Outbreaks of infection caused by community-acquired methicillin-resistant *Staphylococcus aureus* in a Canadian correctional facility

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BACKGROUND: Methicillin-resistant *Staphylococcus aureus* (MRSA) has been identified in prison settings in the United States. The present study investigated two clusters of skin and soft tissue infection caused by community-acquired (CA) MRSA in a correctional facility in southern Ontario.

METHODS: Outbreak investigations were conducted by the responsible public health authority. Strain relatedness was assessed through comparison of pulsed-field gel electrophoresis and antibiograms.

RESULTS: Two distinct outbreaks of CAMRSA-associated disease occurred in 2002 and 2004. Most patients presented with abscesses in the lower extremities. All isolates had identical DNA banding patterns on pulsed-field gel electrophoresis. One-half of the affected inmates resided in a cellblock with one other affected inmate. No other risk factors were identified.

CONCLUSIONS: One of the first outbreaks of CAMRSA infections in a correctional facility in Canada is documented. Taken in conjunction with outbreaks elsewhere, this suggests that residence in correctional facilities may be a risk factor for CAMRSA infection.

Key Words: Canada; Community-acquired MRSA; Correctional facility; Panton-Valentine leukocidin

Community-acquired methicillin-resistant *Staphylococcus aureus* (CAMRSA) is an increasingly prevalent pathogen (1-3), with outbreaks reported in numerous settings, including pediatric populations (3), athletic teams (4-6), Aboriginal populations (7) and prison inmates (8-12). The rates of infection and colonization may be difficult to determine due to variable definitions of what constitutes CAMRSA (13). Additionally, many patients do not have identifiable risk factors for acquiring MRSA (1,2,14,15).

Skin and soft tissue infections (SSTIs) with CAMRSA may be severe and infect individuals who are young and previously healthy (2,16-21). Soft tissue infections due to CAMRSA frequently present as abscesses, which may result from the elaboration

Éclosions d'infections extra-hospitalières à staphylocoque doré résistant à la méthicilline dans un établissement correctionnel canadien

HISTORIQUE : On a découvert des souches de staphylocoque doré méthicilline-résistant (ou MRSA pour méthicilline-résistant *Staphylococcus aureus*) dans des prisons des États-Unis. L'étude actuelle se penchait sur deux séries de cas d'infections de la peau et des tissus mous extra-hospitalières à MRSA dans un établissement correctionnel du Sud de l'Ontario.

MÉTHODES : Les enquêtes sur ces éclosions ont été menées par le service de santé publique concerné. Le lien avec les souches soupçonnées a été évalué par le biais de comparaisons des résultats de l'électrophorèse sur gel en champ pulsé et des antibiogrammes.

RÉSULTATS : Deux éclosions distinctes de cas extra-hospitaliers associés au MRSA sont survenues, en 2002 et en 2004. La plupart de patients présentaient des abcès aux membres inférieurs. Tous les isolats présentaient le même ordre d'enchaînement des composantes de leur matériel génétique à l'électrophorèse sur gel en champ pulsé. La moitié des détenus atteints partageaient leur bloc cellulaire avec un autre détenu infecté. Aucun autre facteur de risque d'a été identifié.

CONCLUSION : L'une des premières éclosions d'infections extra-hospitalières à MRSA dans un établissement correctionnel au Canada se trouve ainsi documentée. Dans le contexte où d'autres éclosions sont survenues ailleurs, on peut conclure qu'un séjour dans un établissement correctionnel constituerait un facteur de risque à l'égard de l'infection extra-hospitalière à MRSA.

of virulence factors, such as Panton-Valentine leukocidin (PVL) by CAMRSA strains (16-18,21-23), the initial misdiagnosis of infections as spider bites, delaying diagnosis and treatment (9), the lack of effectiveness of empirical antibiotic regimens against CAMRSA, or some combination of these factors.

In August 2002, the Hamilton Department of Public Health and Community Services was informed of three patients with SSTIs caused by CAMRSA in a local correctional facility. An outbreak investigation was initiated with the goals of characterizing the CAMRSA transmission in the facility and controlling the outbreak. Although the source of this outbreak was not identified, cases subsided and the outbreak

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Figure 1 Epidemic curve for the outbreak of community-acquired methicillin-resistant *Staphylococcus aureus* in a Canadian correctional facility. Presence or absence of shading denotes the year the patient infection occurred

appeared to be controlled by increasing inmate hygiene. Two years later, five new patients with identical MRSA strains were identified.

