We report two autopsy cases of severe fever with thrombocytopenia syndrome (SFTS) with a high fatality rate in aged Japanese patients. Both cases were caused by a tick-bite. The pathognomonic histological feature was necrotizing lymphadenitis of systemic lymphoid tissue with SFTS viruses and SFTSV-RNA copies. Marked fungal infections were also observed in the lungs of both patients. Since cellular immune function may be suppressed in SFTS patients, physicians should be aware of possible fungal infections.

Key words: autopsy cases, fungal infection, necrotizing lymphadenitis, SFTS virus-nucleoprotein antigen, SFTS

Severe fever with thrombocytopenia syndrome (SFTS) is a recently identified viral infectious disease in China that is caused by a novel bunyavirus, which is classified into the genus *phlebovirus* in the family *Bunyaviridae*, SFTS virus (SFTSV). SFTS occurs mainly in the spring and summer and is transmitted by tick bite. The virus can also infect people by contact with bodily fluids from SFTS patients. The disease is characterized by a sudden onset of fever, thrombocytopenia, hemorrhagic tendency, and gastrointestinal symptoms. Multi-organ dysfunction is also observed, and the mortality rate ranges from 12% to 30%. Cases of SFTS in Japanese patients with a higher fatality rate (55%) are very similar to the severe cases reported in Chinese patients. Phylogenetic analyses have indicated that some SFTSVs isolated from Japanese patients formed a genotype independent of those of Chinese patients. There is only one previous report of an autopsy case with SFTS, but there are no published reports describing the pathological findings of fungal infection. Another tick-borne phlebovirus, Heartland virus (HRTV), was recently discovered in the USA, which is phylogenetically associated with SFTS, causes severe febrile illness with thrombocytopenia, leukopenia, and elevated liver enzymes.

We report the autopsy findings of two SFTS patients who died due to severe fungal infection. Since SFTS patients have a tendency for immunodeficiency, we discuss the need for awareness of possible fungal infections in these patients.

**CLINICAL SUMMARY**

**Case 1**

An 83-year-old Japanese female, who lived in the Kagoshima prefecture of Japan, had a tick bite in the left inguinal area in
April, 2013, and 6 days later she had a sudden onset of a mild fever with general fatigue and appetite loss. On day 5 after onset, she suffered from remittent fever (around 38 degrees) with systemic muscle pain, and laboratory test revealed leukopenia, thrombocytopenia, and elevated AST, and CPK (Table 1). She was admitted to Kanoya Medical Center and was treated with intravenous minocycline, but there was no improvement in either her clinical symptoms or laboratory data.

On day 8 after onset, she had marked oral hemorrhage. The laboratory test data indicated thrombocytopenia, hemorrhagic tendency, marked liver damage, and disseminated intravascular coagulation (Table 1). A contrast CT scan showed enlargement of the left inguinal lymph node. A bone marrow aspirate showed an increase in hemophagocytes (Fig. 1a). She was treated with methylpredonisolone (1400 mg equivalent to prednisolone), anti-thrombin III, and recombinant thrombomodulin and given preventive therapy with ampicillin/sulbactam and micafungin. Her clinical features suggested SFTS, which was confirmed by RT-PCR of her blood at the National Institute of Infectious Diseases (NIID) in Japan.

On day 12 after onset when she was admitted to our hospital, she was afebrile, and her hemodynamics were approaching stable levels with use of low-dose dopamine. However, her oxygenation status had not recovered with the use of 80% fractional inspired O₂. A chest X-ray revealed a bilateral infiltrative shadow (Fig. 1b). Laboratory tests on admission are shown in Table 1. (1-3)-β-D glucan was markedly increased (Table 1), and her blood culture was positive for pseudomonas aeruginosa. Despite administration of meropenem, amikacin, and amphotericin B, she died on day 14 after onset.

### Case 2

An 88-year-old Japanese male, who lived in the Kagoshima prefecture of Japan, had a tick bite in the anterior neck in August, 2013, and 2 days later he had a remittent fever (around 38 degrees). He was treated with minocycline tablets, but there was no improvement in his clinical symptoms, and he developed anorexia and diarrhea. On day 2 after onset, he had lymph node swelling in the anterior neck and livedo reticularis-like skin rashes on both legs. Laboratory tests revealed leukopenia, thrombocytopenia, and liver damage (Table 2). The clinical features suggested SFTS, which was confirmed by RT-PCR using his blood at the NIID. Since a bone marrow aspirate showed an increase of hemophagocytes, he was given prednisolone (900 mg equivalent to prednisolone).

