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The antimicrobial susceptibility and contribution of *Staphylococcus aureus* to surgical site infections in patients hospitalized in the West Pomeranian region (Poland) during the COVID-19 pandemic period – a 3-year follow-up

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ABSTRACT

Introduction: Surgical site infections (SSIs) are at the forefront of healthcare-associated infections and the second most common cause of hospital readmission. The etiology of these infections is generally monobacterial with a predominance of *Staphylococcus aureus*. Although it is a preventable infection it significantly increases the cost of hospitalization and doubles the mortality rate.

The aim of the present study was to evaluate the prevalence of SSIs and antimicrobial susceptibility of *S. aureus* isolated from SSIs in patients hospitalized at the Clinical Hospital No. 1 of the Pomeranian Medical University in Szczecin (Poland) – CH-1 – during the period 2019–2021, in the course of the COVID-19 pandemic.

Materials and methods: Analysed specimens were collected from patients with diagnosed skin and soft tissue infections (SSTIs) caused by *S. aureus*, collected in 2019–2021, and then examined during routine microbiological diagnostics. The collection included specimens from patients hospitalized at CH-1, as well as consulted at the Emergency Department (ED) of CH-1, patients from healthcare facility of the Ministry of Internal Affairs and Administration in Szczecin, and from detainees of the Szczecin Detention Centre (Poland). Out of the total of 1140 results, 232 were classified as SSIs caused by *S. aureus* and then analysed. All the data were systemically entered into a spreadsheet (Excel 2019) and later subjected to several statistical tests (using StatSoft Statistica 13 package).

Results: From the collection of results, 20.4% (232/1140) were considered as SSIs of *S. aureus* etiology. In the following years 2019, 2020, and 2021, the SSIs of *S. aureus* etiology were 17.1% (72/421), 19.5% (66/339), and 24.7% (94/380), respectively. Methicillin-resistant *S. aureus* (MRSA) was the cause of 7% of analysed SSIs. The highest incidence of SSIs was observed mainly in patients admitted to the trauma and orthopaedic wards and in patients seen in the ED. The majority of SSIs were mono-etiologic, caused by *S. aureus* only (93%). Mixed etiology was determined in 7% of SSIs with a significant share of beta hemolytic streptococci, enterobacteria, and non-fermentative Gram-negative rods. A slight increase in the prevalence of SSIs was observed during the analysed period. The overall antimicrobial susceptibility of all examined strains was noted, with a low rate of MRSA. The obtained results demonstrate the good practice of both strategies of hospital infection control, as well as the coherent and rational antibiotic policy in the CH-1 in the West Pomeranian region in Poland. The 2017–2018 data on the incidence of SSTIs and the percentage of SSIs among patients hospitalized in CH-1 show that in 2017, SSIs represented 17.9% of all cases classified as SSTIs (57/318), followed by SSIs in 2018, which represented 19.5% of all SSTIs analysed in CH-1 (68/348). The percentage of SSIs among the total SSTIs confirmed during the 2-year pre-pandemic period did not indicate significant changes in the number of SSIs among CH-1 patients hospitalized during the period of analysis. Therefore, it can be concluded that pandemic restrictions did not significantly affect the trend in the predominant proportion of *S. aureus* among SSIs during the study period.

Keywords: skin and soft tissue infections; methicillin-resistant *S. aureus* (MRSA); osteomyelitis; hospital-acquired infections.

INTRODUCTION

Staphylococcus aureus is one of the most important human pathogens and the main etiological factor of skin and soft tissue infections (SSTIs), ostitis and osteomyelitis, necrotizing pneumonia, and infective endocarditis. *Staphylococcus aureus* is part of the human skin and mucous membranes and contributes to a carrier state in the nasal vestibule and pharynx. It has been confirmed that approx. 30% of the healthy human population is asymptomatically colonized by *S. aureus* in the prenares [1], while the percentage of *S. aureus* carriers among hospitalized patients and healthcare workers is much higher, reaching 50% [2].

Over the past 2 decades, a significant increase in the number of infections caused by *S. aureus* has been observed, especially among hospitalized patients [3]. However, data available up to 2013 show that the annual prevalence of *S. aureus* infections

in the ambulatory population began to increase [4]. It is worth noting that worldwide, methicillin-resistant *S. aureus* (MRSA) infections in hospitalized patients account for up to half of all *S. aureus* infections, and in Europe, the overall percentage of infections caused by MRSA is also increasing [5]. In addition, MRSA infections in outpatients, presumably associated with the hospital environment, are being reported with increasing frequency. This indicates an alarming trend of transmission of hospital-associated strains into the community [6, 7].

The prevalence of infections with *S. aureus* etiology in the community population is primarily related to the widespread carriage of this microorganism in the nasal vestibule and on the skin, which predisposes to endogenous infections through auto-transmission [8]. In contrast, in the hospital setting, immobilized patients following severe trauma or surgery are at increased risk of *S. aureus* colonization and infection because they often require prolonged hospital stays and are routinely transferred between hospitals and rehabilitation centers, facilitating the spread of hospital pathogens [9].

Skin and soft tissue infections caused by *S. aureus* affect patients of all ages and are characterized by a range of clinical manifestations, from superficial and self-limiting to severe and life-threatening infections [10]. Skin and mucous membranes are the first lines of defense against external agents, including microorganisms, and any breakdown in their integrity predisposes to systemic infection. Nevertheless, the host body has developed mechanisms to prevent the further spread of pathogens, including the components of the innate immune system [11]; therefore, clinical isolates of *S. aureus* are mainly associated with opportunistic infections, the clinical picture of which is related to the relationship between the virulence factors of the pathogen and the immune status of the patient. Numerous toxins and enzymes allow this bacterium for more effective colonization, evasion of the host immune system, invasion and significantly influence the clinical manifestation of infections.

Staphylococcus aureus is characterized by variable virulence, which is related to the expression of virulence factors that play an important role in the invasion of the host organism as well as specific clinical manifestations [12, 13]. Primarily purulent SSTIs can ultimately lead to the spread of the microorganism via the blood, posing an immediate threat to the patient's life. It is estimated that hematogenous spread of *S. aureus* is associated with a 20% increased risk of fatal outcomes [14].

