

Myelodysplastic syndrome after COVID-19 infection in a living-donor kidney transplant recipient: a case report and literature review

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Case Report

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Abstract

Background

Myelodysplastic syndrome (MDS) after kidney transplantation is rare, and its association with coronavirus disease 2019 (COVID-19) is unclear.

Case report

A 57-year-old man was treated with maintenance hemodialysis for end-stage kidney disease presumed to have been caused by chronic glomerulonephritis diagnosed 10 years earlier. After 1 year, he received an ABO-compatible living-donor kidney transplant from his sister. Renal graft function remained stable thereafter, with serum creatinine levels ranging from 1.49 to 1.62 mg/dL. Two years before presentation, he developed COVID-19, which resolved after administration of molnupiravir. However, following the COVID-19 infection, nucleated red blood cells and immature myeloid cells were persistently observed in his peripheral blood. Laboratory analyses showed hemoglobin 16.3 g/dL, white blood cell count 5,700/ μ L, neutrophil count 2,896/ μ L and platelet count 117,000/ μ L. Bone marrow aspiration revealed 1.4% myeloblasts, dysplastic erythroid cells with nuclear atypia, and megakaryocytes with poor platelet production and release, leading to a diagnosis of MDS. Cytogenetic analysis revealed trisomy 8, and Wilms' tumor 1 messenger RNA (WT1 mRNA) expression was elevated to 690 copies/ μ gRNA, suggesting a risk of leukemic transformation. Although hematopoietic stem cell transplantation was considered because of his age, no suitable related donor was available, as the kidney donor sister had developed rheumatoid arthritis. Registration with the bone marrow donor program is currently under consideration.

Conclusion

We experienced a case of MDS diagnosed after COVID-19 in a kidney transplant recipient. Persistent appearance of immature blood cells after COVID-19 infection may warrant careful hematological evaluation in immunocompromised patients, including kidney transplant recipients.

Background

Myelodysplastic syndrome (MDS) is caused by genetic abnormalities in hematopoietic stem cells that lead to production of morphologically abnormal blood cells (dysplasia), impaired blood cell function, and a decrease in the number of normal blood cells. In addition, some patients with MDS exhibit increased numbers of immature blood cells (blasts), which can progress to acute myeloid leukemia. MDS is therefore considered a preleukemic condition. The incidence of MDS increases with advancing age and rises markedly in individuals aged 70 years or older; thus a rising incidence of MDS has been noted in aging societies. Also at greater risk of developing MDS are individuals who have received radiation

therapy or anticancer drug therapy. In recent years, two conditions, clonal hematopoiesis of indeterminate potential (CHIP) and clonal cytopenia of undetermined significance (CCUS), have become widely recognized as precursors to MDS. CHIP is defined as the presence of somatic mutations commonly associated with myeloid neoplasms, with a variant allele frequency of 2% in individuals with normal peripheral blood cell counts. By contrast, CCUS is characterized by persistent cytopenia with evidence of clonal hematopoiesis, though it does not fulfill the diagnostic criteria for MDS.

Case report

A 57-year-old Japanese man who had undergone living-donor kidney transplantation was referred to the hematology department of a public hospital for further evaluation of persistent abnormalities in peripheral blood findings. The patient had no subjective symptoms at presentation. His past medical history included a gastroduodenal ulcer of unknown onset. Ten years before the current presentation, the patient began maintenance hemodialysis because of end-stage kidney disease considered secondary to chronic glomerulonephritis. Nine years before presentation, he underwent ABO-compatible living donor kidney transplantation from his elder sister and received immunosuppressive therapy with basiliximab, tacrolimus, mizoribine and steroids. Prior to the transplantation, C9 deficiency had been suspected, and he had received meningococcal vaccination. Two years after kidney transplantation, the patient developed microscopic hematuria, and an allograft kidney biopsy performed at the transplant center revealed CN1 nephrotoxicity, prompting a reduction in the tacrolimus dose and the addition of everolimus. Thereafter, his serum creatinine level remained stable at 1.49–1.62 mg/dL, and allograft renal function was well preserved.

