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Case Report

Successful treatment of streptococcal toxic shock syndrome complicated by primary peritonitis and bilateral empyema in a healthy young woman: Identification of uncommon clone *emm103* and novel sequence type 1363

Yoshihiko M. Sakaguchi [a,d,e], Koichiro Murakami [a,*], Hiroyuki Akebo [a], Ryuichi Minoda Sada [a,f,g], Noriyuki Abe [b], Takahiro Maeda [c], Mieko Goto [c], Takashi Takahashi [c], Yusuke Takahashi [a,g], Eriko Kashihara [a], Jaegi Shim [a], Hirofumi Miyake [a], Kazuhiro Hatta [a]

[a] *Department of General Internal Medicine, Tenri Hospital, Nara, Japan*
[b] *Department of Clinical Laboratory Medicine, Tenri Hospital, Nara, Japan*
[c] *Laboratory of Infectious Diseases, Graduate School of Infection Control Sciences and Ōmura Satoshi Memorial Institute, Kitasato University, Tokyo, Japan*
[d] *International Institute for Integrative Sleep Medicine (WPI-IIIS), University of Tsukuba, Tsukuba, Ibaraki, Japan*
[e] *Ph.D. Program in Humanics, School of Integrative and Global Majors, University of Tsukuba, Tsukuba, Ibaraki, Japan*
[f] *Department of Transformative Protection to Infectious Disease, Graduate School of Medicine, Osaka University, Osaka, Japan*
[g] *Department of Infection Control, Graduate School of Medicine, Osaka University, Osaka, Japan*

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ABSTRACT

Streptococcal toxic shock syndrome (STSS) has a dramatic clinical course and high mortality rate. Here, we report a case of STSS complicated by primary peritonitis and bilateral empyema. A previously healthy young woman was diagnosed with STSS complicated by primary peritonitis and bilateral empyema. Blood culture results on admission were negative. Sever shock, respiratory failure, systemic inflammation, thrombocytopenia, renal failure, ascites, and pleural effusion occurred, mimicking thrombocytopenia, anasarca, fever, reticulin fibrosis/renal failure and organomegaly (TAFRO) syndrome. Retesting blood cultures identified *Streptococcus pyogenes*. Gram staining of ascites and pleural fluid indicated gram-positive cocci in chains. Antibiotics, immunoglobulins, and surgical intervention led to recovery without complications. Ex-post genotypic analyses showed uncommon *emm103*.0 (cluster E3) of *emm* long sequence (784 base) and novel sequence type 1363. STSS diagnosis can be difficult as it mimics other systemic inflammatory diseases. Therefore, it is crucial for clinicians to perform microbiological examinations from infection foci, even if the initial culture is negative.

Introduction

Streptococcal toxic shock syndrome (STSS) is a complication of the invasive group A streptococcal disease. It is defined by an isolation of *Streptococcus pyogenes* with the carbohydrate A antigen (group A *Streptococcus*; GAS) from a normally sterile site with shock and multi-organ failure [1,2]. Soft tissue is the most common site; primary peritonitis or empyema is rare. Blood cultures are positive in approximately 60–80 % of GAS cases [1,3]. Additionally, the mortality rate of STSS ranges from 30–70 % [4]; therefore, early diagnosis, while difficult, is important.

STSS is life-threatening, and it results from capillary leakage and tissue damage due to the release of inflammatory cytokines induced by bacterial superantigen toxins. Symptoms include fever, liver/kidney function disorders, thrombocytopenia, and rarely, body cavity effusions. Therefore, STSS may mimic **TAFRO** syndrome, a systemic inflammatory disorder characterized by **T**hrombocytopenia, **A**nasarca, including pleural effusion and ascites, **F**ever, **R**enal insufficiency, and **O**rganomegaly, including hepatosplenomegaly and lymphadenopathy [5].

Herein, we describe a case of successfully treated STSS complicated by primary peritonitis and bilateral empyema.