Skin and soft tissue infections caused by *S. aureus* are among the most common infections reported in outpatients worldwide. Skin and soft tissue infections affect the epidermis, dermis, subcutaneous tissue, and fascia and are classified as superficial, often mild, and self-limiting infections, or as systemic and life-threatening infections requiring the full resources of modern medicine. The main risk factors for the development of SSTIs include previous contact with the hospital environment, open wounds, diabetes, underlying health conditions, and congenital and acquired immunodeficiencies. Both types of SSTIs – complicated and uncomplicated – usually require prolonged and targeted antibiotic therapy [10, 15, 16].

Skin and soft tissue infections are also divided into primary and secondary infections. A primary skin infection is not preceded by clinically apparent skin lesions and includes impetigo, folliculitis, boils, and primary abscesses. Primary infections often occur in patients without comorbidities. In contrast, secondary skin infections are associated with pre-existing skin lesions and their disruption of integrity and often occur as a result of medical procedures in immunosuppressed patients. The latter include secondary wound infections (including surgical site infections – SSIs). However, the division of SSTIs into primary and secondary is not strict and has a theoretical form [17, 18].

Uncomplicated SSTIs account for a large number of patients seeking medical care each year. The number of such visits in the USA reaches more than 14 mil per year [19, 20]. The challenge of developing effective therapy for recurrent SSTIs has prompted the search for alternative treatments. Clinical trials dating back to the early 20th century and the use of autovaccines are strategies with potential efficacy for the control of cutaneous infections caused by *S. aureus* [21, 22, 23].

Surgical site infections are a potential complication following any surgical procedure, and despite advanced surgical techniques, are among the most common infections worldwide [24]. Surgical site infections occur in approx. 1–5% of patients undergoing so-called clean surgery, while the incidence of SSIs following colon or genitourinary surgery can reach 30% [25].

Classification of postoperative wounds in terms of potential wound contamination allows the risk of infection to be assessed and a good treatment strategy to be planned in advance. Several factors allow to estimate the risk of postoperative complications, including the mechanism of injury and the operated field, which significantly influence the assessment of the risk of infection [26]. A superficial SSI must meet at least one of the following criteria: purulent discharge from the incision site, obvious signs of inflammation, and microorganisms cultured from an aseptically collected specimen. Deep SSI, on the other hand, may be found even a year after surgery and may involve abscess formation in addition to purulent discharge from the incision site, confirmed by reoperation, histopathology, or radiology. Another situation classified as SSI is an infection of an organ or surgical space and has also been diagnosed using the same criteria as for deep infections [27].

Patients may develop SSI as a result of infection by their microbiota or caused by highly resistant nosocomial pathogens from external sources, and in either case, treatment may be complicated by the risk of dissemination of infection [28]. Notably, the risk of SSI can be significantly reduced with appropriate antibiotic prophylaxis [29]. Patients undergoing clean surgery are often asymptomatic carriers of *S. aureus* but pre-screening procedures for all patients are not well defined [30]. Screening for *S. aureus* carrier status is routinely performed before open heart surgery and hip replacement and is not mandatory for other procedures [31]. Recommendations emphasize the need to decolonize patients at risk for serious *S. aureus* infection and those undergoing surgical procedures at risk of infection only if carrier status is documented [32]. Consequently, many patients undergoing procedures other than cardiac and orthopedic surgery who are asymptomatically colonized by *S. aureus* become infected during their hospital stay and the wound healing

process and are also a source of the microorganism infecting other patients. Nasal carriage of *S. aureus* has been shown to be an independent risk factor for SSIs, yet eradication protocols are limited to the use of intranasal mupirocin [33, 34].

It is estimated that approx. 7% of surgical procedures in cardiac, orthopedic, and trauma units are associated with SSIs [35]. The indications for antibiotic SSI prophylaxis are a narrow catalog of procedures, including open heart surgery, but also orthopedic procedures such as joint replacement or open fracture surgery. The antibiotics administered take into account the most likely etiologic factors that pose the risk of infection. It is recommended to use β-lactam antibiotics from the cephalosporin group and, in the case of mucosal access, glycopeptides and metronidazole, especially for colorectal surgery. Antibiotic prophylaxis for surgical procedures involving tissues or systems other than the gastrointestinal, genital, and urinary tracts should include *S. aureus* as the most likely etiology of a potential SSI [36].

Since the 1990s, MRSA has been most commonly associated with previously hospitalized individuals (previous antibiotic therapy and prolonged hospitalization), whereas methicillin-sensitive *Staphylococcus aureus* (MSSA) isolates are no longer exclusively community-associated pathogens. The emergence of MRSA and MSSA strains in both healthcare and community human populations is associated with the asymptomatic carriage of this bacterium even in healthy individuals [37].

MATERIALS AND METHODS

The aim of the present study was to determine the prevalence of SSIs caused by *S. aureus* and the antimicrobial susceptibility in patients hospitalized and consulted at the Clinical Hospital No. 1 of the Pomeranian Medical University in Szczecin in Poland (CH-1), collected during the years 2019–2021. From a total of 1140 microbiological results of clinical specimens from patients diagnosed with SSTIs caused by *S. aureus*, 232 results were classified as SSIs and then analysed. The overall percentage of SSIs among all SSTIs and the percentage of SSIs among SSTIs in each year were determined. The percentage of SSI cases on individual CH-1 wards and remaining centers and the overall proportion of MRSA in staphylococcal SSIs were also described. The percentage of polyetiologic SSIs, the microorganisms involved, and the resistance phenotypes of identified MRSA were analysed.

All analysed results were derived from clinical specimens collected and analysed for routine microbiologic diagnostic purposes. Clinical specimens sent for microbiological analysis were described according to the terms available in the system, which were then automatically applied when the clinical specimens were registered in the database, and were defined as follows: abscess, furunculus, paronychia, diabetic foot, decubitus, stump, wound or wound aspirate swab, and fistula swab. The number of clinical specimens collected from each patient is determined by the physician; however, the analysis included 1 specimen from each patient because the determination of *S. aureus* presence is performed from specimens cultured under aerobic conditions.