METHODS

Outbreak investigation

Medical records for the infected individuals were obtained from the correctional facility and from local hospitals. Additional historical records from within the correctional facility were reviewed with the correctional staff to establish contact between inmates.

Because all three patient infections initially recognized in 2002 occurred in a single cellblock (cellblock A), permission was obtained from the facility to discuss the outbreak investigation with the 12 uninfected inmates residing in that cellblock in August 2002. These individuals expressed a willingness to cooperate with the outbreak investigation, and agreed to complete a brief, anonymous questionnaire. The questionnaire addressed risk factors for acquiring MRSA, such as previous hospitalizations, recent antibiotics, contact with health care providers, intravenous drug use and residence in northern communities or Native reserves. Inmates also voluntarily provided nares, axillae and groin swabs under the supervision of the correctional facility staff during the same investigation period. The swabs were examined for the presence of MRSA by initial culture on a mannitol salt plate containing 6 µg/mL of oxacillin (24). Any presumptive *S. aureus* colonies growing were analyzed for the *mecA* and *nuc* genes by polymerase chain reaction (PCR) (24).

Patient infections occurring in 2004 were reviewed at the time of diagnosis for MRSA risk factors by one of the investigators (CLM) or the medical staff in the correctional facility as part of MRSA surveillance. All identified patients were screened for MRSA carriage in the nares, groin and rectum at the time of diagnosis. MRSA isolates obtained in 2004 were characterized in the same way as those obtained in 2002.

Laboratory investigations

Isolates that were positive for the *mecA* and *nuc* genes by PCR were isolated for further testing. Antimicrobial susceptibility was tested using the VITEK 1 automated instrument (bioMérieux, USA). Oxacillin resistance was confirmed using the National Committee for Clinical Laboratory Standards' oxacillin screen plate

(Mueller-Hinton agar with 4% sodium chloride and 6 µg/mL of oxacillin) and results were interpreted in accordance with National Committee for Clinical Laboratory Standards' guidelines (25). Isolates were typed by *Sma*I restriction fragment length polymorphism using pulsed-field gel electrophoresis (PFGE). DNA banding patterns of isolates were compared using BioNumerics molecular fingerprinting software (version 2.5, Applied Maths, Belgium) and Tenover's criteria (26,27). Analysis of strain virulence factors (enterotoxins A to D, staphylococcus toxic shock toxin and the PVL gene) was performed at the Centers for Disease Control and Prevention in Atlanta, Georgia, USA. Isolates from 2004 were analyzed for the presence of the PVL gene by PCR (18,24). SCCMec typing was not performed.

Disease control measures

Patients were placed under contact precautions until the lesions stopped draining, although no procedure was in place to perform a terminal clean of the cells of inmates placed in isolation. Inmates in isolation had their own toilets but shared the shower areas. During the outbreaks, improved hygiene was encouraged within the cellblock, and pHisoderm soap (Chattem, Canada) was provided during the outbreak in 2002. Laundry was issued to the inmates daily and bedding was cleaned weekly.

RESULTS

Outbreak investigations

The correctional facility houses less than 500 inmates at a given time, with both young offenders and adult offenders housed in the same facility. Adults and young offenders were not allowed to mix and had different prison guards. Inmates within a given cellblock were in contact in a communal 'day room' area and the shower area. They did not engage in any contact sports. Staff reported that inmates frequently switched cells within a given cellblock; however, inmates did not interact with residents of other cellblocks. It was common for inmates to share bedding, towels and clothing within cellblocks. Contact between inmates outside of the correctional facility could not be established, and none of the patients identified during the 2004 cluster had resided in homeless shelters before incarceration.

An epidemic curve for patients with CAMRSA infection is presented in Figure 1. Summary data on individual patients are presented in Table 1. The outbreak investigation retrospectively revealed that the first patient with CAMRSA infection was discovered in May 2002, three months before the cluster of patient infections in August 2002. The final patient infection in this cluster occurred in October 2002, two months after the initial investigation had ended. The cluster of patient infections in August occurred within a single cellblock (cellblock A), while those occurring in May and October occurred in other cellblocks.