Two years before the diagnosis of MDS, the patient developed coronavirus disease 2019 (COVID-19). A nasopharyngeal swab SARS-CoV-2 PCR test was positive, though an antigen test was negative. Chest radiography and computed tomography revealed mild pulmonary involvement (Fig. 1). Although oral molnupiravir was administered, fever persisted and the patient was ultimately hospitalized but recovered with intravenous remdesivir therapy. There have been no changes in his medications since his COVID-19 infection. His current medications are as follows: extended-release tacrolimus 3 mg/day, everolimus 1.5 mg/day, mizoribine 300 mg/day, predonine 5 mg/day, vonoprazan 10 mg/day, fluvastatin 30 mg/day, ezetimibe 10 mg/day, pemafibrate 0.2 mg/day, Jardiance 25 mg/day, mecobalamin 1.0 mg/day and magnesium oxide 1,000 mg/day. However, after his COVID-19 infection, peripheral blood examinations occasionally revealed nucleated red blood cells, metamyelocytes and myelocytes. In January 2026, laboratory examination revealed a white blood cell count of 5,700/ μ L with a neutrophil count of 2,896/ μ L, hemoglobin level of 16.3 g/dL, and platelet count of 117,000/ μ L. A peripheral blood smear showed nucleated erythrocytes at 6/100 white blood cells, metamyelocytes at 1.5% and myelocytes at 6.0%. Serum creatinine was 1.60 mg/dL and estimated glomerular filtration rate was 36.4 mL/min/1.73m². Immunoglobulin levels were within preserved ranges: IgG 1,097 mg/dL, IgA 257 mg/dL and IgM 83 mg/dL. The trough concentrations of tacrolimus and everolimus were 5.38 ng/mL and 4.8 ng/mL, respectively. Because of the persistent presence of immature myeloid cells in his peripheral blood

together with thrombocytopenia (Fig. 2), the patient was referred to the hematology department of a tertiary care hospital for further evaluation. Bone marrow aspiration revealed normocellular marrow with a nucleated cell count of 14,000/ μ L. However, dysplastic erythroid lineage cells with nuclear atypia were observed, and megakaryocytes showed poor platelet production and release. Blast cells accounted for 1.4% of marrow cells (Table 1). Differential marrow counts showed myelocytes at 14.6%, metamyelocytes at 6.4%, band neutrophils at 19.6%, segmented neutrophils at 9.0%, polychromatic erythroblasts at 22.0% and orthochromatic erythroblasts at 9.4%. WT1 mRNA was elevated to 690 copies/ μ g RNA. Results of additional investigations for parvovirus B19-IgM antibody, cytomegalovirus (CMV) DNA, human herpesvirus 6 (HHV6) DNA, and human T-cell leukemia virus type 1 (HTLV) antibody were all negative. Epstein-Barr virus (EBV) DNA was below detectable levels ($< 2 \times 10^2$ copies/mL). Serum copper and zinc levels were 78 μ g/dL and 60 μ g/dL, respectively (Table 1). Based on the bone marrow findings and cytogenetic abnormalities MDS was suspected. Subsequent chromosomal analysis revealed 47, XY, + 8 in 10 analyzed metaphases (Fig. 3). No high-risk cytogenetic abnormalities associated with MDS, including monosomy 7 or deletion of 7q31, were detected (data not shown). According to the International Prognostic Scoring System (IPSS), the score was 0.51, corresponding to lower-risk disease. The International Prognostic Scoring System-Molecular (IPSS-M) score was 0.25, also indicating lower-risk MDS. A chronological review of the peripheral blood findings revealed that immature myeloid cells first appeared approximately 7 days after the onset of COVID-19, and nucleated erythrocytes, metamyelocytes and myelocytes were observed intermittently upon follow-up examinations. Approximately 598 days after his COVID-19 infection, the patient was definitively diagnosed with MDS, based on bone marrow aspiration findings.

Table 1
Bone
marrow
aspiration
examination
and Special
blood tests

Although the patient’s relatively young age would generally support consideration of allogeneic hematopoietic stem cell transplantation, donor selection posed substantial difficulties. His sister, who had donated a kidney for transplantation, had developed rheumatoid arthritis and was therefore not considered an appropriate stem cell donor. No additional sibling donor candidates were available. Therefore, consultation with hematologists regarding registration with the national bone marrow donor program is currently ongoing.

