. aureus strains, those that are Methicillin-resistant (MRSA) have spread within the final decades as hospital-acquired pathogens (HA-MRSA) all through the world, causing serious life-threatening infection not responding to a lot of antimicrobial treatment. More recently, community-acquired (CA-MRSA) and livestock-associated (LA-MRSA) MRSA have too developed [8]. MRSA have been identified in different foods worldwide, and several food-borne MRSA outbreaks have been reported demonstrating the zoonotic risk of transmission to humans [9]. The screening of S. aureus isolates from food of animal origin is therefore essential to estimate the MRSA emergence and the related zoonotic hazard [8].

Methicillin-resistant Staphylococcus aureus has been recognized as major cause of healthcare-associated contaminations around the world. Methicillin-resistant Staphylococcus aureus strains emerge to have been transferred from health care settings into the community and have developed as particularly associated with community-associated diseases in people [10]. Moreover, in recent year, Methicillin-resistant Staphylococcus aureus has been distinguished as an emerging pathogen in livestock and companion animals, as well as a few other farm animal species [11].
In addition, S. aureus is considered one of the most important contributory infective agent of mastitis in dairy cattle [12, 13]. Generally, methicillin is not utilized for treatment of mastitis but MRSA has been recovered from dairy cattle and it has been separated from milk. Additionally the presence of MRSA within the environment might too be one of the sources of MRSA contamination in creatures since it may survive for a few months [14]. This study was conducted to review the prevalence of S. aureus enterotoxins in food, milk and dairy foodstuffs.

II. REVIEW OF LITERATURE

A. General Description
Staphylococcus aureus may be Gram-positive microbes, broadly predominant in nature. It can be separated from many environmental sites (e.g., clean, water, discuss, defecation). Approximately 20 to 60% of people are found permanent or intermittent carriers of S. aureus and related sites include the front nacres, maxillae, perineum and vagina [15, 16]. S. aureus is also an essential food-borne pathogen. Staphylococcal food poisoning is cause by drinking of food containing one or more preform enterotoxins (SEs) delivered by S. aureus. Staphylococcal food poisoning positions third among reported food-borne infections within the world [17]. In 2006, S. aureus toxins were responsible for 49% of 482 human food-borne outbreaks caused by bacterial toxins and 4% of all reported outbreaks reported by EU Member States (EFSA, 2007). Side effects have a fast onset and include nausea, vomiting and diarrhoea [18].Staphylococcus aureus Food Poisoning is a common cause of food-borne infection around the world. Of specific relevance is the capacity of a few Staphylococcus aureus strains to create heat stable enterotoxins that cause Staphylococcal food poisoning, which positions one of the mainly predominant around the world causes of gastroenteritis [19]. The significance of the enterotoxins comes due to their heat stability and their resistance to inactivation by gastrointestinal proteases like pepsin. Although Staphylococcus can be killed at standard cooking temperature, the toxins continue active [20].

B. Historical Background Of MRSA
Alexander Fleming conducted a research and reported the bactericidal impacts of a fungal contaminant that produced penicillin against S. aureus developing on culture plate [21]. A mass manufacture of the drug from vats of corn steep fluid developing on the mold was preceded due to the high mortalities in World War II [22]. Hence, there was a sensational drop in death rates from bacterial pneumonia and meningitis in World War II compare to World War I. This led to the improvement of penicillin as the primary key driver in select for resistant S. aureus. In 1940, active β- lactam ring enzyme was described in Escherichia coli that are capable of hydrolyzing the penicillin. This enzyme was named penicillinase [23] whereas in 1944; penicillinase was also revealed in the S. aureus [24]. In 1948, it was observed that over 50% of staphylococcal recovered from patients in a hospital were resistant to penicillin [25]. Since at that point to date, 90 to 95% of S. aureus strains around the world is penicillin safe, with the plasmid encoded penicillinase readily transferable through transduction or conjugation. A penicillinase-resistance penicillin recognized as methicillin was presented in 1959 to conflict penicillin-resistant S. aureus, but inside a year, Professor Patricia Jevons report the earliest human S. aureus strain methicillin safe in United Kingdom hospital [26]. In year 1968, United States record the primary epidemic of Methicillin-resistant Staphylococcus aureus [27] whereas within the 1970s, S. aureus strains contain safe to most penicillinase-stable penicillins. It was first unspecified to be an infection of human origin until MRSA was first isolated in 1972 in a mastitic bovine. After that, report of MRSA disease become establish in domestic and wild animals [28].