Case report

A gravida 0 para 0 22-year-old healthy woman, without relevant medical history, presented at our hospital with gradually worsening

* Correspondence to: Department of General Internal Medicine, Tenri Hospital, 200 Mishima-cho, Tenri-city, Nara 632-8552, Japan.
E-mail address: murakami@tenriyorozu.jp (K. Murakami).

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lower left abdominal pain that began 1 day prior. The patient had no smoking or drinking habits and no travel record, but she had eaten raw chicken approximately 7 days prior. She had sexual intercourse with her male partner, and her last menstruation occurred 2 weeks prior (regular cycle). On admission, she was alert. She had a fever of 39.6 °C, 108/63 mmHg blood pressure, 125 beats/min heart rate, oxygen saturation (on room air) of 98 %, and respiratory rate of 18 cycles/min, and displayed lower abdominal and rebound tenderness. The patient did not exhibit any redness or swelling of the throat, and there were no skin lesions or rashes. Initial laboratory examinations revealed an increased white blood cell count ($12 \times 10^3/mm^3$, standard value $3.3–8.6 \times 10^3/mm^3$) and an elevated C-reactive protein (CRP) level (18 mg/dL, standard value 0–0.14 mg/dL). Blood culture results were negative. Contrast-enhanced computed tomography (CT) revealed a localized thickened peritoneum in the left lower abdomen (Fig. 1A). The patient was given conservative treatment without antimicrobial therapy and was closely monitored in the ward.

On hospitalization day 5, chest pain occurred with continued fever. A second contrast-enhanced CT scan revealed a generalized thickened peritoneum, increased ascites, and bilateral pleural effusions (Fig. 1A). Samples for blood culture were collected once again. Overnight, respiratory and circulatory failure developed, and on the morning of the day 6 she was immediately transferred to the intensive care unit (ICU) for intubation for hypoxemia. Epinephrine, norepinephrine, and arginine vasopressin were administered to treat severe shock. Laboratory examinations revealed a normal hemoglobin level (12 g/dL, standard value 11.6–14.8 g/dL), a normal white blood cell count ($6.3 \times 100^3/mm^3$), a remarkably decreased platelet count (6.8×10^4 /mm^3, standard value $15.8–34.8 \times 10^4$ /mm^3), a remarkably elevated CRP level (43 mg/dL), an increased blood urea nitrogen level (40 mg/dL, standard value 8–20 mg/dL), an elevated creatine level (2.2 mg/dL, standard value 0.5–0.8 mg/dL), an increased interleukin-6 level (4.0×10^4 pg/mL, standard value <7 pg/mL), and an elevated vascular endothelial growth factor level (1.4×10^3pg/mL, standard value <38 pg/mL). Systemic inflammation, thrombocytopenia, and renal failure had occurred. Given septic shock with unknown foci or TAFRO syndrome as a differential diagnosis, we initiated intravenous meropenem and methylprednisolone. Re-examined blood cultures from the day 5 and gram staining of ascites and bilateral pleural effusions yielded gram-positive cocci in chains. The isolate was also identified as GAS from blood cultures