Discussion

MDS is a heterogeneous group of clonal myeloid neoplasms originating from hematopoietic stem cells. It is characterized by ineffective hematopoiesis, which leads to hematopoietic cell dysplasia as well as to peripheral blood cytopenias, particularly anemia, and a propensity to progress to acute leukemia [1–5]. MDS is more common in men than in women, and its incidence increases with advancing age, with a

median age at diagnosis of over 70 years. The incidence of MDS is estimated to be approximately 4-11.5 cases per 100,000 persons annually in the general population [6–8]. Previous exposure to chemotherapy or radiotherapy is a well-established risk factor for the development of MDS [9, 10]. We report here a rare case of lower-risk MDS that developed after a COVID-19 infection in a recipient of a living donor kidney transplant. Although MDS has previously been reported after solid organ transplantation, its occurrence after kidney transplantation remains uncommon. The GBD 2023 Kidney Failure with Replacement Therapy Collaborators estimated that approximately 1.02 million individuals are living with a functioning kidney transplant worldwide [11]. Assuming a prevalence of MDS of 5–10 per 100,000 population [6–8], it is estimated that approximately 51–102 kidney transplant recipients worldwide have concomitant MDS. Morton et al. analyzed data from 15 population-based cancer registries in the U.S. Scientific Registry of Transplant Recipients. They found that among 207,859 solid organ transplant recipients who underwent transplantation between 1987 and 2009, there were 45 cases of MDS following kidney transplantation [12]. Chronic immunosuppressive therapy, viral infections, chronic antigenic stimulation and genetic susceptibility may contribute to the pathogenesis of post-transplant hematological malignancies. In the present case, immature myeloid cells and nucleated erythrocytes began to appear in peripheral blood samples after COVID-19 infection. Recent studies have suggested that SARS-CoV-2 infection may induce persistent immune dysregulation, chronic inflammation and abnormalities within the bone marrow microenvironment. Moreover, inflammatory cytokine activation and immune-mediated hematopoietic stress associated with COVID-19 could potentially unmask latent clonal hematopoiesis or accelerate the progression of underlying hematologic disorders. Whether COVID-19 directly contributed to the development of MDS in this patient remains uncertain, however.

Our patient exhibited trisomy 8, which is a relatively common cytogenetic abnormality in MDS. Importantly, no chromosome 7 abnormalities associated with a high-risk disease were identified. Both IPSS and IPSS-M classified the patient as lower-risk. However, given that 10–20% of patients with lower-risk MDS eventually progress to acute leukemia, careful long-term follow-up in collaboration with hematologists was deemed essential for this patient, and future consideration of hematopoietic stem cell transplantation may become necessary, depending on disease progression.

Table 2 summarizes the reported three cases of MDS after kidney transplantation. Prischl et al. reported the case of a patient who developed two malignancies following kidney transplantation and subsequently developed secondary MDS following postoperative chemotherapy and radiotherapy, both of which are established risk factors for the development of MDS [13]. In a case that closely resembles ours, Juric et al. described a kidney transplant patient who developed MDS 6 months after COVID-19. However, they also reported reactivation of CMV and EBV following COVID-19, raising the possibility that these viruses may have contributed to the development of MDS [14]. In addition, Ikeda et al. reported a case of MDS that developed 6 years after kidney transplantation from the patient's mother. Thereafter, active/acute antibody-mediated rejection and chronic active antibody-mediated rejection were confirmed. Deterioration of renal function was successfully controlled by resuming mycophenolate mofetil administration and administering local irradiation to the renal allograft. The patient subsequently underwent two allogeneic hematopoietic stem cell transplantations from his older sister, the first using

bone marrow and the second using peripheral blood stem cells. Complete remission of the MDS was achieved following the hematopoietic stem cell transplantations [15]. Several other cases of hematologic disorders occurring after COVID-19 infection have also been reported in patients with prior kidney transplantation [16–19].

Table 2
Reported cases of MDS after kidney transplantation.