C. Colonization
S. aureus procured scince an external source could be the cause of a contamination when inoculate into an open enjury. More frequently, the human have is infected by microbes that colonized her or his skins or mucosal surface [29]. The mucosal surface so as to harbor S. aureus contains nose, throat, vaginal divider, and gastrointestinal tract. Nasal carriage is likely most critical since nose-picking may successfully spread the bacteria to other body surface and other hosts. Remarkably, 20% of people are persistently colonized within the nose and 30% are briefly colonized. The explanation of persistent and transient carriage varies according to the study, for the most part is depicted as a single positive culture on a nasal swab (transient) versus at least two continuous positive culture one week apart (persistent). Colonization is also more frequent among younger children, and patients with HIV and diabetes [29]. Though colonization inclines an individual to S. aureus contamination, one study shows that following nosocomial infection, colonized people have less serious S. aureus disease compare to non-colonized individuals [29], whether colonization may bring on low-level adaptive resistance so that consequent infections become milder. In support of this view, a study showed that carriage of S. aureus harboring the Toxic Shock Syndrome Toxin (TSST) is related with manufacture and preservation of antibody to the toxin.
[30]. Alternately, most persons who obtain Staphylococcus toxic shock syndrome do not have antibody to Toxic Shock Syndrome Toxin (TSST).

Once colonization is recognized, *S. aureus* is located in close to the, ears, mouth, throat and sinus; yet, shockingly nasal carriage infrequently leads to overt contamination of these sites. Studies of *S. aureus* regulation recommend that during colonization, many *S. aureus* virulence genes may be down regulated [31]. Among the genes that control *S. aureus* colonization and virulence, the best identified global regulator is the accessory gene regulator *agr* which has been describe in details in many excellent reviews [31]. Briefly, *agr* is a majority detecting locus, which directly controls expression of a number of virulence and colonizing factors. Down regulation of *agr* is related by colonization and activation of *agr* with host attack.

**D. Pathogenesis**

*S. aureus* are one the most common bacterial infection in humans and are the causative agents of multiple human infections, including bacteremia, infective endocarditis, skins and soft tissue infections (e.g., impetigo, folliculitis, furuncles, carbuncles, cellulitis, scalded skin syndrome, and others), osteomyelitis, septic arthritis, prosthetic device infections, pulmonary infections (e.g., pneumonia and empyema), gastroenteritis, meningitis, toxic shock syndrome, and urinary tract infections [32]. Depending on the strains involved and the site of infection, these bacteria can cause invasive infections and/or toxin-mediated diseases [33]. The pathophysiology varies greatly depending on the type of *S. aureus* infection [32]. Mechanisms for evasion of the host immune response include the production of an antiphagocytic capsule, sequestering of host antibodies or antigen masking by Protein A, biofilm formation, intracellular survival, and blocking chemotaxis of leukocytes [34]. Endocarditic, sepsis, as well as toxic shock syndrome [35].Binding of the micro organism to extracellular matrix proteins and fibronecin in infectious endocarditis is mediated by way of bacterial cellular wall-related proteins together with fibrinogen-binding proteins, clumping elements, and teichoic acids [33]. Also, Staphylococcal superantigens (TSST-1 or poisonous shock syndrome toxin 1) are essential virulence elements in infectious endocarditic, sepsis, in addition to poisonous shock syndrome [35]. Pneumonia infections are related to the bacterial production of PVL (Panton-Valentine leukocidin), Protein A, and alpha-hemolysin, and infections are extra not unusual following influenza virus contamination in addition to a prognosis of Cystic Fibrosis. Prosthetic tool infections are often mediated with the aid of the capability of *S. Aureus* strains to form biofilms in addition to speak the usage of quorum sensing in a bacterial cellular density-established manner [36].