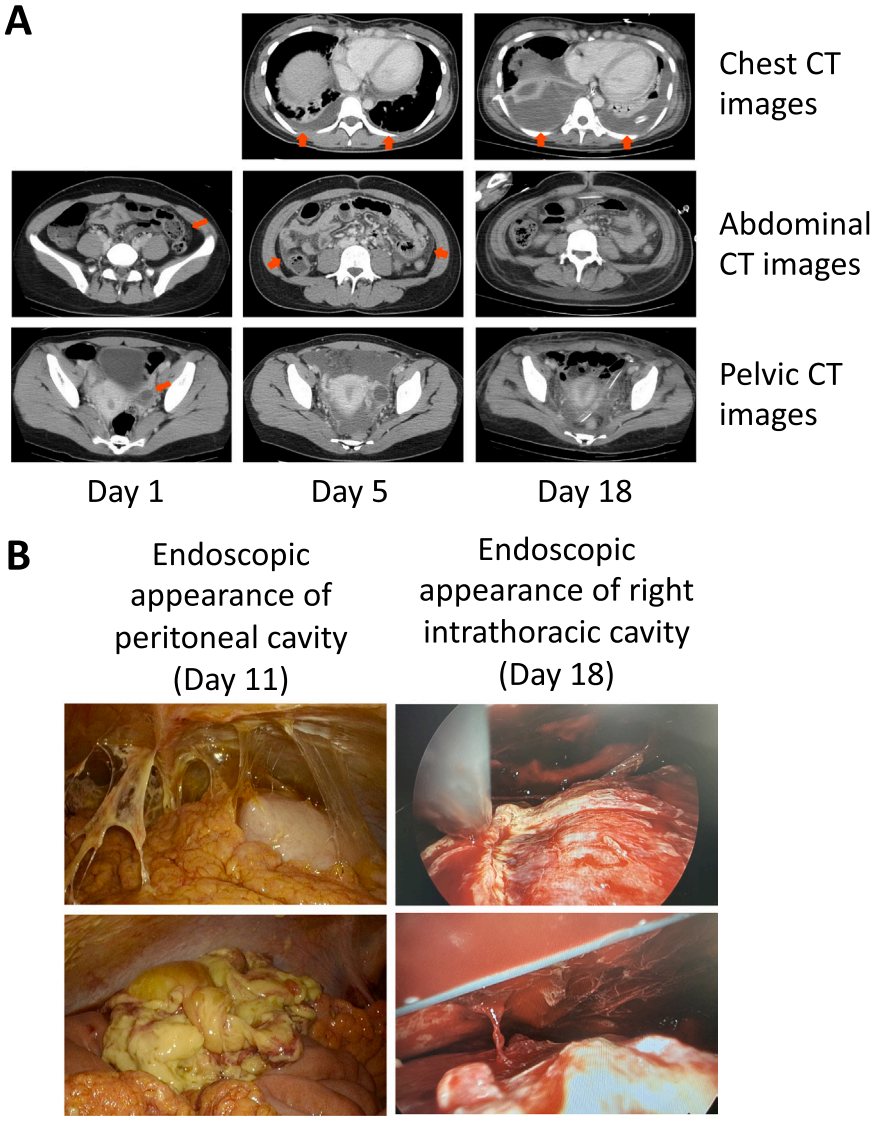

Fig. 1. (A) Contrast-enhanced computed tomography (CT) images from the chest to pelvic cavity on hospitalization days 1, 5, and 18 and (B) endoscopic appearances of peritoneal and right intrathoracic cavities on hospitalization days 11 and 18. A left ovarian cyst and a localized thickened peritoneum of the left lower abdomen on the day 1 (arrows). Generalized thickened peritoneum, increased ascites, and bilateral pleural effusions on the day 5 (arrows). Increased bilateral pleural effusions on the day 18 (arrows).

Table 1
Phenotypic/genotypic traits of blood-origin *S. pyogenes* and genotypic traits of ascites-origin DNA from the patient with toxic shock syndrome.

Sample ID	AB1	AB2
Isolation source	Blood	Ascites
Matrix assisted laser desorption/ ionization-time of flight mass spectrometry	*S. pyogenes* (score value 2.259)	NA
Reaction for pyrrolidonylarylamidase production	Positive	NA
Similarity to 16 S rRNA gene sequence of the JCM 5674(T) (sequencing size)	100 % (718-bp)	100 % (718-bp)
emm type (subtype) based on the hyper-variable region of 180 base [*emm*-cluster]	*emm103*.(0) [E3]	*emm103*.(0) [E3]
emm sequence of AB1 and AB2[a]	Identical to that (638-bp) of *S. pyogenes* RE612 strain from Japan[b]	Identical to that (638-bp) of *S. pyogenes* RE612 strain from Japan[b]
Profile of exotoxin/superantigen genes	*speB* alone	*speB* alone
Sequence type (ST, allelic profile: *gki-gtr-murI-mutS-recP-xpt-yqiL*)	Novel ST1363 (83-2-8-6-2-3-4)	Novel ST1363 (83-2-8-6-2-3-4)
Antimicrobial susceptibility testing data	Susceptible to antimicrobials examined	NA
Profile of antimicrobial resistance (AMR) genes	Not detected	Not detected