Study	Age	Sex	Diseases and/or treatments associated with the development of MDS	Treatment of MDS	Outcome
Prischl et al. [13]	68	Female	Breast cancer: chemotherapy and radiotherapy Urinary tract TCC: surgery followed by radiotherapy and/or chemotherapy	Azacitidine	Died after leukemic transformation
Juric et al. [14]	55	Male	COVID-19 Reactivation of CMV and EBV	Not reported	Not reported
Ikeda et al. [15]	56	Male	Unknown	Two allo-SCTs	Alive in complete remission 474 days after the second allo-SCT
allo-SCT, allogeneic stem cell transplantation; CMV, cytomegalovirus; EBV, Epstein–Barr virus; MDS, myelodysplastic syndrome					
TCC, transitional cell carcinoma.					

Current treatment strategies for MDS are based on risk stratification according to the IPSS-R [20] and IPSS-M [21], which incorporate molecular genetic profiling.

In the present case, the patient was classified as having lower-risk MDS according to both the IPSS-R and IPSS-M and was managed with observation alone. Nevertheless, Jain et al. reported that patients with lower-risk MDS remain at substantial risk for disease progression, with 15.5% eventually progressing to AML [23]. Moreover, even in the absence of AML transformation, anemia, infection and bleeding can profoundly impair quality of life and are associated with inferior overall survival [24, 25]. Bahmani et al. reported that elevated blood levels of WT-1 mRNA are associated with high-risk MDS and disease progression, and that WT-1 mRNA is a useful biomarker for diagnosis, prognostic assessment and disease monitoring [26]. The WT-1 mRNA level was elevated in our patient, indicating that careful long-term follow-up is warranted. Treatment options for MDS include luspatercept for patients with lower-risk disease [27], azacitidine for those who fail to respond adequately to luspatercept [28], and allogeneic hematopoietic stem cell transplantation for patients with progressive disease [29]. Although our patient had lower-risk MDS, he was relatively young (57 years) and was therefore considered a

candidate for allogeneic hematopoietic stem cell transplantation. He is currently being evaluated for registration with the Japan Marrow Donor Program.

Conclusion

We report a case of low-risk MDS that developed after COVID-19 infection in a patient who had previously undergone living-donor kidney transplantation. Although the patient presented with only mild thrombocytopenia and immature cells circulating in his peripheral blood, lower-risk MDS may progress in some patients. Therefore, allogeneic hematopoietic stem cell transplantation is being considered as a potential treatment option.

Declarations

Acknowledgment

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Authors' contributions

RM, KM, and KI were responsible for the clinical management of the patient and participated in the treatment decision-making. RM wrote the main manuscript text. All authors read and approved the final manuscript.

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Availability of data and materials

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Declaration of generative AI in scientific writing

We used GPT-5 as a proofreading tool to correct grammar and sentence structure in the manuscript. All sentences revised by Chat GPT were reviewed and verified by the authors. No content was generated by the ChatGPT or any other AI service.

Ethics approval and consent to participate

The study was conducted in compliance with the Declaration of Helsinki and approved by the ethics committee in the Fujita Memorial Hospital, Fukui, Japan (Approval number: 70).

Consent for publication

Written informed consent was obtained from the present patient.

Competing interests

No authors have conflicts to report.