**III. EPIDEMIOLOGY OF MRSA IN INDIA**

There has been a sluggish increase in the number of reports describing MRSA in India over years (Center for Disease Dynamics, Economics & Policy (CDDEP). Varied costs of MRSA infection were reported, from rural to urban, from paediatric to geriatric, and from easy pores and skin infections to critical sepsis instances. Several studies have mentioned the prevalence of MRSA in healthcare employees. The MRSA carriage fee amongst healthcare employees in hospitals, especially amongst individuals who were worried in vital care areas, became reported to be from 10 to 14.3% [37]. The isolation rate for MRSA from outpatients ward inpatients and intensive care units (ICUs) were 27%, 49% and 47%, respectively, in 2009. The majority of *S. Aureus* isolates were obtained from patients with SSTIs, observed through the ones affected by bloodstream infections and respiration infections [38].

According to a current examine, the frequency of 45% of *S. Aureus* clinical isolates being methicillin-resistant in India within the early 2010s is similar to what has been mentioned in the rest of the Asian countries (forty one.9% in Pakistan, forty five.8% in China, 41% in Japan, 35.3% in Singapore and 55.9% in Taiwan), except Hong Kong, Indonesia (28% every) and South Korea (>70%) [39]. Similar to global traits, but, MRSA in India has been located to be of geographically various origins [40].

Initial research of medical samples from hospitals showed that the incidence of HA-MRSA changed into as low as 6.9% in 1988 [41], however improved to 27%, 32%, 42.5% and 47% in Mumbai, Vellore, Delhi and Bengaluru, respectively, via the overdue 1990s [42]. Infections with CA-MRSA have been reported in school children and in healthful individuals both from rural and urban regions [43].

**IV. CONCLUSION**

Foodborne pathogens like toxigenic *S. Aureus* can without problems be transmitted via the food made from raw milk under not noted hygienic situations. Animal-related strains of MRSA isolation from specific dairy products like raw milk or a raw-milk cheese has said global. This observe find out the status of the MRSA present in milk and dairy products to be had in Northern India. Considering the final results of this examine a control strategy can be developed to save you and control the harmful results of MRSA on public health in factor of our country. In conclusion, our findings discovered that the milk and dairy product sold in and around Ghaziabaad city are contaminated with multidrug resistant *S. Aureus* which are very alarming for both human and animal fitness.
REFERENCES

[1] Garcia-Alvarez, L., M. T. Holden, H. Lindsay, C. R. Webb, D. F. Brown, M. D. Curran (2011) Methicillin-resistant Staphylococcus aureus with a novel mecA homologue in human and bovine populations in the UK and Denmark: a descriptive study. Lancet Infect. 11(5): 595-603.

[2] Benkerroum N. 2017 Staphylococcal enterotoxins and enterotoxin-like toxins with special reference to dairy products: An overview. Critical Reviews in Food Science and Nutrition 0(0):1-28. DOI: 10.1080/10408398.2017.1289149.

[3] Boss R., Cosandeay A., Luini M., Artursson K., Bardiau M., Breitenwieser F. and Graber H.U. 2016. Bovine Staphylococcus aureus: Subtyping, evolution, and zoonotic transfer. Journal of Dairy Science, 99(1):515–528. DOI: 10.3168/jds.2015-9589.

[4] D’amico D.J. and Donnelly C.W. 2011. Characterization of Staphylococcus aureus strains isolated from raw milk utilized in small-scale artisan cheese production. Journal of Food Protection 74(8):1353-1358. DOI: 10.4315/0362-028X.JFP-10-533.

[5] Rola J.G., Czubowska A., Korpysa-Dzibra W. and Osek J. 2016. Occurrence of Staphylococcus aureus on Farms with Small Scale Production of Raw Milk Cheeses in Poland. Toxins 8(3). DOI: 10.3390/toxins8030062.