NA, not available. When doing genotypic analyses, we included S. pyogenes American Type Culture Collection 12344(T) as a positive control.
Profile of exotoxin/superantigen genes covered the detection of *speA*, *speB*, *speC*, *ssa*, and *smeZ* genes.
Profile of AMR genes contained the detection of *blaZ*, *erm*(A), *erm*(B), *mef*(A), *linB*, *lnu*(D), *tet*(M), *tet*(O), *tet*(K), *tet*(L), and *tet*(S).
[a] GenBank accession numbers of *emm* sequences (784-bp) of AB1 and AB2 are LC761208 and LC761209, respectively.
[b] GenBank accession number of *S. pyogenes* stMrp6.0 and *emm*103 genes encoding M related protein and M protein is AB549958.1.

(Table 1). The patient was diagnosed with STSS complicated by primary peritonitis and bilateral empyema.

Fig. 2 shows the main clinical and therapeutic courses. Intensive care including three vasopressors, invasive positive pressure ventilation, and renal replacement therapy continued, Antibiotics changed to penicillin G plus clindamycin, with the intravenous immunoglobulin. We performed thorough drainage; the refractory bilateral empyema required continuous drainage and surgical intervention on the day 18. Peritonitis was treated surgically on the day 11, followed by continuous drainage (Fig. 1B). The patient's condition gradually improved, and she was discharged from the ICU on the day 27. After rehabilitation, she was discharged with full recovery on the day 77.

Mass spectrometry identified the isolate as GAS (Score value 2.259), with a positive reaction for pyrrolidonyl arylamidase production. Table 1 summarizes phenotypic/genotypic traits of the blood-origin isolate (sample ID AB1) and ascites-origin DNA (sample ID AB2). Both samples with identical sequences (718-bp) were identified as GAS based on 16 S rRNA sequencing data, indicating identity with the GAS JCM 5674(T) sequence. Phenotypic analyses included hemolysis, Lancefield carbohydrate antigen, pyrrolidonyl arylamidase production reaction, and antimicrobial susceptibility testing. Genotypic analyses included *sagA* (111-bp fragment) and *slo* (548-bp fragment) encoding hemolysin amplification, *emm* type/subtype based on the hyper-variable region of 180 base [6], *emm* sequence, superantigen gene profile [7], sequence type (ST) (allelic profile; *gki-g tr -murI-mutS-recP-xpt-yqiL*) by multilocus sequence typing [6], and antimicrobial resistance (AMR) gene profile [6, 8,9]. The *emm* genotyping was based on the Centers for Disease Control and Prevention database (https://www2.cdc.gov/vaccines/biotech/strepblast.asp). The superantigen genes included *speA*, *speB*, *speC*, *ssa*, and *smeZ* detected. We used the PubMLST website (https://pubmlst.org/bigsdb?db=pubmlst_spyogenes_seqdef) to determine the ST. AMR genes included *blaZ*, *erm*(A), *erm*(B), *mef*(A), *linB*, *lnu*(D), *tet*(M), *tet*(O), *tet*(K), *tet*(L), and *tet*(S) detected. For genotypic analyses, we used the GAS American Type Culture Collection 12344(T) as a positive control.