References

1. Garcia-Manero G. Myelodysplastic syndromes: 2023 update on diagnosis, risk-stratification, and management. *Am J Hematol*. 2023;98:1307–1325.
2. Oster HS, van de Loosdrecht AA, Mittelman M. Diagnosis of myelodysplastic syndromes: the classic and the novel. *Haematologica* 2025;110:300–311.
3. Cazzola M. Myelodysplastic Syndromes. *N Engl J Med* 2020;383:1358–1374.
4. Arber DA, Orazi A, Hasserjian RP, Borowitz MJ, Calvo KR, Kvasnicka HM, et al. International Consensus Classification of Myeloid Neoplasms and Acute Leukemias: integrating morphologic, clinical, and genomic data. *Blood* 2022;140:1200–12
5. Sekeres MA, Taylor J. Diagnosis and Treatment of Myelodysplastic Syndromes. *JAMA* 2022;328:872–880.
6. Ma X. Epidemiology of Myelodysplastic Syndromes. *The American Journal of Medicine* 2012;125:S2-S5.
7. Gou X, Chen Z, Shangguan Y. Global, regional, and national burden of myelodysplastic syndromes and myeloproliferative neoplasms, 1990–2021: an analysis from the global burden of disease study 2021. *Frontiers in Oncology* 2025;15:1559382.
8. Xie S, Yan J, Tse P, Humphries B, Xie F. Burden of Myelodysplastic Syndromes: A Literature Review of Epidemiological and Humanistic Aspects. *Journal of Evidence-Based Medicine* 2025;18:e70065.
9. Sun LM, Lin CL, Lin MC, Liang JA, Kao CH. Radiotherapy- and chemotherapy-induced myelodysplasia syndrome: A nationwide population-based nested case–control study. *Medicine* 2015;94:e737.
10. Zahid MF, Parnes A, Savani BN, Litzow MR, Hashmi SK. Therapy-related myeloid neoplasms - what have we learned so far? *World J Stem Cells* 2016;8:231–242.
11. GBD 2023 Kidney Failure with Replacement Therapy Collaborators. Global, regional, and national prevalence of kidney failure with replacement therapy and associated aetiologies, 1990–2023: a systematic analysis for the Global Burden of Disease Study 2023. *Lancet Glob Health* 2025;13:e1378-e1395.
12. Morton LM, Gibson TM, Clarke CA, Lynch CF, Anderson LA, Pfeiffer R, et al. Risk of myeloid neoplasms after solid organ transplantation. *Leukemia* 2014;28:2317–2323.
13. Prischl FC, Burgstaller S, Wallner M, Seiringer E, Dinkhauser P, Pauer W, et al. Three solid malignancies and a myelodysplastic syndrome with a protracted course after kidney transplantation. *Clin Kidney J* 2013;6:319–321.

14. Juric I, Katalinic L, Furic-Cunko V, Basic-Jukic N. Myelodysplastic syndrome in a kidney transplant recipient after SARS-CoV-2 infection: can SARS-CoV-2 induce myelodysplastic syndrome? *Int Urol Nephrol* 2022;54:1775–1776.
15. Ikeda M, Tsukada N, Chikai H, Tasaki M, Saito K, Nakagawa Y, et al. Successful second allogeneic stem cell transplantation from a sibling donor for relapse of myelodysplastic syndrome in a recipient of a renal transplant from his mother: Case report. *Transplant Proc* 2016;48:3085–3087.