During routine microbiologic diagnostics, all clinical specimens were cultured on Columbia Agar with 5% sheep blood (bioMerieux, France) and Mannitol-Salt Agar (MSA) (bioMerieux, France) for isolation and identification of *S. aureus* and incubated for 24 h at 37°C under aerobic conditions. After the incubation period, presumptive identification of microorganisms was performed based on colony morphology and type of hemolysis. *Staphylococcus aureus* species identification was performed based on its ability to coagulate rabbit plasma and biochemical properties (VITEK system, bioMerieux). The presence of *S. aureus* on MSA was determined based on the positive reaction, described as the presence of yellow colonies surrounded by yellow medium.

The data analysed in this retrospective study were obtained from the database archive of the CH-1 microbiology laboratory. The data were used for research purposes in a non-identifiable manner. The study included results from patients hospitalized in CH-1, consulted in the Emergency Department (ED), patients from healthcare facilities of the Ministry of Internal Affairs and Administration in Szczecin, and detainees in the Detention Centre in Szczecin, Poland, between 2019–2021. Clinical assessment of SSTIs was performed by the subjects who commissioned the analysis. All data were systematically entered into the spreadsheet (Excel 2019) and later subjected to several statistical tests (using the StatSoft Statistica 13 package).

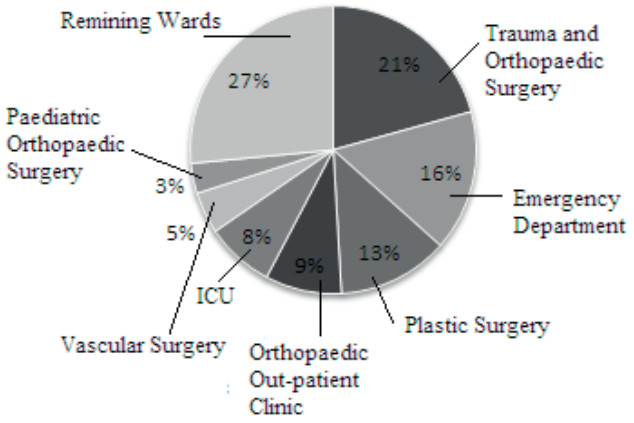
The antimicrobial susceptibility of all the strains studied was determined by the disc diffusion method on Mueller–Hinton agar plates (bioMerieux) with following antibiotic paper discs – disc content: cefoxitin 30 μg, erythromycin 15 μg, clindamycin 2 μg, mupirocin 10 μg, gentamicin 10 μg, amikacin 10 μg, tetracycline 30 μg, chloramphenicol 30 μg, trimethoprim/sulfamethoxazole (cotrimoxazole) 1.25/23.75 μg (Becton Dickinson, USA). Testing was performed and interpreted according to the guidelines of the European Committee for Antimicrobial Susceptibility Testing (EUCAST). Data were analysed using the ProMic system (BioMerieux, Poland), a comprehensive system for analysing research results, which provides a selective report based on specified criteria. The analysis included year of isolation, type of infection, organisms identified, ordering department, and *S. aureus* drug resistance; data were generated excluding patient-identifiable data.

RESULTS

During the 3 years of the study, 1140 results analyzed were classified as SSTIs, of which 232, i.e. 20.4% (232/1140), were considered SSIs. The proportion of *S. aureus*-associated SSIs among all SSTIs increased slightly in each of the 3 years of analysis, reaching slightly 17.1% (72/421), 19.5% (66/339), and 24.7% (94/380) in 2019, 2020, and 2021, respectively. Antimicrobial susceptibility testing revealed methicillin resistance in 7% of SSIs caused by *S. aureus.* Ninety-four percent of all SSIs analyzed were monoetiologic and caused exclusively by *S. aureus.* The remaining 8% of SSIs involved also other microorganisms and were described as polyetiologic. The organisms

isolated simultaneously with *S. aureus* and classified as the etiology of the analysed SSIs were, in order of most frequent presence: β-hemolytic streptococci, *Pseudomonas aeruginosa*, *Proteus mirabilis*, *E. coli*, *Enterobacter* sp., *Klebsiella* sp., *Acinetobacter sp.*, *Citrobacter* sp., *Serratia* sp., *Candida* sp., and *Morganella morganii*.

20.7% of all SSIs were confirmed in patients admitted to the Trauma and Orthopaedic Ward, 15.9% in persons consulted in the ED, 12.5% in patients admitted to the Plastic Surgery Ward, 8.6% in patients consulted in the Orthopedic Outpatient Clinic, 7.7% in the Vascular Surgery Ward, 4.7% in the Intensive Care Unit, and 3.4% in the Paediatric Orthopedic Ward. The remaining 26.5% of SSTIs were almost equally distributed among all other wards and represented 1–5 SSIs. The percentage of SSTIs in the described units and the remaining wards included in the present study is shown in Figure 1. The percentage of clinical specimens classified as SSTIs recovered in each ward in each year of analysis is shown in Figure 2.

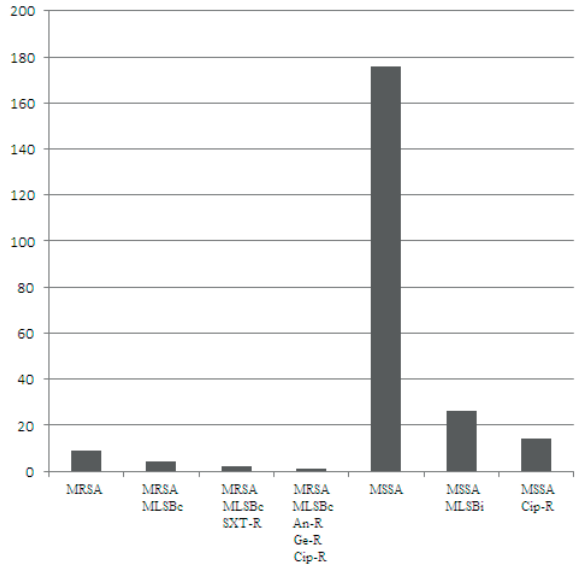


ICU – Intensive Care Unit

FIGURE 1. Percentage of surgical site infections in specific departments out of the total number of skin and soft tissue infections identified during the years of analysis

Methicillin resistance was confirmed in 6.9% (16/232) of the SSIs analyzed. Among MRSA strains, ¼ (4/16) had concomitant constitutive type of macrolides, lincosamides, streptogramins B (cMLSB) resistance phenotype, 2 strains had cotrimoxazole resistance in addition to MRSA and cMLSB phenotypes, and 1 strain had MRSA and cMLSB, ciprofloxacin, gentamicin, and amikacin resistance.