Phenotypic analyses revealed weak hemolysis, Lancefield carbohydrate antigen A, and susceptibility to antimicrobials. Genotypic analyses indicated amplified *sagA* and *slo*, *emm103*.0 [*emm*-cluster E3], *emm* long sequence of 784 base (GenBank accession numbers from AB1 and AB2: LC761208 and LC761209), *speB* alone, novel ST1363 (83−2-8−6-2−3-4), and no detection of AMR genes. Both samples showed the same genotypic data.

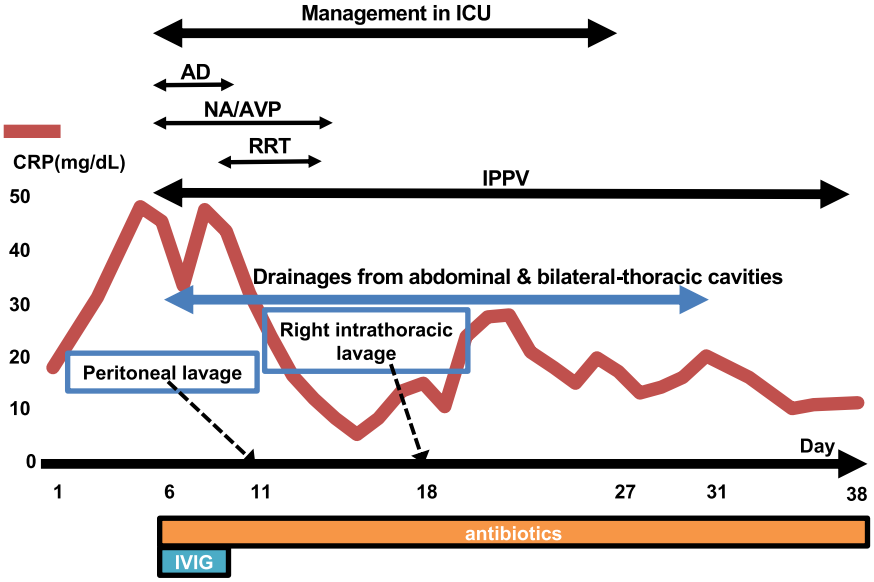

Fig. 2. Clinical and therapeutic course from hospitalization day 1 to 38. We administered 1 g/kg of IVIG on the day 1, and 0.5 g/kg on the days 2 and 3. CRP, C-reactive protein; ICU, intensive care unit; IPPV, invasive positive pressure ventilation; AD, epinephrine; NA, norepinephrine; AVP, arginine vasopressin; IVIG, intravenous immunoglobulin; RRT, renal replacement therapy.

Discussion

We presented a case of STSS complicated by primary peritonitis and bilateral empyema in a healthy young woman. Peritonitis or empyema caused by GAS in adults is uncommon; literature reviews and case reports are limited [10–12]. We reviewed the literature via the keywords "streptococcal toxic shock syndrome, empyema, abscess" or "streptococcal toxic shock syndrome, peritonitis" into the PubMed database (https://pubmed.ncbi.nlm.nih.gov/), Medline and Igaku Chuo Zasshi. Four cases occurred (as of May 18, 2023). Two patients died [13,14], one had a good outcome with peritonitis and slight unilateral pleural effusion [15], and one had primary peritonitis and bilateral empyema, as in our case, and was successfully treated [16]. Thus, primary peritonitis and bilateral empyema in patients with STSS is very rare, and few have survived.

STSS can mimic TAFRO syndrome. The early pathological conditions in our case mimicked TAFRO regarding the negative blood culture at admission, and severe systemic inflammatory reactions, including marked elevation of serum interleukin-6 and vascular endothelial growth factor levels. However, some differences were noted retrospectively (exudative ascites and pleural effusion, lack of hepatosplenomegaly or lymphadenopathy, and normal range alkaline phosphatase). Ultimately, the second blood culture confirmed the diagnosis which helped save the patient. Thus, clinicians should perform microbiological examinations from infection foci, even with initial negative cultures [17].