16. Sharifi MJ, Gheibi N, Panahi F, Sharifzadeh S, Nasiri N. MDS-type morphologic abnormalities of peripheral blood granulocytes in symptomatic COVID-19 patients. *International Journal of Hematology-Oncology and Stem Cell Research* 2024;18:249–253.
17. Costa BA, Luz KV, Campos SEV, Lopes GS, Vasconcelos Leitão JP, Duarte FB. et al. Can SARS-CoV-2 induce hematologic malignancies in predisposed individuals? A case series and review of the literature. *Hematology, Transfusion and Cell Therapy* 2022;44:26–31.
18. Qing X, Cai J, Rock A. Myelodysplastic syndrome in a 30-year-old man with coronavirus disease 2019 (COVID-19): a diagnostic challenge. *Autopsy and Case Reports* 2021;11:e2021274.
19. Huerga Encabo H, Grey W, Garcia-Albornoz M, Wood H, Ulferts R, Valle Aramburu I. et al. Human erythroid progenitors are directly infected by SARS-CoV-2: implications for emerging erythropoiesis in severe COVID-19 patients. *Stem Cell Reports* 2021;16:428–436.
20. Greenberg PL, Tuechler H, Schanz J, Sanz G, Garcia-Manero G, Solé F, et al. Revised International Prognostic Scoring System for Myelodysplastic Syndromes. *Blood* 2012;120:2454–2465.
21. Bernard E, Tuechler H, Greenberg PL, Hasserjian RP, Arango Ossa JE, Nannya Y, et al. Molecular International Prognostic Scoring System for Myelodysplastic Syndromes. *NEJM Evid.* 2022;1(7).
22. Rosenberg M, Thapa S, Jeurkar C, Kasner M. Copper Deficiency Mimicking Myelodysplastic Syndrome: Zinc Supplementation in the Setting of COVID-19. *Case Rep Oncol* 2023;16:55–61.
23. Jain AG, Ball S, Aguirre L, Al Ali N, Kaldas D, Tinsley-Vance S, et al. Patterns of lower risk myelodysplastic syndrome progression: factors predicting progression to high-risk myelodysplastic syndrome and acute myeloid leukemia. *Haematologica* 2024;109:2157–2164.
24. Brunner AM, Leitch HA, van de Loosdrecht AA, Bonadies N. Management of patients with lower-risk myelodysplastic syndromes. *Blood Cancer Journal* 2022;12:166.
25. Merz AMA, Platzbecker U. Treatment of lower-risk myelodysplastic syndromes. *Haematologica* 2025;110:330–338.
26. Bahmani F, Rostami S, Alizadehghandforoush N, Safa M, Chahardouli B, Zaker F, et al. WT1 as a Biomarker in Myelodysplastic Neoplasms: Clinical Correlations and Preliminary Data from an Iranian Cohort. *International Journal of Hematology Oncology and Stem Cell Research* 2026;20:1–13.
27. Fenaux P, Platzbecker U, Mufti GJ, Garcia-Manero G, Buckstein R, Santini V, et al. Luspatercept in Patients with Lower-Risk Myelodysplastic Syndromes. *N Engl J Med.* 2020;382:140–151.
28. Komrokji R, Swern AS, Grinblatt D, Lyons RM, Tobiasson M, Silverman LR, et al. Azacitidine in Lower-Risk Myelodysplastic Syndromes: A Meta-Analysis of Data from Prospective Studies. *The Oncologist* 2018;23:159–170.