Among methicillin-susceptible strains (216/232), the inducible macrolide–lincosamide–streptogramin B (MLSB) phenotype was confirmed in 12% (26/216) of isolates and ciprofloxacin resistance in 6.5% (14/216) of MSSA. The absence of resistance to all other antimicrobials tested was confirmed. The overall good antimicrobial susceptibility and the low proportion of MRSA in the etiology of SSIs were noted. The distribution of resistance phenotypes and the proportion of MSSA in the etiology of SSIs is presented in Figure 3.



MRSA – methicillin-resistant *S. aureus*; MSSA – methicillin-susceptible *S. aureus*; MLSBc – constitutive type of macrolides, lincosamides, streptogramins B resistance; MLSBi – inducible type of macrolides, lincosamides, streptogramins B resistance; SXT-R – cotrimoxazole resistance; An-R – amikacin resistance; Ge-R – gentamicin resistance; Cip-R – ciprofloxacin resistance

FIGURE 3. Distribution of resistance phenotypes and methicillin-susceptible *Staphylococcus aureus* contribution in surgical site infections etiology – number of cases

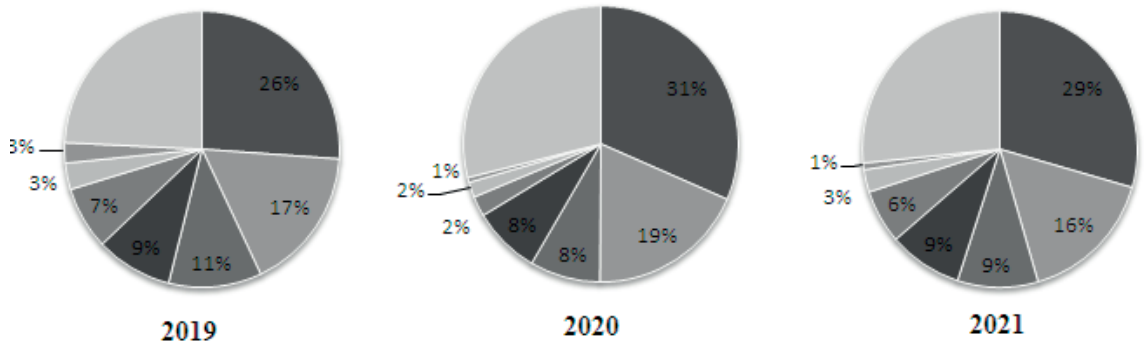


FIGURE 2. The percentage of clinical materials classified as skin and soft tissue infections recovered in each ward in the individual years of analysis. Listed in descending order from highest percentage: Trauma and Orthopaedic Surgery, Emergency Department, Plastic Surgery, Trauma and Orthopaedic Outpatient Clinic, Vascular Surgery, Intensive Care Unit, and Paediatric Orthopaedic Surgery. Unspecified field refers to skin and soft tissue infections detected in patients other wards

DISCUSSION

Staphylococcus. aureus is known to rapidly develop resistance to multiple antimicrobial agents; therefore, exposure of *S. aureus* to healthcare settings poses a great risk of adaptation followed by transmission of the bacterium to the hospital environment. In resource-rich countries SSTIs remain the third most common nosocomial infection (20%) [38]. Interestingly, according to data provided by Haque et al., those caused by *S. aureus* are reported as the most common among all healthcare-associated infections in the USA (HA-infections – an overview 2018) [39].

Acquisition of wound infections in hospitalized patients may occur in as many as 1/3 of them, and in ¼ the risk of developing severe sepsis as a consequence increases significantly [40]. According to Jernigan, SSIs due to *S. aureus* increased by nearly 23% over a 10-year period, with a significant increase in MRSA as the cause [41].

The incidence of SSI depends on the type of surgery – clean and dirty, the latter most often resulting from translocation of the patient's endogenous flora – enterobacteria, non-fermentative Gram-negative rods, and *Enterococcus* sp. The incidence of SSI is determined by the type of surgery with the significant risk for orthopedic (with 50% of *S. aureus* involvement), cardiac, and abdominal surgery [39]. According to O'Donnell et al., in the UK postoperative complications complicate about 5% of all surgical procedures [42]. Fifty percent of SSIs are caused by members of the patients' skin microbiota, but also by bacteria on the skin of healthcare workers [43]. A significant proportion of *S. aureus* in the etiology of SSIs is associated with transient or intermittent skin carriage (60% and 20% respectively), as demonstrated by the analysis of several authors [44].

Polish guidelines recommend *S. aureus* decolonization of patients prior to planned cardiac surgery, but do not specify the need for this procedure in orthopaedic patients. However, orthopaedic surgeons prefer to decolonize their patients because *S. aureus* remains the leading cause of bone and bone marrow infections despite the practice of preoperative antibiotic prophylaxis [45, 46, 47].

Since the late 1990s, MRSA has been most commonly associated with previously hospitalized individuals (with prior antibiotic therapy and prolonged pre-infection hospitalization history), whereas MSSA isolates are no longer the exclusive community-associated pathogens. The emergence of MRSA and MSSA strains in both healthcare and community human populations is associated with asymptomatic carriage of this bacterium in as many as 40% of healthy individuals [37, 48]. According to available data, the incidence of MRSA or multiresistant strains isolated from infections in Polish hospitals varies. The unpublished follow-up data on the incidence of MRSA infections in the West Pomeranian region, which we continuously collect and analyse, show that MRSA infection rates are increasing, which may be a consequence of restrictions and antibiotic consumption during COVID-19, leading to uncontrolled spread of this microorganism in the hospital environment. Interestingly, the majority of *S. aureus* analyzed in this study were methicillin-susceptible, and SSIs accounted for 1/5 of all diagnosed SSTIs. It can be concluded that MRSA is not yet a major cause of SSIs in Poland compared to studies from different European countries where methicillin-resistant strains are identified at higher rates [49, 50, 51, 52, 53].