The *emm103* is an uncommon clone [18]. Table 2 displays the twenty-four isolates with *emm103* from the PubMLST isolate database (https://pubmlst.org/bigsdb?db=pubmlst_spyogenes_isolates) [19], identifying four isolates of ST327 (83–2-8–6-74–3-4; a single locus variant of ST1363)/*emm103* (as of March 24, 2023). A Japanese isolate from an 81-year-old man with necrotizing fasciitis in Aichi (Table 2), suggested that clonal complex 327 (ST327 and ST1363) harboring *emm103* may possess virulent properties. The *emm103*/ST1330 isolates causing primary invasive diseases may show clonal spread from 2019–2020 in South Africa, with their virulent properties.

Literature reviews on GAS-associated primary peritonitis ($n = 45$) recovered this pathogen in cervicovaginal fluid (3), skin (2), nose/throat cultures (2), and histological examination of the appendix (1), suggesting the entry or colonized sites [10]. Our patient had recent sexual intercourse, and an enlarged ovarian cyst (Fig. 1A) which improved with the severity of disease, suggesting vaginal entry sites, although cervicovaginal fluid cultures were not performed. We did not demonstrate the isolation on the entry or colonized sites (as a limitation in our patient).

Conclusion

We encountered a healthy woman with STSS complicated by primary peritonitis and bilateral empyema, a rare pathological condition caused by a clone with rare *emm103* and novel ST1363. STSS can mimic TAFRO syndrome, and retesting cultures of blood and other sterile specimens

Table 2
A list of *S. pyogenes* isolates with *emm103* ($n = 24$) on PubMLST Isolate database.

Isolate	Country (region)	Year	Sex	Age (year)	Primary disease	Tissue source	Other disease	Sequence type (ST) [*gki-gtr-murI-mutS-recP-xpt-yqiL*]
SS1370	Papua New Guinea	1992			Invasive	Sterile site		ST327 [83-2-8-6-74-3-4]
708-01	Nepal (village B)	2000			Impetigo	Impetigo lesion		ST201 [3-2-8-5-58-54-4]
SMH086	UK (London)	2003			Pharyngitis and/or tonsillitis	Upper respiratory tract		ST311 [4-2-2-51-1-59-1]
SMH112	UK (London)	2003				Purulent exudate		ST327 [83-2-8-6-74-3-4]
BSPY079	Mexico	1999						ST327 [83-2-8-6-74-3-4]
TW128	Taiwan	1992			Bacteremia	Blood		ST392 [97-3-1-7-39-69-3]
EU 533	Germany	2004	Male	62	Toxic shock syndrome	Skin/soft tissue	Wound	ST450 [4-2-3-4-1-5-72]
NS4007	Australia (Northern Territory)	2004	Male	12	Impetigo	Impetigo lesion		ST611 [83-24-8-6-74-3-4]
K12537	Kenya (Kilifi)	2004	Male		Pneumonia	Blood	Bacteremia	ST233 [4-2-2-4-1-5-72]
K49285	Kenya (Kilifi)	2011	Male		Skin/soft tissue infection	Skin/soft tissue		ST733 [47-2-2-4-1-5-72]
KK1614	Japan (Aichi)	2011	Male	81	Necrotizing fasciitis	Blood		ST327 [83-2-8-6-74-3-4]
2251-8_S14	The Gambia				Skin/soft tissue infection	Skin		ST1201 [3-2-8-5-2-3-4]
SH11042A	Portugal	2015	Female	60		Blood		ST1283 [4-2-3-4-1-5-35]
57857	South Africa (Kwa-Zulu Natal)	2019	Female	0	Invasive			ST1330 [3-2-8-5-58-3-4]
58208	South Africa (Kwa-Zulu Natal)	2019	Female	20	Invasive			ST1330 [3-2-8-5-58-3-4]
58544	South Africa (Western Cape)	2019	Female	53	Invasive			ST1330 [3-2-8-5-58-3-4]
60233	South Africa (Eastern Cape)	2019	Female	54	Invasive			ST1330 [3-2-8-5-58-3-4]
61258	South Africa (Western Cape)	2019	Male	64	Invasive			ST1330 [3-2-8-5-58-3-4]
61530	South Africa (Eastern Cape)	2020	Male	31	Invasive			ST1330 [3-2-8-5-58-3-4]
62088	South Africa (Kwa-Zulu Natal)	2020	Female	67	Invasive			ST1330 [3-2-8-5-58-3-4]
62329	South Africa (Gauteng)	2020	Female	22	Invasive			ST1330 [3-2-8-5-58-3-4]
63930	South Africa (Mpumalanga)	2020	Male	9	Invasive			ST1330 [3-2-8-5-58-3-4]
64114	South Africa (Eastern Cape)	2020	Female	2	Invasive			ST1330 [3-2-8-5-58-3-4]
AB1 (this case)	Japan (Nara)	2022	Female	22	Peritonitis	Blood	Toxic shock syndrome, empyema	ST1363 [83-2-8-6-2-3-4]