On day 5 after onset when he was admitted to our hospital, he was not fully conscious and had respiratory failure. A non-contrast whole body CT scan indicated acute hepatitis, but no intracranial or lung lesions were observed. Laboratory tests revealed leukopenia, thrombocytopenia, and liver damage (Table 2). The clinical features suggested SFTS, which was confirmed by RT-PCR using his blood at the NIID. Since a bone marrow aspirate showed an increase of hemophagocytes, he was given prednisolone (900 mg equivalent to prednisolone).

On day 5 after onset when he was admitted to our hospital, he was not fully conscious and had respiratory failure. A non-contrast whole body CT scan indicated acute hepatitis, but no intracranial or lung lesions were observed. Laboratory tests indicated a worsening of thrombocytopenia, liver damage, and a coagulation abnormality (Table 2). On day 9 after onset, renal failure and a circulatory disturbance developed, and the respiratory failure also worsened. A chest X-ray revealed a bilateral infiltrative shadow (Fig. 2a). The thrombocytopenia peaked, but a marked increase in AST, ALT, and LDH indicated significant liver damage (Table 2). A

### Table 1 Laboratory data of case 1

| Case 1 | Reference range, adult | 5 days after onset | 8 days after onset | 12 days after onset |
|--------|-----------------------|-------------------|-------------------|-------------------|
| Hematocrit (%) | 35.0–48.0 | 37.3 | 40.9 | 28.5 |
| Hemoglobin (g/dl) | 12.0–16.0 | 12.9 | 14.3 | 9.8 |
| White-cell count (/μL) | 4500–8500 | 2200 | 5460 | 13180 |
| Lymphocyte count (/μL) | 1000–4000 | 315 | 4.1 | 5.0 |
| Neutrophil count (/μL) | 1000–7500 | 1232 | 679 | 3210 |
| Aspartate aminotransferase (IU/L) | 13–33 | 679 | 3210 | 323 |
| Alanine aminotransferase (IU/L) | 6–30 | 290 | 1073 | 282 |
| Lactate dehydrogenase (IU/L) | 119–229 | 972 | 2736 | 648 |
| Creatine phosphokinase (IU/L) | 45–163 | 4414 | 5926 | 304 |
| C-reactive protein (mg/dl) | <0.3 | 0.4 | 0.52 | 4.94 |
| Prothrombin time (%) | 70–120 | 61.4 | 100 |
| Activated partial thromboplastin time (seconds) | 26.1–35.6 | 64.1 | 35.1 |
| Fibrinogen (mg/dl) | 150–450 | 142 | 407 |
| Antithrombin III (mg/dl) | 80–120 | 124 |
| Fibrin/fibrinogen degradation products (μg/ml) | <5.0 | 14.0 | 4.7 |
| D dimer (μg/ml) | <1.0 | 5.4 | 0.8 |
| (1-3)-β-D glucan (pg/ml) | <3.8 | 261.7 |
Figure 1  Clinical images and pathological findings of Case 1. (a) Bone marrow finding and (b) chest X-ray image, (c,d) gross findings in the lungs, (e,g–j) hematoxylin and eosin staining, (f) Grocott staining, and (k,l) immunohistochemistry (IHC) using anti-SFTS-NP antibody. (a) In the bone aspirate, many histiocytes show hemophagocytosis (×400). (b) A chest X-ray reveals a bilateral infiltrative shadow without consolidation. The cut surface of the right lung shows (c) many dispersed white nodular legions, (d) mainly in the lower lobe. (e) In the lung, there is necrotizing inflammation (×40) and (f) Aspergillus infection in the nodular lesions (Grocott staining ×400). (g) A tracheal ulcer with Aspergillus was also noted (×100). (h) Hyaline membrane formation indicating secondary diffuse alveolar damage is seen (×100). (i,j) In the left inguinal lymph node, the basic architecture of the lymph node is replaced by massive necrosis with infiltration of lymphocytes, histiocytes, some atypical lymphoid cells, and a significant amount of nuclear debris, but no neutrophils are observed (i, ×40; j, ×200). (k) In IHC of the lymph node, SFTS-NP-positive cells are found (×100), and (l) positive staining for the SFTS-NP antigen is detected in the cytoplasm of atypical lymphoid cells (×400).
blood culture was negative for both bacteria and fungi. He died 12 days after onset of the fever.