Exposure to *S. aureus* in healthcare settings poses a high risk of transmission, adaptation of the bacterium to the hospital environment, and thus acquisition of resistance to multiple antimicrobial agents. It is noteworthy that MRSA often shows the simultaneous presence of a resistance mechanism to MLSB. The phenomenon of co-occurrence of both resistance phenotypes is most likely related to the nature of *SCCmec* cassette, which may act as a genetic reservoir for the insertion of various resistance determinants, including genes that confer MLSB resistance [51, 54]. In the present study, only ¼ of the MRSA isolated from SSIs showed a simultaneous constitutive MLSB phenotype, which is more frequently identified in MRSA strains isolated worldwide [55, 56].

Ciprofloxacin resistance in *S. aureus* is associated with either an altered target site (enzyme DNA gyrase namely topoisomerase II) or decreased permeability of the cell membrane and efflux pump of the drug [57]. Quinolones were originally intended for use against a wide range of pathogens, including hospital-associated *S. aureus*, but unfortunately many of its clinical isolates are now showing increasing resistance to this group of antimicrobial agents [58, 59]. In the present study, 1 MRSA and 14 MSSA isolates showed resistance to ciprofloxacin. The use of ciprofloxacin in the prevention of SSIs is limited to complications following abdominal or genitourinary surgery, where most infections are polyetiologic and the etiology originates from the patient's microbiota, including enterobacteria and strict anaerobic bacteria. The overall good susceptibility of *S. aureus* to ciprofloxacin allows its use in the treatment of SSTIs; however, the use of quinolones is associated with serious side effects, so its implementation requires discernment.

In the present study, more than a quarter of all SSIs were confirmed in patients hospitalized in the Trauma and Orthopaedic Ward of CH-1. These results are consistent with those of other authors, where the predominant proportion of *S. aureus* in SSIs was observed among isolates from orthopaedic wards. Nearly 16% of SSIs in individuals consulted at the ED of CH-1, followed by SSIs in patients hospitalized at the Plastic Surgery Ward (12.5%), and 8.6% in patients consulted at the orthopedic outpatient clinic. The percentages of SSIs in patients hospitalized in the Vascular Surgery Ward, Intensive Care Unit, and Paediatric Surgery Ward were 7.7%, 4.7%, and 3.4%, respectively. The remaining SSIs were confirmed in the other wards and represented 26.5% of all diagnosed SSIs examined in the present study [60, 61, 62, 63]. It is interesting to note that the number of SSIs in the Orthopaedic Paediatric Ward was relatively lower, but the incidence of nasal colonization with *S. aureus* in children was equal to or higher than in adults [64, 65]. Therefore, despite clear guidelines for decolonization of patients before orthopedic surgery, physician awareness and order of screening and eradication of *S. aureus* carriage may contribute to a reduction in the prevalence of these infections as a result of autoimmunity, as shown in the present study; 8.6% of SSIs

examined in the current study were confirmed in patients consulted in orthopedic outpatient clinics, indicating patients who underwent surgical procedures. According to available data SSIs in orthopaedic patients seen in outpatient clinics are quite often qualified for reoperation due to simultaneous bone involvement and infection [66].

In patients seen in the ED and included in the present study, SSTIs accounted for nearly 16% of all SSIs reviewed. These patients were consulted in the ED as outpatients, but all cases of SSIs resulted from infection or reinfection of the surgical site. It is estimated that SSIs in outpatients are among the leading causes of hospital readmissions. Available data indicate that outpatient SSIs account for ¼ of all hospital readmissions annually. However, the majority of outpatient SSIs occur after abdominal, genitourinary, vascular, or gynecologic surgeries. These infections are often the result of wound contamination during healing and postoperative rehabilitation [67, 68].

In the present study, 12.5% and 7.7% of SSIs were observed in patients admitted to the Plastic Surgery and Vascular Surgery Wards respectively. Skin decolonization prior to planned plastic surgery procedures plays a critical role in reducing the risk of SSI, and the risk of infection is strongly related to the type of plastic or reconstructive procedure performed, so the low rate of SSIs observed in this study demonstrates the essential role of preoperative procedures to prevent *S. aureus* wound contamination and its consequences [69, 70]. The study conducted by Bandyk describes the main risk factors associated with SSIs after vascular surgery as nasal carriage of *S. aureus*, recent hospitalization, unsuccessful arterial reconstruction, and groin incision [71]. Available data indicate that the incidence of SSIs after vascular surgery is dependent on type of the procedure performed, with estimates of 1–25% for open or endovascular surgery and lower extremity bypass surgery.

Despite advances in SSI prophylaxis, SSIs remain a major clinical problem associated with significant mortality and morbidity. Surgical site infections prevention takes into account the full range of risk factors, including aseptic practice and proper implementation of antibiotic prophylaxis. It is also important to emphasize the role of asymptomatic *S. aureus* carrier status in outpatients, which most likely has a huge impact on the development of wound infection during the recovery period as a result of autotransmission of the pathogen to the surgical site [72, 73, 74].

CONCLUSIONS

The results of the present study are in line with global data, which indicate that SSIs are most common in patients undergoing clean surgical procedures in which *S. aureus* is the predominant etiologic agent. The 20% share of staphylococcal SSIs among all staphylococcal SSTIs in patients at the CH-1 indicates a significant contribution of this microorganism to SSIs in the West Pomeranian region in Poland. When comparing the results from 2 years prior to preceding the study, there was no significant difference in the percentage of SSIs among CH-1 patients, so it can be concluded that there was no significant impact of COVID-19-related restrictions on the incidence and etiology of SSIs among patients of CH-1 during the observed study period. This indicates a good practice of infection prevention under the pandemic restrictions; however, the considerable number of patients in the convalescence period consulted in the ED may reveal a significant concern about acquiring *S. aureus* infection in the community settings after hospitalization. The overall good susceptibility and low percentage MRSA are optimistic for the choice of safe and effective SSI therapy but the absence of clear guidelines for mandatory screening of patients undergoing surgical procedures and control of carriage among healthcare personnel may contribute to the further spread of susceptible strains in the hospital environment and their acquisition of multidrug resistance. Strengthening infection prevention, continuous control, and systematic surveillance in healthcare facilities is recommended.