from infection foci can be key to diagnose and improve patient prognosis.

Ethical approval

NA

Consent

Informed consent was obtained from the patient and her family for the publication of this case report and may be made available upon request.

Role of the funding source

This study was supported in part by General Research Fund (2022) from the Graduate School of Infection Control Sciences and Ōmura Satoshi Memorial Institute, Kitasato University.

CRediT authorship contribution statement

Yoshihiko M. Sakaguchi: Conceptualization, Formal analysis, Data curation, Writing – original draft, Writing – review & editing, Visualization. **Koichiro Murakami:** Conceptualization, Formal analysis, Data curation, Writing – review & editing, Visualization, Project administration. **Hiroyuki Akebo:** Formal analysis, Writing – review & editing. **Ryuichi Minoda Sada**: Conceptualization, Formal analysis, Investigation, Data curation, Project administration, Supervision, Writing – original draft, Writing – review & editing. **Noriyuki Abe:** Formal analysis, Writing – review & editing. **Takahiro Maeda**: Formal analysis, Writing – review & editing. **Mieko Goto:** Formal analysis, Writing – review & editing. **Takashi Takahashi**: Conceptualization, Formal analysis, Investigation, Writing – original draft, Writing – review & editing, Supervision, Project administration, Funding acquisition. **Yusuke Takahashi:** Formal analysis, Writing – review & editing. **Eriko Kashihara**: Writing – review & editing. **Jaegi Shim:** Writing – review & editing. **Hirofumi Miyake**: Writing – review & editing. **Kazuhiro Hatta:** Writing – review & editing.

Declaration of Competing Interest

None.

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Consent for publication

Informed consent was obtained from the patient and her family for the publication of this case report.

Data availability

Novel ST1363 has been deposited into the PubMLST isolate database (https://pubmlst.org/bigsdb?db=pubmlst_spyogenes_isolates), where the sender was Takahiro Maeda, B.P. (Laboratory of Infectious Diseases, Graduate School of Infection Control Sciences and Ōmura Satoshi Memorial Institute, Kitasato University, Tokyo, Japan), and is available with the patient and other isolate information.

Authorship statement

All authors meet the ICMJE authorship criteria. Conceptualization – YMS, KM, RMS, TT; data curation – YMS, KM, RMS; formal analysis – YMS, KM, NA, YT, HA, RMS, TM, MG, TT; funding acquisition – TT; investigation – RMS, TT; methodology – None; project administration – KM, RMS, TT; resource – NA; software – None; supervision – RMS, TT; validation – None; visualization – YMS, KM, TT; Writing – original draft – YMS, RMS, TT; Writing – review & editing – YMS, KM, HA, RMS, NA, TM, MG, TT, YT, EK, JS, HM, KH. All authors read and approved the final manuscript.

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