[6] André M.C.D.P.B., Campos M.R.H., Borges L.J., Kipnis A., Pimenta F.C. and Serafini Á.B. 2008. Comparison of Staphylococcus aureus isolates from food handlers, raw bovine milk and Minas Frescal cheese by antibiogram and pulsed-field gel electrophoresis following Smal digestion. Food Control 19(2):200-207. DOI: 10.1016/j.foodcont.2007.03.010.

[7] De Buyser M.-L., Dufour B., Maire M. and Lafarge V. 2001. Implication of milk and milk products in bovine-borne diseases in France and in different industrialised countries. International Journal of Food Microbiology 67(1-2):1-17. DOI: 10.1016/S0168-1605(01)00443-3.

[8] Bardiau M., Yamazaki K., Duprez J.-N., Taminiau B., Mainil J. G. and Ote I. 2013. Genotypic and phenotypic characterization of methicillin-resistant Staphylococcus aureus (MRSA) isolated from milk of bovine mastitis. Letters in Applied Microbiology 57(3):181–186. DOI: 10.1111/lam.12099.

[9] Doulgeraki A.I., Di Ciccio P., Ianieri A. and Nychas G.-J.E. 2017. Methicillin-resistant food-related Staphylococcus aureus: a review of current knowledge and biofilm formation for future studies and applications. Research in Microbiology 168(1):1-15. DOI: 10.1016/j.resmic.2016.08.001.

[10] Scientific Report of EFSAC and ECDC EU Summary Report on antimicrobial resistance in zoonotic and indicator bacteria from humans, animals and food in 2013 EFSA J., 13 (2015), p. 406 Google Scholar

[11] E. Antoci, M.R. Pinzone, G. Nunnari, S. Stefani, B. CacopardoPrevalence and molecular characteristics of methicillin-resistant Staphylococcus aureus (MRSA) among subjects working on bovine dairy farms Infez. Med., 21 (2013), pp. 125-129 View Record in Scopus Google Scholar

[12] Moon, A.R. Lee, H.M. Kang, E.S. Lee, M.N. Kim, Y.H. Paik, Y.H.Park, Y.S. Joo, H.C. KooPhenotypic and genetic antibiogram of methicillin-resistant staphylococci isolated from bovine mastitis in Korea J. Dairy Sci., 90 (2007), pp. 1176-1185

[13] H.M. Nam, A.R. Lee, S.C. Jung, M.N. Kim, G.C. Jung, S.H. Wee, S.K. LimAntimicrobial susceptibility of Staphylococcus aureus and characterization of methicillin-resistant Staphylococcus aureus isolated from bovine mastitis in Korea Foodborne Pathog. Dis., 8 (2011), pp. 231-238 CrossRefView Record in ScopusGoogle Scholar

[14] S.K. Lim, H.M. Nam, G.C. Jung, H.S. Lee, S.C. Jung, T.S. KimTransmission and persistence of methicillin-resistant Staphylococcus aureus in milk, environment, and workers in dairy farms Foodborne Pathog. Dis., 10 (2013), pp. 731-736 CrossRefView Record in ScopusGoogle Scholar

[15] Zaidi, A.K. and Syed, A. (2010) Methicillin-resistant Staphylococcus aureus in human and animal isolates. J. Food Microbiol. 107:192–201.

[16] Lombard L., Bohach GA (1997) Staphylococcus aureus. In: (eds.: Doyle MP, Beuchat LR, Montville TD) Food Microbiology: fundamentals and frontiers. ASM Press, Washington, D.C., USA, pp. 353-375.

[17] Abrahim EP, Chain E (1940). An enzyme from bacteria able to destroy penicillin. Nature. 146: 837-842.

