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The Division conducts clinical, pathological, and therapeutic research on hematological diseases, including hematopoietic tumors. In the field of genomic medicine, which has been developing in recent years, research is also being conducted for clinical application. In collaboration with HGC, our laboratory conducts research on clinical sequencing, curation through artificial intelligence, automation and efficiency of clinical implementation, and clinical significance of clinical sequencing. Specifically, we demonstrated that childhood-onset clonal hematopoiesis is the origin of combined histiocytosis and myeloid tumors through analysis of such cases, proposed a mosaicism hypothesis for TLR8 mutation-induced immunodeficiency in adults, and reported a case of AML with bilateral TP53 mutations derived from somatic mosaic mutations. We also analyzed the significance of reducing mutation risk prior to transplantation in MDS. In ATL, we proposed a novel prognostic indicator utilizing organ infiltration. We also demonstrated for the first time that proviral load increases in carriers and proposed a prognostic model considering both age and proviral load. For histiocytosis, we established a nationwide registry and conducted multiple observational studies. We also compiled and reported the outcomes of the Special-C treatment regimen. Furthermore, we created Japan's first guidelines for adult LCH.

1. Multi-hit Somatic Mosaicism of *TP53* Pathogenic Variants in a Patient Mimicking Li-Fraumeni Syndrome

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Li-Fraumeni syndrome (LFS) is a hereditary cancer predisposition syndrome caused by germline pathogenic variants in *TP53*. Somatic mosaicism of *TP53* has been increasingly recognized, yet its clinical and biological spectrum remains incompletely understood. We report a unique case of multihit somatic mosaicism involving biallelic *TP53* pathogenic variants that clinically mimicked LFS. A 38-year-old woman with a history of anaplastic oligodendroglioma and subsequent therapy-related myelodysplastic neoplasm was found to harbor two hotspot *TP53* pathogenic variants, p.(Tyr220Ser) and p.(Arg273His), initially suspected to be germline because of high variant allele frequencies in bone marrow and buccal swabs. Comprehensive multitissue genomic analysis across derivatives of all three embryonic germ layers revealed highly variable variant allele frequencies, establishing postzygotic somatic mosaicism. Allele-specific expression analysis confirmed that the two variants resided on separate alleles, representing a rare instance of biallelic *TP53* somatic mosaicism. Despite harboring two functionally severe *TP53* variants, the patient exhibited an unexpectedly indolent clinical course, highlighting the critical role of mosaic distribution and gene dosage in shaping disease phenotype. This case expands the spectrum of *TP53*-related disorders and underscores the importance of comprehensive multitissue molecular testing to avoid misdiagnosis and to guide appropriate cancer surveillance strategies.

2. Prognostic impact of organ involvement in aggressive adult T-cell leukemia/lymphoma: definition of risk organ and proposal of a prognostic index

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Adult T-cell leukemia/lymphoma (ATL) is a lymphoma with a poor prognosis. Although organ involvement is common in aggressive ATL, the relationship between specific organ involvement and prognosis remains unclear. This single-center, retrospective study included 140 patients with aggressive ATL. The rates of involvement for each organ were as follows: peripheral blood 78%, bone marrow 67%, lymph nodes 90%, skin 56%, lung 27%, liver 50%, spleen 48%, central nervous system (CNS) 20%, gastrointestinal tract 16%, pleural effusion 11%, ascites 8%, kidney 4%, pancreas 3%, muscle 2%, pleura 2%, and peritoneum 2%. Involvement of the CNS, lung, liver, spleen, and pleura was associated with poorer overall survival (OS) in the univariate analysis; however, only CNS, lung, and liver involvement remained significant in the multivariate analysis. These three organs were therefore defined as risk organs. OS was significantly stratified when patients were grouped by score (0, 1, or ≥2), with 2 points assigned for CNS involvement and 1 point each for lung and liver involvement ($P < 0.001$). This novel prognostic model, the risk organ index for aggressive ATL, showed the highest concordance index among existing prognostic indicators. This study reveals the organ involvement profile in aggressive ATL and highlights its prognostic value.