REFERENCES

1. Sakr A, Brégeon F, Mège JL, Rolain JM, Blin O. *Staphylococcus aureus* nasal colonization: an update on mechanisms, epidemiology, risk factors, and subsequent infections. Front Microbiol 2018;9:2419.
2. Yüksel YT, Edslev SM, Janstrup AK, Goldeman MS, Nørreslet LB, Andersen PS, et al. Colonization with *Staphylococcus aureus* in healthcare workers: consequences of hand eczema. Br J Dermatol 2022;187(4):609-11.
3. Garoy EY, Gebreab YB, Achila OO, Tekeste DG, Kesete R, Ghirmay R, et al. Methicillin-resistant *Staphylococcus aureus* (MRSA): prevalence and antimicrobial sensitivity pattern among patients – a multicenter study in Asmara, Eritrea. Can J Infect Dis Med Microbiol 2019;2019:8321834. doi: 10.1155/2019/8321834.
4. Laupland KB, Lyytikäinen O, Søgaard M, Kennedy KJ, Knudsen JD, Ostergaard C, et al. The changing epidemiology of *Staphylococcus aureus* bloodstream infection: a multinational population-based surveillance study. Clin Microbiol Infect 2013;19(5):465-71.
5. Shoaib M, Aqib AI, Muzammil I, Majeed N, Bhutta ZA, Kulyar MF, et al. MRSA compendium of epidemiology, transmission, pathophysiology, treatment, and prevention within one health framework. Front Microbiol 2022;13:1067284.
6. European Centre for Disease Prevention and Control. Antimicrobial resistance (EARS-Net). In: ECDC. Annual epidemiological report for 2014. Stockholm: ECDC; 2018.
7. Tong SY, Davis JS, Eichenberger E, Holland TL, Fowler VG Jr. *Staphylococcus aureus* infections: epidemiology, pathophysiology, clinical manifestations, and management. Clin Microbiol Rev 2015;28(3):603-61.
8. Laux C, Peschel A, Krismer B. *Staphylococcus aureus* colonization of the human nose and interaction with other microbiome members. Microbiol Spectr 2019;7(2).
9. Dana AN, Bauman WA. Bacteriology of pressure ulcers in individuals with spinal cord injury: what we know and what we should know. J Spinal Cord Med 2015;38(2):147-60.
10. Olaniyi R, Pozzi C, Grimaldi L, Bagnoli F. *Staphylococcus aureus* – associated skin and soft tissue infections: anatomical localization, epidemiology, therapy and potential prophylaxis. Curr Top Microbiol Immunol 2017;409:199-227.
11. Coates M, Blanchard S, MacLeod AS. Innate antimicrobial immunity in the skin: a protective barrier against bacteria, viruses, and fungi. PLoS Pathog 2018;14(12):e1007353.
12. Mertz PM, Cardenas TC, Snyder RV, Kinney MA, Davis SC, Plano LR. *Staphylococcus aureus* virulence factors associated with infected skin lesions: influence on the local immune response. Arch Dermatol 2007;143(10):1259-63.
13. Shettigar K, Murali TS. Virulence factors and clonal diversity of *Staphylococcus aureus* in colonization and wound infection with emphasis on diabetic foot infection. Eur J Clin Microbiol Infect Dis 2020;39(12):2235-46.