[18] Kirby WMM (1944). Extraction of a highly potent penicillin inactivator from penicillin resistant staphylococci. Science. 99(2579): 452-453. http://dx.doi.org/10.1016/j.sciences.2011.01.022

[19] Palavecino E (2004). Community-acquired methicillin-resistant Staphylococcus aureus infections. Clin Lab Med 24(2): 403-418. http://dx.doi. org/10.1016/j.cll.2004.03.007 PMid:15177847

[20] Woddy SE, Kauffman UK, Smith TC (2012). Methicillin-resistant Staphylococcus aureus in Central Iowa Wildlife. J Wildl Dis: 48(4): 1069-1073. http:// dx.doi.org/10.7589/2011-10-295 PMid:23060511

[21] Wertheim HF, Vos MC, Otto A, van Belkum A, Meester MH, Verbrugh HA. Risk and outcome of nosocomial Staphylococcus aureus bacteremia in nasal carriers versus non-carriers. Lancet. 2004;364:703–705. [PubMed] [Google Scholar]

[22] Ritz HL, Kirkland JJ, Bond GG, Warner EK, Petty GP. Association of high levels of serum antibody to staphylococcal toxic shock antigen with nasal carriage of toxic shock antigen-producing strains of Staphylococcus aureus. Infect Immun. 1984;43:954–958. [PMC free article] [PubMed] [Google Scholar]

[23] Novick RP. Autoinduction and signal transduction in the regulation of staphylococcal virulence. Mol Microbiol. 2003;48:1429–1449. [PubMed] [Google Scholar]

[24] Tong SY, Davis JS, Eichenberger E, Holland TL, Fowler VG. Staphylococcus aureus infections: epidemiology, pathophysiology, clinical manifestations, and management. Clin. Microbiol. Rev. 2015 Jul;28(3):603-61. [PMC free article] [PubMed]
[33] DeLeo FR, Diep BA, Otto M. Host defense and pathogenesis in Staphylococcus aureus infections. Infect. Dis. Clin. North Am. 2009 Mar;23(1):17-34. [PMC free article] [PubMed]

[34] Foster TJ. Immune evasion by staphylococci. Nat. Rev. Microbiol. 2005 Dec;3(12):948-58. [PubMed]

[35] Salgado-Pabón W, Breshears L, Spaulding AR, Merriman JA, Stach CS, Horsswill AR, Peterson ML, Schlievert PM. Superantigens are critical for Staphylococcus aureus Infective endocarditis, sepsis, and acute kidney injury. MBio. 2013 Aug 20;4(4) [PMC free article] [PubMed]

[36] Le KY, Otto M. Quorum-sensing regulation in staphylococci—an overview. Front Microbiol. 2015;6:1174. [PMC free article] [PubMed]

[37] Radhakrishna M, D’souza M, Kotigadde S, Saralaya KV, Kotian MS. Prevalence of methicillin resistant Staphylococcus aureus carriage amongst health care workers of critical care units in Kasturba Medical College, Hospital, Mangalore, India. J Clin Diagn Res 2013;7:2697–700.

[38] Indian Network for Surveillance of Antimicrobial Resistance (INSAR) group, India. Methicillin resistant Staphylococcus aureus (MRSA) in India: prevalence and susceptibility. Indian J Med Res 2013;137:363–9.

[39] Chen CJ, Huang YV. New epidemiology of Staphylococcus aureus in Asia. Clin Microbiol Infect 2014;20:605–23.

[40] Verma S, Joshi S, Chitnis V, Hemwani N, Chitnis D. Growing problem of methicillin resistant staphylococci—Indian scenario. Indian J Med Sci 2000;54:535–40

[41] Pal N, Ayyagari A. Drug resistance pattern of methicillin resistant Staphylococcus aureus. Indian Pediatr 1991;28:725–9.

[42] Chatterjee SS, Ray P, Agarwal A, Das A, Sharma M. A community-based study on nasal carriage of Staphylococcus aureus. Indian J Med Res 2009;130:742–8

[43] Fomda BA, Thokar MA, Khan A, Bhat JA, Zahoor D, Bashir G, et al. Nasal carriage of methicillin-resistant Staphylococcus aureus among healthy population of Kashmir, India. Indian J Med Microbiol 2014;32:39–43