**PATHOLOGICAL FINDINGS**

Immunohistochemistry (IHC) was performed as previously described. Rabbit anti-SFTSV-nucleoprotein (NP) serum and peroxidase-labeled polymer-conjugated anti-rabbit immunoglobulin (En Vision/HRP, Dako, Glostrup, Denmark) were used.

SFTSV RNA was extracted from paraffin-embedded tissue sections using a Pure Link FFPE RNA isolation kit (Invitrogen, Thermo Fisher Scientific Inc., Waltham, MA) at the NIID. The SFTSV copy number was determined by performing quantitative real-time RT-PCR on the tissue extracts as previously described. The amount of human β-actin mRNA in the RNA extracted from each section was also determined and used as an internal reference for normalization.

The study was conducted in accordance with the guiding principles of the Declaration of Helsinki. Informed written consent was obtained from the family of each patient.

**Case 1**

At the autopsy, subcutaneous hemorrhages were observed at the neck, bilateral inguinal regions, and limbs. The lungs were heavy (Lt.: 904g, Rt.: 1284g) with many white nodular lesions (Fig. 1c,d) showing necrotizing inflammation with Aspergillus infection, and many bronchi and blood vessels were invaded by Aspergillus (Fig. 1e,f). A tracheal ulcer with Aspergillus was also noted (Fig. 1g). There was focal hyaline membrane formation, indicating diffuse alveolar damage (DAD) (Fig. 1h). Hemophagocytosis was not found in all the sections examined. The left inguinal node near the location of the tick-bite showed necrotizing lymphadenitis with apoptotic cells and necrosis (Fig. 1i). The basic architecture of the lymph node was replaced by massive necrosis with infiltration of lymphocytes, histiocytes, atypical lymphoid cells, and abundant nuclear debris, but without neutrophils (Fig. 1j). The palatine tonsil also showed similar findings. The other lymph nodes did not show necrotizing lymphadenitis. The liver (1204 g) showed single cell necroses and mild periportal lymphocytic infiltration with focal bile stasis. The central nervous system (CNS; 1130 g) showed no pathological changes.

**Case 2**

At the autopsy, a tick-bite wound was found in the anterior neck, and the associated cervical lymph nodes were swollen. Subcutaneous hemorrhages in the bilateral forearms, anterior chest, and abdomen were observed. The lungs were heavy (Lt.: 700g, Rt.: 800g) and showed foci of pulmonary