29. Della Porta MG, Jackson CH, Alessandrino EP, Rossi M, Bacigalupo A, van Lint MT, et al. Decision analysis of allogeneic hematopoietic stem cell transplantation for patients with myelodysplastic syndrome stratified according to the revised International Prognostic Scoring System (IPSS-R). *Leukemia* 2017;31:2449–2457.

Table 1

Table 1 is available in the Supplementary Files section.

Figures

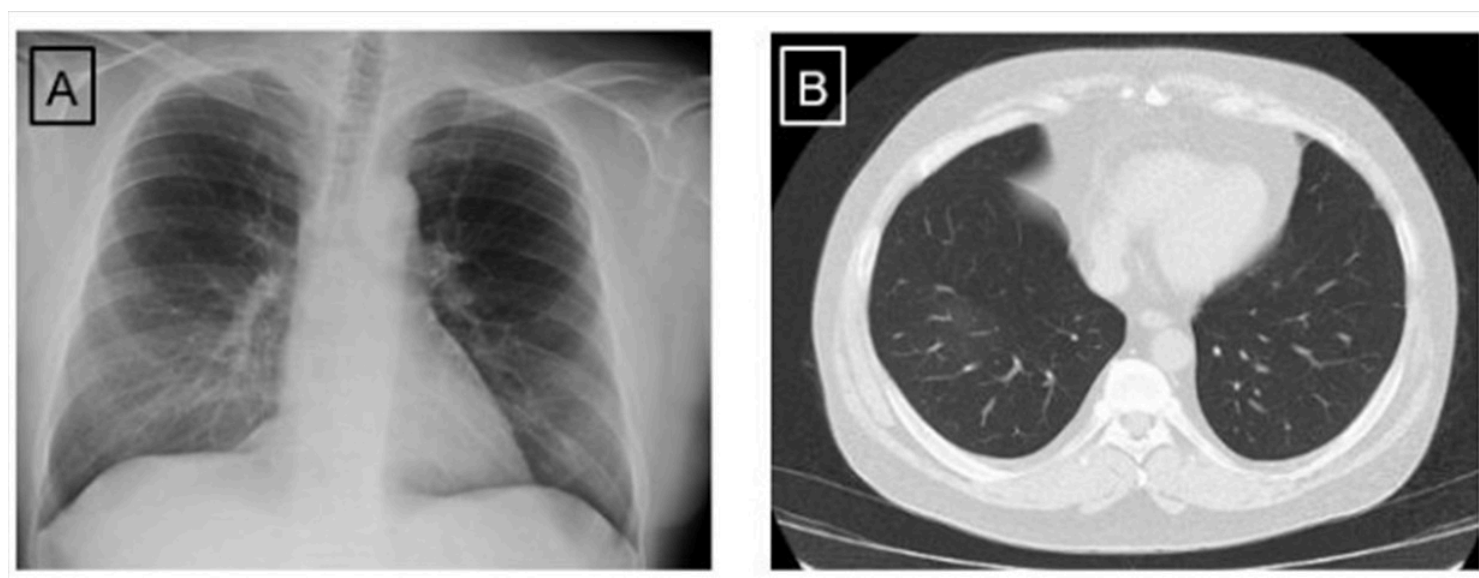
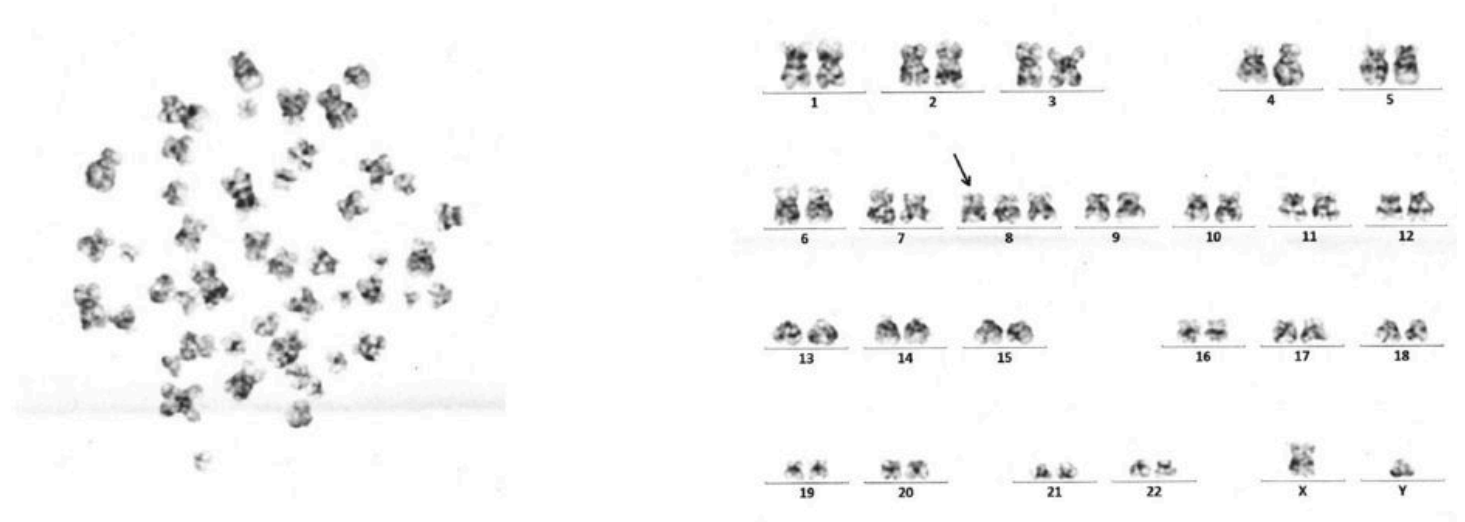


Figure 1

Chest radiograph (A) and concurrent computed tomography (B) obtained at the time of COVID-19 infection. Neither image showed consolidation in either lung, and both findings were unremarkable.



47, XY, +8 [10]

Figure 2

Clinical course. COVID-19 infection was followed by persistent mild thrombocytopenia despite preservation of the neutrophil count. Initially, peripheral blood examinations occasionally revealed nucleated red blood cells, but immature myeloid cells, including myelocytes and metamyelocytes, were subsequently detected. The patient was diagnosed with MDS 1 year and 9 months after COVID-19 infection.