The second outbreak began in August 2004 with a single patient infection. The next patient infection occurred in a different cellblock and no contact could be established between the two patients. Two more patient infections identified in November of 2004 occurred in another cellblock (cellblock B); these patients were cellmates. The final patient was housed in a private cell in a separate cellblock.

Disease control measures

Interventions appeared to be effective in halting the outbreak in 2002. When patient infections began appearing again in 2004,

TABLE 1
Summary of individual patients

Patient number	Date of onset*	Site of infection	Initial treatment	Clinical course	Epidemiological links to other patients	Traditional risk factors for MRSA carriage†
1	May 21, 2002	Buttock	Incision and drainage, oral cephalixin	Cultures grew MRSA. Therapy changed to trimethoprim-sulfamethoxazole. Resolution of infection	Resident of cellblock A, but not concurrently with other patients	None identified
2	August 10, 2002	Right calf (cellulitis)	Oral cephalixin; incision and drainage of abscesses	Multiple leg abscesses, therapy changed to oral linezolid once antibiotic susceptibilities were available. Developed abscess at second site (shoulder) while on linezolid; shoulder abscess was culture-positive for MRSA	Resident of cellblock A, shared cellmate with patient 3. The shared cellmate lanced boils of other inmates, but was not an MRSA carrier, and did not develop an infection	None identified
3	August 13, 2002	Right thigh abscess	Incision and drainage, oral ciprofloxacin Resolution of infection	Cultures grew MRSA. Antimicrobial therapy changed to rifampin and trimethoprim-sulfamethoxazole The shared cellmate lanced boils of other inmates, but was not an MRSA carrier, and did not develop an infection	Resident of cellblock A, shared a cell with patient 2 and had been a cellmate of patient 4	None identified
4	August 29, 2002	Cellulitis of the thigh	Oral cloxacillin; inmate lanced own abscess	Abscess cultures grew MRSA. Resolution of infection	Resident of cellblock A. Cellmate of patient 3 before development of symptoms	None identified
5	October 29, 2002	Right buttock abscess	Incision and drainage; oral cloxacillin and penicillin V	Antibiotics changed to oral trimethoprim-sulfamethoxazole. Resolution of infection	None identified. Not a resident of cellblock A	None clearly identified. Previous emergency room visit for treatment of stab wound five months before onset of infection
6	August 18, 2004	Right calf	Incision and drainage, oral cephalixin	Changed to oral trimethoprim-sulfamethoxazole. Resolution of infection	None identified, first patient identified after outbreak in 2002	None identified
7	November 16, 2004	Right thigh	Incision and drainage, clindamycin	Changed to oral trimethoprim-sulfamethoxazole. Resolution of infection	No connection to patient 6	None identified. Brief imprisonment in Canada in past
8	November 20, 2004	Left nipple	Attempted incision and drainage (inmate refused), cephalixin	Changed to oral trimethoprim-sulfamethoxazole. Resolution of infection	No connection to patient 7, incarcerated during the same time period, in different cellblocks	None identified
9	November 22, 2004	Right knee	Incision and drainage, oral cephalixin	Changed to oral trimethoprim-sulfamethoxazole. Resolution of infection	Cellmate of patient 7	Two visits to the emergency room for lacerations
10	December 14, 2004	Groin and scrotum	Intravenous cefazolin, fluconazole	Changed to oral trimethoprim-sulfamethoxazole. Resolution of infection	No connection to patient 9, incarcerated during the same time period in different cellblocks	HIV/AIDS patient on combination antiretroviral therapy. Hospital admission in 1993, outpatient clinic visits in hospital

*Date when symptoms were first reported to the medical staff; †Traditional risk factors for methicillin-resistant *Staphylococcus aureus* (MRSA) carriage and infection included history of chronic medical illness, extensive antibiotic exposure and hospitalization or frequent interaction with health care settings

Figure 2) Pulsed-field gel electrophoresis of genomic DNA from methicillin-resistant *Staphylococcus aureus* isolates from a Canadian correctional facility. All inmate-derived strains had identical pulsed-field gel electrophoresis patterns. Patient numbers are indicated at the base of each row and correspond to the patient numbers indicated in Table 1. M Molecular weight markers

active surveillance was implemented for all inmates presenting with skin abscesses. Infection control staff provided education for the correctional facility staff regarding appropriate environmental cleaning procedures and improving inmate hygiene. The correctional facility nurses have since been in contact with the infection control practitioners at a neighbouring health care facility to deal with issues following the outbreak. Two isolated new patients have been identified since 2004; however, these have not resulted in transmission to other inmates, leading us to assume that the infection controls imposed are being followed.