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**Table 2**

| Case 2 | Reference range, adult | 2 days after onset | 5 days after onset | 9 days after onset | 11 days after onset |
|--------|-----------------------|-------------------|-------------------|-------------------|-------------------|
| Hematocrit (%) | 35.0–48.0 | 38.9 | 31.7 | 33.5 | 34.5 |
| Hemoglobin (g/dl) | 12.0–16.0 | 13.7 | 11.4 | 11.7 | 12.4 |
| White-cell count (×10³/μL) | 4500–8500 | 1000 | 1050 | 5230 | 6240 |
| Lymphocyte count (×10³/μL) | 1000–4000 | 347 | 359 |
| Neutrophil count (×10³/μL) | 1000–7500 | 535 |
| Aspartate aminotransferase (IU/L) | 13–33 | 144 | 218 | 2162 | 5897 |
| Alanine aminotransferase (IU/L) | 6–30 | 64 | 75 | 370 | 544 |
| Alkaline dehydrogenase (IU/L) | 119–229 | 350 | 634 | 6765 | 10018 |
| Creatine phosphokinase (IU/L) | 45–163 | 424 | 1401 | 930 | 795 |
| Blood urea nitrogen (mg/dl) | 8.0–22.0 | 25 | 16.5 | 47.5 | 20.0 |
| Creatinine (mg/dl) | 0.4–0.7 | 0.88 | 0.74 | 3.20 | 1.19 |
| C-reactive protein (mg/dl) | <0.3 | 0.1 | 0.07 | 1.74 | 0.82 |
| Prothrombin time (%) | 70–120 | 73.0 | 84 | 66 |
| Activated partial thromboplastin time (seconds) | 26.1–35.6 | 48.7 | 66.4 | 126.0 |
| Fibrinogen (mg/dl) | 150–450 | 137 | 113 | <70 |
| Antithrombin III (mg/dl) | 80–120 | 75.0 | 67 | 95 | 73 |
| Fibrinogen/fibrinogen degradation products (μg/ml) | <5.0 | 5.1 | 5.1 | 13.0 | 8.5 |
| D dimer (μg/ml) | <1.0 | 3.1 | 2.0 | 6.6 | 3.6 |
Figure 2  Clinical images and pathological findings of case 2. (a) Chest X-ray image, (b,c) gross findings in the lungs, (d,e,g–j) hematoxylin and eosin staining, (f) Grocott staining, and (k,l) immunohistochemistry (IHC) using anti-SFTSV-NP antibody. (a) A chest X-ray reveals a bilateral infiltrative shadow without consolidation. (b,c) The cut surface of the right lung shows foci of pulmonary hemorrhage and infarction. (d) Diffuse hemorrhagic infarction (×40) and (e,f) angio-invasion of Mucor (e, ×200; f, Grocott staining ×400) are seen in the lung. (g,h) Necrotizing lymphadenitis is present in the lymph node around the abdominal aorta (g, ×40; h, ×400), and (i) hemophagocytosis is also observed (i, ×400). (j) The liver shows lobular necroses and mild portal fibrosis (×100). (k) IHC of the lymph node (×400) shows numerous SFTSV-NP-positive cells and positive signals for the SFTSV-NP antigen in the cytoplasm of atypical lymphoid cells. (l) IHC of the liver shows SFTSV-positive cells, but hepatocytes were negative for the SFTSV-NP antigen (×100).
hemorrhage and infarction (Fig. 2b,c), with pleural effusions (Lt.: 1100 mL, Rt.: 1200 mL). The bilateral lungs showed marked pulmonary hemorrhage, edema with DAD (Fig. 2d), numerous Mucor infection-forming parenchymal lesions, angio-invasion with thrombosis, and infarction (Fig. 2e,f). Necrotizing lymphadenitis, which was similar to that of the left inguinal node of case 1, with some hemophagocytes was observed in the systemic lymph nodes (Fig. 2g–i). The liver (1012g) showed multiple lobular necroses, mainly in zone 2, and mild portal fibrosis (Fig. 2j). The CNS was not available for analysis.

Eighteen tissues were analyzed using IHC (Table 3). SFTSV-NP antigen-positive atypical lymphoid cells were detected in all the organs examined except for the trachea, and the levels were especially high in both the systemic lymph nodes (Fig. 2k) and spleen. The parenchymal cells of each organ, including hepatocytes (Fig. 2l), were negative for the SFTSV-NP antigen. Consistent with the IHC, high copies (45–266000 copies/cell) of SFTSV-RNA were detected in all of the samples, including the systemic lymph nodes and spleen (Table 3).

**DISCUSSION**

The main pathological finding of SFTSV infection was necrotizing lymphadenitis with both numerous apoptotic cells and nuclear debris. However, there was a big difference in the locations of the necrotizing lymphadenitis in our cases. Case 1 had necrotizing lymphadenitis positive for the SFTSV-NP antigen only in the left inguinal lymph node and palatine tonsil. In contrast, Case 2 had necrotizing lymphadenitis in the systemic lymph nodes, and numerous SFTSV-NP antigen-positive cells were present in all examined organs. There may be a large difference in the viral infection load between our two cases i.e. significantly higher SFTSV amounts in Case 2 than