3. Long-Term Kinetics of Proviral Load in HTLV-1 Carriers: Defining Risk for the Development of Adult T-cell Leukemia/lymphoma

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Background: Assessment of adult T-cell leukemia/lymphoma (ATL) development among human T-lym-

photropic virus 1 (HTLV-1)-infected individuals (carriers) constitute a significant issue. A high HTLV-1 proviral load (PVL) in carriers has been used as a risk factor for ATL development and PVLs are considered to remain unchanged over time among carriers.
Methods: This single-center analysis used a cohort from a prospective observational study of HTLV-1 carriers in Japan (JSPFAD). Carriers whose PVL was measured at least twice between October 2004 and March 2023 were included. We used trajectory analysis to construct a kinetic model of the PVL.
Results: Analysis of 1371 samples from 252 carriers revealed a slight but significant increase in the PVL with age (P<0.001). Trajectory analysis of PVL kinetics classified the carriers into six groups, in three of which increased over time. When we applied the model to 15 carriers who subsequently developed ATL, 12 (80%) were classified into the highest PVL group, with an estimated 15-year ATL development of 47.5% (95% confidence interval: 20.4-74.2%). Notably, younger patients are at greater risk of developing ATL if their PVL values are comparable. Our risk estimation model is available online (https://atlriskpredictor.shinyapps.io/ATL_risk_calculator/).
Conclusions: This study demonstrated that the PVLs increases over time, allowing for prospective risk estimation for ATL development. Further validation is needed to assess the validity of this model.

4. Prognosis of aggressive adult T-cell leukemia/lymphoma with central nervous system infiltration and utility of CD7 versus CADM1 flow-cytometric plots of cerebrospinal fluid

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The prognosis of adult T-cell leukemia/lymphoma (ATL) with primary central nervous system (CNS) involvement has been unclear since the advent of new therapies. Recently, we have shown that flow cytometric CD7/CADM1 analysis of CD4+ cells (HAS-Flow) is useful to detect ATL cells that are not morphologically diagnosed as ATL cells. We investigated the role of CNS involvement in ATL using cytology and HAS-Flow by analyzing cerebrospinal fluid (CSF) from 73 aggressive ATL cases. Based on the findings in CSF, the study subjects were classified into CNS+ (cytologically malignant, n=18), CNS- (cytologically non-malignant and ATL cell population negative in HAS-Flow, n=44), and CNS-Micro (cytologically non-malignant and ATL cell population positive in HAS-Flow, n=11) groups. As expected, the CNS+ group had a shorter overall survival than the CNS- groups (P<0.001). However, the CNS-Micro group showed no adverse impact on overall survival compared to the CNS- group (P=0.506), even without additional CNS-targeted treatments. HAS-Flow also demonstrated clinical utility in the diagnosis of CSF lesions in ATL patients with cerebral white matter lesions and in the detection of ATL cells on post-treatment CSF examination in patients with CNS involvement. Our study demonstrates that ATL with CNS involvement have a poor prognosis and that CSF HAS-Flow is useful to assist in the diagnosis of suspected CNS involvement and to detect ATL cells with high sensitivity after treatment.

5. Common ancestral origin of indeterminate dendritic cell tumor and chronic myelomonocytic leukemia in clonal hematopoiesis

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We report that IDCT, chronic myelomonocytic leukemia (CMML), and hematopoietic stem cells (HSCs) share two major drivers, *IDH2* p.R140Q and *RUNX1* p.S167R. Our results indicate that, although

IDCT and CMML share common driver mutations, clonal divergence had occurred long before disease onset, as revealed by trajectory analysis based on whole-genome sequencing (WGS). This observation suggests that transdifferentiation is unlikely to be the underlying mechanism and instead supports the association of CH-related mutations, such as *IDH2*, with the development of hematopoietic malignancies, including histiocytic neoplasms.

6. Combination therapy of vinblastine, prednisolone, methotrexate and 6-mercaptoprine as a front-line therapy for adult patients with Langerhans cell histiocytosis