14. Pérez-Montarelo D, Viedma E, Larrosa N, Gómez-González C, Ruiz de Gopegui E, Muñoz-Gallego I, et al. Molecular epidemiology of *Staphylococcus aureus* bacteremia: association of molecular factors with the source of infection. Front Microbiol 2018;9:2210.
15. Vella V, Galgani I, Polito L, Arora AK, Creech CB, David MZ, et al. *Staphylococcus aureus* skin and soft tissue infection recurrence rates in outpatients: a retrospective database study at 3 US Medical Centers. Clin Infect Dis 2021;73(5):e1045-53.
16. De Rose DU, Pugnaloni F, Martini L, Bersani I, Ronchetti MP, Diociaiuti A, et al. *Staphylococcal* infections and neonatal skin: data from literature and suggestions for the clinical management from four challenging patients. Antibiotics (Basel) 2023;12(4):632.
17. Del Giudice P. Skin infections caused by *Staphylococcus aureus*. Acta Derm Venereol 2020;100(9):adv00110.
18. Sabbagh P, Riahi SM, Gamble HR, Rostami A. The global and regional prevalence, burden, and risk factors for methicillin-resistant *Staphylococcus aureus* colonization in HIV-infected people: a systematic review and meta-analysis. Am J Infect Control 2019;47(3):323-33.
19. McCaig LF, McDonald LC, Mandal S, Jernigan DB. *Staphylococcus aureus*--associated skin and soft tissue infections in ambulatory care. Emerg Infect Dis 2006;12(11):1715-23.
20. Creech CB, Al-Zubeidi DN, Fritz SA. Prevention of Recurrent Staphylococcal Skin Infections. Infect Dis Clin North Am 2015;29(3):429-64.
21. Zagólski O, Stręk P, Kasprowicz A, Białecka A. Effectiveness of polyvalent bacterial lysate and autovaccines against upper respiratory tract bacterial colonization by potential pathogens: a randomized study. Med Sci Monit 2015;21:2997-3002.
22. Holtfreter S, Jursa-Kulesza J, Masiuk H, Verkaik NJ, de Vogel C, Kolata J, et al. Antibody responses in furunculosis patients vaccinated with autologous formalin-killed Staphylococcus aureus. Eur J Clin Microbiol Infect Dis 2011;30(6):707-17.
23. Tattevin P, Schwartz BS, Graber CJ, Volinski J, Bhukhen A, Bhukhen A, et al. Concurrent epidemics of skin and soft tissue infection and bloodstream infection due to community-associated methicillin-resistant *Staphylococcus aureus*. Clin Infect Dis 2012;55(6):781-8.
24. Pal S, Sayana A, Joshi A, Juyal D. *Staphylococcus aureus*: a predominant cause of surgical site infections in a rural healthcare setup of Uttarakhand. J Family Med Prim Care 2019;8(11):3600-6.
25. Nichols RL. Postoperative infections in the age of drug-resistant Gram-positive bacteria. Am J Med 1998;104(5A):11-6S.
26. Antibacterial sutures for wound closure after surgery: a review of clinical and cost-effectiveness and guidelines for use. Ottawa (ON): Canadian Agency for Drugs and Technologies in Health; 2014.
27. Deptuła A, Trejnowska E, Dubiel G, Żukowski M, Misiewska-Kaczur A, Ozorowski T, et al. Prevalence of healthcare-associated infections in Polish adult intensive care units: summary data from the ECDC European Point Prevalence Survey of Hospital-associated Infections and antimicrobial use in Poland 2012–2014. J Hospital Infect 2017;96(2):145-50.
28. Bowler PG, Duerden BI, Armstrong DG. Wound microbiology and associated approaches to wound management. Clin Microbiol Rev 2001;14(2):244-69.
29. Khan AKA, Mirshad PV, Rashed MR, Banu G. A Study on the usage pattern of antimicrobial agents for the prevention of surgical site infections (SSIs) in a Tertiary Care Teaching Hospital. J Clin Diagn Res 2013;7(4):671-4.
30. Kalmerijer MD, van Nieuwland-Bollen E, Bogaers-Hoffman D, de Baere GA. Nasal carriage of *Staphylococcus aureus* is a major risk factor for surgical site infections in orthopedic surgery. Infect Control Hosp Epidemiol 2000;21(5):319-23.
31. Steiner Z, Natan OB, Sukhotnik I, Coran AG, Keren G. Does *Staphylococcus aureus* nasal carriage require eradication prior to elective ambulatory surgery in children? Pediatr Surg Int 2014;30(5):521-5.
32. Anderson DJ, Podgorny K, Berríos-Torres SI, Bratzler DW, Dellinger EP, Greene L, et al. Strategies to prevent surgical site infections in acute care hospitals: 2014 update. Infect Control Hosp Epidemiol 2014;35(6):605-27.
33. Gompelman M, Wertheim HFL, Bleeker-Rovers CP, Wanten GJA. Eradication of *Staphylococcus aureus* colonization by chronic use of mupirocin in patients on home parenteral nutrition. Nutrition 2021;81:110985.
34. Dupeyron C, Campillo B, Bordes M, Faubert E, Richardet JP, Mangeney N. A clinical trial of mupirocin in the eradication of methicillin-resistant *Staphylococcus aureus* nasal carriage in a digestive disease unit. J Hosp Infect 2002;52(4):281-7.
35. Pianka F, Mihaljevic AL. Prevention of postoperative infections: evidence-based principles. Chirurg 2017;88(5):401-7.
36. Bratzler DW, Dellinger EP, Olsen KM, Perl TM, Auwaerter PG, Bolon MK, et al. Clinical practice guidelines for antimicrobial prophylaxis in surgery. Am J Health Syst Pharm 2013;70(3):195-283.
37. David MZ, Boyle-Vavra S, Zychowski DL, Daum RS. Methicillin-susceptible *Staphylococcus aureus* as a predominantly healthcare-associated pathogen: a possible reversal of roles? PLoS One 2011;6(4):e18217.
38. World Health Organization. WHO guidelines on hand hygiene in health care: first global patient safety challenge clean care is safer care. Geneva: World Health Organization; 2009.
39. Haque M, Sartelli M, McKimm J, Abu Bakar M. Health care-associated infections – an overview. Infect Drug Resist 2018;11:2321-33.
40. Owens CD, Stoessel K. Surgical site infections: epidemiology, microbiology and prevention. J Hosp Infect 2008;70(Suppl 2):3-10.
41. Jernigan JA. Is the burden of *Staphylococcus aureus* among patients with surgical-site infections growing? Infect Control Hosp Epidemiol 2004;25(6):457-60.
42. O'Donnell RL, Angelopoulos G, Beirne JP, Biliatis I, Bolton H, Bradbury M, et al. Impact of surgical site infection (SSI) following gynaecological cancer surgery in the UK: a trainee-led multicentre audit and service evaluation. BMJ Open 2019;9(1):e024853.
43. Otto M. *Staphylococcus* colonization of the skin and antimicrobial peptides. Expert Rev Dermatol 2010;5(2):183-95.
44. Ellis MW, Schlett CD, Millar EV, Crawford KB, Cui T, Lanier JB, et al. Prevalence of nasal colonization and strain concordance in patients with community-associated *Staphylococcus aureus* skin and soft-tissue infections. Infect Control Hosp Epidemiol 2014;35(10):1251-6.
45. Takesue Y, Kusachi S, Mikamo H, Sato J, Watanabe A, Kiyota H, et al. Antimicrobial susceptibility of pathogens isolated from surgical site infections in Japan: comparison of data from nationwide surveillance studies conducted in 2010 and 2014–2015. J Infect Chemother 2017;23(6):339-48.
46. Sganga G, Tascini C, Sozio E, Colizza S. Early recognition of methicillin-resistant *Staphylococcus aureus* surgical site infections using risk and protective factors identified by a group of Italian surgeons through Delphi method. World J Emerg Surg 2017;12:25.
47. Tinelli M, Monaco M, Vimercati M, Ceraminiello A, Pantosti A. Methicillin-susceptible *Staphylococcus aureus* in skin and soft tissue infections, Northern Italy. Emerg Infect Dis 2009;15(2):250-7.
48. Hershow RC, Khayr WF, Smith NL. A comparison of clinical virulence of nosocomially acquired methicillin-resistant and methicillin-sensitive *Staphylococcus aureus* infections in a university hospital. Infect Control Hosp Epidemiol 1992;13(10):587-93.
49. Romaniszyn D, Różańska A, Wójkowska-Mach J, Chmielarczyk A, Pobiega M, Adamski P, et al. Epidemiology, antibiotic consumption and molecular characterisation of *Staphylococcus aureus* infections – data from the Polish Neonatology Surveillance Network, 2009–2012. BMC Infect Dis 2015;15:169.
50. Romaniszyn D, Pobiega M, Wójkowska-Mach J, Chmielarczyk A, Gryglewska B, Adamski P, et al. The general status of patients and limited physical activity as risk factors of methicillin-resistant *Staphylococcus aureus* occurrence in long-term care facilities residents in Krakow, Poland. BMC Infect Dis 2014;14:271.
51. Ilczyszyn WM, Sabat AJ, Akkerboom V, Szkarlat A, Klepacka J, Sowa-Sierant I, et al. Clonal sructure and characterization of *Staphylococcus aureus* strains from invasive infections in paediatric patients from South Poland: association between age, spa types, clonal complexes, and genetic markers. PLoS One 2016;11(3):e0151937.
52. Lee A, de Lencastre H, Garau J, Kulytmans J, Malhotra-Kumar S, Peschel A, et al. Methicillin-resistant *Staphylococcus aureus*. Nat Rev Dis Primers 2018;4:18033.
53. Antimicrobial resistance in the EU/EEA (EARS-Net). Annual Epidemiological Report for 2021. Stockholm: EDC; 2022. https://www.ecdc.europa.eu/sites/default/files/documents/AER-EARS-Net-2021_2022-final.pdf (2.06.2023).
54. Rasmussen G, Monecke S, Brus O, Ehricht R, Söderquist B. Long term molecular epidemiology of methicillin-susceptible *Staphylococcus aureus* bacteremia isolates in Sweden. PLoS One 2014;9(12):e114276.
55. Sasirekha B, Usha MS, Amruta JA, Ankit S, Brinda N, Divya R. Incidence of constitutive and inducible clindamycin resistance among hospital-associated *Staphylococcus aureus*. 3 Biotech 2014;4(1):85-9.