| Tissue                  | IHC (anti-SFTSV-NP) | RT-PCR (SFTSV-RNA, copy/cell) |
|-------------------------|---------------------|-------------------------------|
|                         | Case 1              | Case 2                        | Case 1   | Case 2                      |
| Cerebrum                | –                   | –                             | –        | 9.14 × 10^4                 |
| Cerebellum              | –                   | –                             | –        | 4.50 × 10^4                 |
| Mid brain               | –                   | –                             | –        | –                            |
| Pons                    | –                   | –                             | –        | –                            |
| Spinal cord             | –                   | –                             | –        | 8.99 × 10^4                 |
| Stomach                 | –                   | +                             | –        | 5.72 × 10^4                 |
| Colon                   | –                   | +                             | –        | 4.35 × 10^4                 |
| Appendix vermiformis    | –                   | +++                           | –        | 1.13 × 10^4                 |
| Pancreas†               | –                   | +++                           | –        | 1.27 × 10^4                 |
| Spleen                  | –                   | +++                           | –        | 5.40 × 10^3                 |
| Heart                   | –                   | +                             | –        | 3.99 × 10^2                  |
| Liver                   | –                   | +++                           | –        | 6.85 × 10^3                 |
| Left kidney             | –                   | +                             | –        | 4.11 × 10^3                 |
| Thyroid gland           | –                   | +                             | –        | 6.58 × 10^2                 |
| Adrenal gland           | –                   | ++                            | –        | 1.46 × 10^2                  |
| Uterus                  | –                   | –                             | –        | –                            |
| Ovary                   | –                   | –                             | –        | –                            |
| Urinary bladder         | –                   | +                             | –        | 3.93 × 10^1                 |
| Palatine tonsil         | +                   | –                             | –        | 2.43 × 10^2                 |
| Trachea                 | –                   | –                             | –        | 6.70 × 10^3                 |
| Esophagus               | –                   | +                             | –        | –                            |
| Bone marrow             | –                   | –                             | –        | –                            |
| Pituitary gland         | –                   | –                             | –        | –                            |
| Aorta                   | –                   | +                             | –        | 6.82 × 10^2                 |
| Gallbladder             | –                   | ++                            | –        | 1.46 × 10^2                  |
| Testis                  | –                   | +                             | –        | –                            |
| Lymph node              | –                   | +++                           | –        | 2.66 × 10^3                 |
| Mediastinum             | –                   | +++                           | –        | 1.34 × 10^3                 |
| Left lung hilum         | –                   | +++                           | –        | –                            |
| Right lung hilum        | –                   | –                             | –        | –                            |
| Left inguinal region    | ++                  | –                             | –        | 1.14 × 10^2                 |
| Right inguinal region   | –                   | +                             | –        | 1.59 × 10^2                 |
| Intraperitoneum         | –                   | +++                           | –        | 5.74 × 10^3                 |
| Paraabdominal aorta     | –                   | +++                           | –        | 1.16 × 10^5                 |

Blank spaces indicate that IHC and TR-PCR were not done on those tissues.

The results were graded as follows: −, no positively-stained cells; +, under 10 cells; ++, 10–100 cells; ++++, 100–500 cells; ++++, more than 500.

†including para-pancreatic lymph nodes.
that in Case 1. We hypothesize that the discrepancy between
the number of SFTSV-NP-positive cells and the SFTSV-RNA
levels may be due to the influence of the SFTSV-infected cells
in the peripheral blood. There were no pathological changes,
SFTSV-NP-positive cells, or SFTSV-RNA detected in the CNS
of Case 1.

A retrospective study of 115 hospitalized SFTS patients
found that 33.7% of the patients had abnormalities suggest-
ive of pneumonia in either the chest X-ray or CT.11 Our
cases showed pulmonary mycosis that was not detected
previously. Particularly in Case 1, the aggressive pulmonary
aspergillosis exacerbated the clinical course resulting in
death, although the thrombocytopenia and the other organ
pathologies were improving. SFTS patients are somewhat
immunodeficient6 or have damage to their immune system
with lower levels of CD3+ and CD4+ T lymphocytes,7,8
which may be due to a viral-associated hemophagocytic
syndrome, playing an important role in disease progression,
disease severity, and clinical outcome. Also in our cases,
the total number of the lymphocytes decreased
(Tables 1,2). These findings suggest that physicians should
be aware of potential fungal infection in SFTS patients. In
both of our cases, the patients had received corticosteroids
for the hemophagocytosis before admission to our hospital.
Some lethal SFTS cases have reported a history of early
treatment with dexamethasone, which acts to repress
immune functions.2 Since use of corticosteroids for SFTS
patients is controversial9,12 a prospective study is needed
to evaluate this.