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Standard therapy based on risk stratification remains undefined in adult Langerhans cell histiocytosis (LCH), in contrast to pediatric LCH. We report the long-term outcomes, including the prognosis and adverse events (AEs) of 44 patients who underwent first-line chemotherapy with the Special C regimen, consisting of vinblastine, prednisolone, methotrexate, and 6-mercaptopurine. The patient population consisted of 30 patients (68.2%) with multiorgan LCH. The median follow-up period was 81.5 months (range: 16–287 months). The overall response rate was 84.1%. Complete response rate was low in cases with brain or pituitary gland (PG) involvement (p = 0.007 or 0.03, respectively). The five-year overall survival (OS) rate was 94.6% and the 5-year event-free survival (EFS) rate was 52.6%. The presence of additional malignancy was associated with a lower OS rate. Brain involvement at treatment (hazard ratio: 3.36; 95% CI: 1.23–9.15; p = 0.018) was correlated with inferior EFS. The EFS or OS probability at 5 years with or without *BRAF* V600E in plasma cell-free was 28.6% vs. 53.6%, p = 0.11, or 85.7% vs. 100.0, respectively. Grade 3–4 AEs were rare, observed in 8 patients (18.2%) as neutropenia and in 3 patients (6.8%) as febrile neutropenia, with no peripheral neuropathy. As shown in the swimmer plot for the treatment sequence of the 20 patients who relapsed, they were able to survive for a long time after receiving retreatment or salvage therapy. The Special C regimen is useful as initial outpatient chemotherapy for adult LCH without brain involvement.

7. Pre-transplant Genetic Debulking Fails to Improve Outcomes in Myelodysplastic Syndromes

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The Molecular International Prognostic Scoring System (IPSS-M) is widely used for risk stratification in myelodysplastic syndromes (MDS). However, whether genetic debulking achieved by bridging therapy before allogeneic hematopoietic stem cell transplantation (HSCT) improves post-transplant outcomes remains unclear. We retrospectively analyzed patients with MDS who underwent HSCT and had available IPSS-M–related genetic and clinical data. Patients were classified according to the timing of IPSS-M assessment, and changes in IPSS-M category and score following bridging therapy were evaluated. Overall survival (OS) was estimated using the Kaplan–Meier method and compared using the log-rank test. A total of 216 patients were included in the analysis. IPSS-M at diagnosis stratified post-HSCT survival ($p = 0.0075$). IPSS-M score before and after bridging therapy was available in 58 patients. Bridging therapy resulted in a reduction in IPSS-M score (mean change, -0.84). Improvement of the IPSS-M category was observed in 28 patients (48%), and progression in 12 patients (21%). There was no significant difference in OS between IPSS-M category changes ($p = 0.49$). IPSS-M score reduction occurred frequently in patients harboring a *TP53* mutation, while improvement of IPSS-M did not lead to improved OS ($p = 0.89$). Pre-transplant genetic debulking, as reflected by an improvement in IPSS-M, does not translate into improved post-transplant outcomes in MDS.

8. Adult-onset immunodeficiency and bone marrow failure associated with somatic mosaic TLR8 gain-of-function mutation: A case with fatal outcome

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Background: Somatic gain-of-function (GOF) mutations in TLR8 cause inborn errors of immunity characterized by chronic neutropenia, hypogammaglobulinemia, and bone marrow failure, predominantly reported in pediatric male patients with onset before age 20.

Case presentation and functional studies: A 49-year-old woman developed progressive neutropenia at age 42, followed by recurrent colitis, splenomegaly, and polyarteritis nodosa. Despite treatment with G-CSF and cyclosporine A, she experienced recurrent infectious episodes and ultimately died from infection at age 49. Whole genome sequencing revealed a somatic mosaic TLR8 p.P432L mutation (VAF: 18% in peripheral blood, 2% in nail) and a germline DDX41 p.V491I variant of uncertain significance (VAF: 36% in blood, 46% in nail). Bone marrow examination showed agranulocytosis with expanded T-cell population. Compared to previously reported cases, our patient exhibited notably later onset, cutaneous manifestations (polyarteritis nodosa), and initially normal immunoglobulin levels that progressively declined. In vitro luciferase reporter assays using HEK293T cells demonstrated that TLR8 P432L exhibited enhanced NF-κB activation upon stimulation with R848 and guanosine compared to wild-type TLR8. Notably, co-expression of DDX41 (both wild-type and V491I) markedly augmented TLR8 P432L-mediated signaling, particularly in response to PolyU stimulation, while having minimal effect on wild-type TLR8. Cyclosporine A showed minimal inhibitory effects on TLR8-mediated signaling, whereas adalimumab did not suppress the intracellular NF-κB activation.

Conclusions: This case represents the first reported adult-onset, female presentation of TLR8 GOF-associated immunodeficiency with fatal outcome. Our findings suggest a novel functional interaction between TLR8 GOF variants and DDX41 in modulating innate immune signaling, potentially explaining the unique phenotype in patients harboring both variants. The co-occurrence of these mutations in nucleic acid-sensing pathways may contribute to disease severity and warrants further investigation as a potential digenic mechanism in immune dysregulation.

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