56. Schreckenberger PC, Ilendo E, Ristow KL. Incidence of constitutive and inducible clindamycin resistance in *Staphylococcus aureus* and coagulase-negative staphylococci in a community and a tertiary care hospital. J Clin Microbiol 2004;42(6):2777-9.
57. Marangon FB, Miller D, Muallem MS, Romano AC, Alfonso EC. Ciprofloxacin and levofloxacin resistance among methicillin-sensitive *Staphylococcus aureus* isolates from keratitis and conjunctivitis. Am J Ophthalmol 2004;137(3):453-8.
58. Weber SG, Gold HS, Hooper DC, Karchmer AW, Carmeli Y. Fluoroquinolones and the risk for methicillin-resistant *Staphylococcus aureus* in hospitalized patients. Emerg Infect Dis 2003;9(11):1415-22.
59. Norvell MR, Porter M, Ricco MH, Koonce RC, Hogan CA, Basler E, et al. Cefazolin vs second-line antibiotics for surgical site infection prevention after total joint arthroplasty among patients with a beta-lactam allergy. Open Forum Infect Dis 2023;10(6):ofad224.
60. Saadatian-Elahi M, Teyssou R, Vanhems P. *Staphylococcus aureus*, the major pathogen in orthopaedic and cardiac surgical site infections: a literature review. Int J Surg 2008;6(3):238-45.
61. Levy PY, Ollivier M, Drancourt M, Raoult D, Argenson JN. Relation between nasal carriage of *Staphylococcus aureus* and surgical site infection in orthopedic surgery: the role of nasal contamination. A systematic literature review and meta-analysis. Orthop Traumatol Surg Res 2013;99(6):645-51.
62. Al-Mulhim FA, Baragbah MA, Sadat-Ali M, Alomran AS, Azam MQ. Prevalence of surgical site infection in orthopedic surgery: a 5-year analysis. Int Surg 2014;99(3):264-8.
63. Tucci G, Romanini E, Zanoli G, Pavan L, Fantoni M, Venditti M. Prevention of surgical site infections in orthopaedic surgery: a synthesis of current recommendations. Eur Rev Med Pharmacol Sci 2019;23(2 Suppl):224-39.
64. Janeczek K, Emeryk A, Zimmer Ł, Poleszak E, Ordak M. Nasal carriage of *Staphylococcus aureus* in children with grass pollen-induced allergic rhinitis and the effect of polyvalent mechanical bacterial lysate immunostimulation on carriage status: a randomized controlled trial. Immun Inflamm Dis 2022;10(3):e584.
65. Preoțescu LL, Streinu-Cercel O. Prevalence of nasal carriage of *S. aureus* in children. Germs 2013;3(2):49-51.
66. Mitchell P, Gottschalk M, Butts G, Xerogeanes J. Surgical site infection: a comparison of multispecialty and single specialty outpatient facilities. J Orthop 2013;10(3):111-4.
67. Shah R, Pavey E, Ju M, Merkow R, Rajaram R, Wandling MW, et al. Evaluation of readmissions due to surgical site infections: a potential target for quality improvement. Am J Surg 2017;214(5):773-9.
68. Merkow RP, Ju MH, Chung JW, Hall BL, Cohen ME, Williams MV, et al. Underlying reasons associated with hospital readmission following surgery in the United States. JAMA 2015;313(5):483-95.
69. ElHawary H, Hintermayer MA, Alam P, Brunetti VC, Janis JE. Decreasing surgical site infections in plastic surgery: a systematic review and meta-analysis of level 1 evidence. Aesthet Surg J 2021;41(7):NP948-58.
70. Drapeau CM, D'Aniello C, Brafa A, Nicastri E, Silvestri A, Nisi G, et al. Surgical site infections in plastic surgery: an italian multicenter study. J Surg Res 2007;143(2):393-7.
71. Bandyk DF. Vascular surgical site infection: risk factors and preventive measures. Semin Vasc Surg 2008;21(3):119-23.
72. Hassan RSEE, Osman SOS, Aabdeen MAS, Mohamed WEA, Hassan RSEE, Mohamed SOO. Incidence and root causes of surgical site infections after gastrointestinal surgery at a public teaching hospital in Sudan. Patient Saf Surg 2020;14(1):45.
73. Muilwijk J, Walenkamp GH, Voss A, Wille JC, van den Hof S. Random effect modelling of patient-related risk factors in orthopaedic procedures: results from the Dutch nosocomial infection surveillance network 'PREZIES'. J Hosp Infect 2006;62(3):319-26.
74. Wójkowska-Mach J, Jaje E, Romaniszyn D, Kasparek M, Frańczuk B, Bulanda M, et al. Comparison of SSI rates in endoarthroplasty of hip and knee in a Cracow patient population and the importance of postdischarge surveillance. Infection 2008;36(1):36-40.