The liver plays a central role in the pathogenesis of
SFTSV infection.1,5 In both of our patients, elevation of liver
damage markers (AST, ALT, and LDH) was observed, as
found in a majority of the patients.11 Especially in Case 2,
marked increases of AST, ALT, and LDH were observed at
the time of death, and histologically the liver showed mul-
tiple lobular necroses. Viruses belong to the family
Bunyaviridae, the Crimean-Congo hemorrhagic fever virus,
and the Rift Valley fever virus show extensive hepatocellu-
lar necroses with necrotic hepatocytes infected with the
viruses.13,14 In SFTS, however, hepatocytes were not
infected with SFTSV, although numerous SFTSV-NP
antigen-positive cells were observed in the liver. Therefore,
it suggests that the liver damage in SFTS may be due to a
secondary pathological process (e.g. shock status, hypercytokinemia, and hemophagocytosis) rather than due
to a direct disturbance by SFTSV, but the pathogenesis is
unknown.

In conclusion, the pathognomonic histological features in
SFTS patients are necrotizing lymphadenitis in the systemic
lymphoid tissue centered on the local lymph node close to
the tick-bite region. Since the cellular immune function may
be suppressed in SFTS patients, physicians should be aware
of potential fungal infections.

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REFERENCES

1 Yu XJ, Liang MF, Zhang SY et al. Fever with thrombocyto-
penia syndrome associated with a novel bunyavirus in China. N En
giJ Med 2011; 364: 1523–32.
2 Gai Z, Liang M, Zhang Y et al. Person-to-person transmis-
sion of severe fever with thrombocytopenia syndrome bunyavirus
through blood contact. Clin Infect Dis 2012; 54: 249–52.
3 Liu Y, Li Q, Hu W et al. Person-to-person transmission of severe
fever with thrombocytopenia syndrome virus. Vector Borne Zoo-
notic Dis 2012; 12: 156–60.
4 Tang X, Wu W, Wang H et al. Human-to-human transmission of
severe fever with thrombocytopenia syndrome bunyavirus
through contact with infectious blood. J Infect Dis 2013; 207:
736–9.
5 Takahashi T, Maeda K, Suzuki T et al. The first identification and
retrospective study of severe Fever with thrombocytopenia syn-
drome in Japan. J Infect Dis 2013; 209: 816–27.
6 Savage HM, Godsey MS Jr, Lambert A et al. First detection of
heartland virus (Bunyaviridae: Phlebovirus) from field collected
arthropods. Am J Trop Med Hyg 2013; 89: 445–52.
7 Sun L, Hu Y, Niyonsaba A et al. Detection and evaluation of
immunofunction of patients with severe fever with thrombocyto-
penia syndrome. Clin Exp Med 2013; doi:10.1007/s10238-013-
0259-0.
8 Weng Y, Chen N, Han Y, Xing Y, Li J. Clinical and laboratory
characteristics of severe fever with thrombocytopenia syndrome
in Chinese patients. Braz J Infect Dis 2013; 18: 88–91.
9 Zhang YZ, He YW, Dai YA et al. Hemorrhagic fever caused by
a novel Bunyavirus in China: Pathogenesis and correlates of
fatal outcome. Clin Infect Dis 2012; 54: 527–33.
10 Kuramochi H, Hayashi K, Uchida K et al. Vascular endothelial
growth factor messenger RNA expression level is preserved in
liver metastases compared with corresponding primary
colorectal cancer. Clin Cancer Res 2006; 12: 29–33.
11 Deng B, Zhou B, Zhang S et al. Clinical features and factors
associated with severity and fatality among patients with severe
fever with thrombocytopenia syndrome bunyavirus infection in
Northeast China. PLoS ONE 2013; 8: e80802.
12 Deng B, Zhang S, Geng Y et al. Cytokine and chemokine levels
in patients with severe fever with thrombocytopenia syndrome
virus. PLoS ONE 2012; 7: e41365.
13 Whitehouse CA. Crimean-Congo hemorrhagic fever. Antiviral
Res 2004; 64: 145–60.
14 Shieh WJ, Paddock CD, Lederman E et al. Pathologic studies on
suspect animal and human cases of Rift Valley fever from an
outbreak in Eastern Africa, 2006–2007. Am J Trop Med Hyg
2010; 83: 38–42.