Risk factors for CAMRSA infection

Four of five patients in the 2002 outbreak had spent time in cellblock A. Patients 3 and 4 had been cellmates, and patients 2 and 3 had both shared a cell with an inmate who 'assisted' other inmates by lancing boils and pimples. This inmate was not colonized with MRSA, and did not develop MRSA infection. No other epidemiological linkage between patients was identified.

In August 2002, 12 uninfected inmates in cellblock A voluntarily completed questionnaires that were aimed at identifying risk factors for asymptomatic MRSA carriage. Screened inmates ranged from 21 to 48 years of age. Four inmates (33%) had tattoos, and no inmates reported prior intravenous drug use. One inmate was a resident of a First Nations (Aboriginal) reserve. Eleven of 12 inmates (92%) had been previously incarcerated. Four inmates (33%) reported antibiotic use in the preceding year, and three (25%) reported skin infections during this same time period. Inmates were not questioned about sexual practices. Thirteen inmates from cellblock A were screened for staphylococcal carriage; two inmates had methicillin-susceptible *S. aureus* identified on screening, but none were colonized with MRSA.

In 2004, no epidemiological link was identified between patients 6 and 7. Patients 8 and 9 were both housed in cellblock B, where they were in direct contact with one another and developed symptoms within 24 h of each other. Patient 10

was housed in a cellblock separate from any of the other patients. All of the patients identified in 2004 were screened for MRSA carriage in the nares, groin and rectum, and all were negative with the exception of patient 10, who had concurrent folliculitis in the groin. Patients 9 and 10 had been exposed to the health care system, patient 9 had brief hospital visits for lacerations and patient 10 had an HIV infection complicated by *Pneumocystis jiroveci* pneumonia requiring hospitalization in 1993. Neither of these individuals were identified as being colonized with MRSA in hospital records. None of the inmates had chronic medical conditions, with the exception of patient 10 who was HIV-positive. All inmates denied intravenous drug use. Thus, the major identifiable risk factor for CAMRSA infection noted in this population was prior and current incarceration in the Canadian penal system.

Laboratory results

All five MRSA isolates in 2002 had identical antibiograms, with susceptibility to ciprofloxacin, trimethoprim-sulfamethoxazole, rifampin, tetracycline, vancomycin and nitrofurantoin. The outbreak strain was resistant to erythromycin (minimal inhibitory concentration of 8 µg/mL) and resistant to all beta-lactam antibiotics (minimal inhibitory concentration greater than 8 µg/mL). All 10 isolates were identical by PFGE (Figure 2). However, PFGE revealed that outbreak isolates were distinct from the health care-associated Canadian MRSA-1 and Canadian MRSA-2 currently circulating in the city. The outbreak strain has been identified as the Canadian community-acquired strain, Canadian MRSA-10 or USA 300 strain. In Canada, the MRSA-10 strain has been identified in outbreaks seen mainly in the western provinces (8).

Virulence factor analysis determined that the 2002 isolate was positive for the PVL gene, negative for enterotoxins A to D and negative for staphylococcal toxic shock toxin. The 2004 isolates additionally possessed the PVL gene. SCCMec typing was not performed.

DISCUSSION

Two outbreaks of SSTIs in a Canadian correctional facility are reported. The outbreak strain was identified as Canadian MRSA-10, which has the same PFGE pattern as USA 300, one of the outbreak strains seen in jails in the United States (1,2,7,15-19,20-22,28-31).

CAMRSA has been identified in a number of prison facilities within Canada (8), the United States and elsewhere (9-12,28). While the apparent predilection of correctional facilities for CAMRSA is puzzling, factors such as crowding, medical comorbidities, poor hygiene and sharing of personal care items in the correctional environment may enhance transmission of the bacterium once it has been introduced. We failed to identify traditional risk factors for MRSA infection or carriage in all but one affected individual in this outbreak; the last identified patient had HIV infection with a distant history of hospitalization, but had not had documented MRSA infection at that time. The absence of risk factors is commonly reported with CAMRSA (1,2,13-15). Personal contact and the sharing of fomites, such as towels, has been linked to the development of MRSA SSTIs among athletes (4-6,32,33), men who have sex with men (33) and prison populations (10,11,33). Sharing of towels, bedding and clothing was common in this facility.