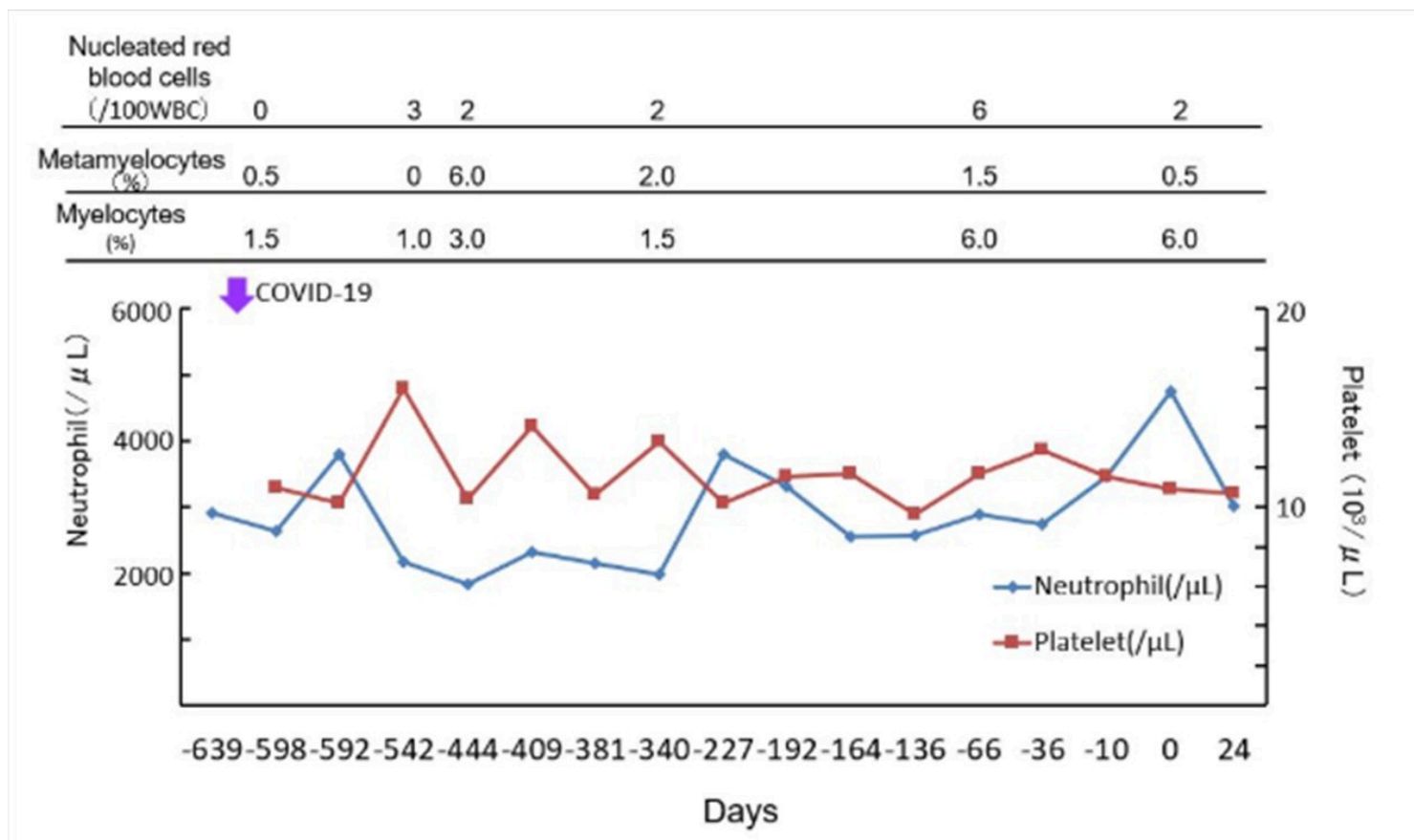


Figure 3

Cytogenetic analysis detected 47,XY,+8 in 10 analyzed metaphases. No high-risk cytogenetic abnormalities associated with MDS, including monosomy 7 or deletion of 7q31, were detected (the data are not shown).

Supplementary Files

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