Although we were unable to establish direct contact between all affected inmates, several of the patients were either cellmates or shared a cell, suggesting that direct contact may have played a role in some patient infections. Brief durations of contact between individuals have apparently been sufficient to result in transmission of MRSA in other outbreaks (4). The practice among inmates of lancing each other's boils or lancing their own boils, as identified here, has been associated with at least one other corrections-based MRSA outbreak (10). Direct contact as a source of transmission is not clear in the patients identified in 2004, with the exception of patients 8 and 9.

We were unable to identify contact between patient-inmates who were housed in distant cellblocks. We were also unable to determine a cause for a second cluster of CAMRSA SSTIs two years after the first cluster was terminated. This may imply ongoing circulation of the outbreak strain at low levels among inmates, circulation of the strain in unrecognized community networks and continuous reimportation of this strain into the jail, or may imply a role for transmission of CAMRSA by correctional facility workers. Unfortunately, contractual concerns among representatives of correctional facility staff prevented us from screening for nasal carriage in this group.

Surprisingly, we were unable to identify any carriers of MRSA within the affected cellblock during a cluster of cases or among patients during the second outbreak. Asymptomatic carriers usually act as the reservoir for infection during outbreaks caused by *S aureus* (1,3,13), and colonization is a predisposing factor for infection with CAMRSA (22,34). Further, a longitudinal study of *S aureus* isolates from prison inmates in San Francisco (USA) revealed a 45% absolute increase in the prevalence of CAMRSA from 1997 to 2002, suggesting that carriage in this population may be increasingly common (28). As noted above, our inability to evaluate the colonization status of correctional facility workers may have prevented the identification of an important reservoir for this pathogen.

The 10 patients identified presented with SSTI mainly involving the lower extremities, and abscess formation was common. SSTI is the most common presentation of CAMRSA, with invasive infection occurring less frequently (9,10,21,22,28). Abscess formation is a common presentation of CAMRSA, especially for those strains that possess the PVL gene, as this strain did (1,16,18,21,23). PVL is more frequently associated with infections resulting from direct invasion of intact skin and tissue destruction than secondary infections (18). Among affected inmates in a San Francisco correctional

facility outbreak, 70% had MRSA isolates that contained the PVL gene (23). The absence of systemic illness in this outbreak may reflect the absence of production of other toxins (such as toxic shock toxin and enterotoxins) by the outbreak strain. However, CAMRSA containing the PVL gene has caused primary community-acquired pneumonia, resulting in fatalities in previously healthy, young individuals (17,18).

Our approach to outbreak control in the present instance focused on improving hygiene, minimizing the sharing of towels and other personal care items among inmates, isolating patients with actively draining lesions and obtaining surveillance swabs of the affected cellblock, which is consistent with guidelines recently formulated by the United States Federal Bureau of Prisons for the control of MRSA outbreaks in correctional facilities (35). These measures may have helped limit the extent and duration of this outbreak. Optimal strategies for clinical management of CAMRSA infections are also still undefined, and there is some evidence that patients may respond clinically to beta-lactam antibiotics (16), especially when adjunctive incision and drainage are performed (36). The lack of a clear risk profile for CAMRSA carriage and infection further complicates empirical coverage of community-acquired SSTI. However, this report and others suggest that incarceration in a correctional facility may represent an increasingly important risk factor for the acquisition and transmission of CAMRSA infection.

BIOGRAPHICAL SKETCH: Dr Cheryl Main is a recent graduate of the Infectious Disease and Medical Microbiology Fellowship program at McMaster University (Hamilton, Ontario). Most of the work contained in this manuscript was completed during her fellowship training. She has recently been appointed to a position as an assistant professor in the department of Pathology and Molecular Medicine at McMaster University and is employed as a Medical Microbiologist and Infectious Disease consultant with Hamilton Health Sciences. She also holds a cross appointment with Public Health and is the Medical Director of the Sexual Health and Sexually Transmitted Disease programs in Hamilton.

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