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Case report

Immunodeficiency-related high-grade B-cell lymphoma with 11q aberration: Further evidence for a lymphoma entity from a patient with simultaneous papillary renal cell carcinoma following pediatric kidney transplant

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ABSTRACT

Various aggressive lymphomas entities have been associated with immunodeficiency. To provide further evidence that also *MYC*-negative high-grade B-cell (formerly Burkitt-like) lymphoma with 11q aberrations comprises an immunodeficiency-related subtype, we here conducted a comprehensive pathological and genetic workup of a 25-year-old patient with this type of lymphoma and simultaneous papillary renal cell carcinoma. The patient developed both malignancies following extensive childhood immunosuppression and a kidney transplant. Germline and somatic genetic analyses included interphase cytogenetics, imbalance mapping, and exome sequencing. We identified potential germline-predisposition to inborn errors of immunity, kidney disease, and cancer, along with a germline region of homozygosity in 20q. Each tumor showed imbalances and single nucleotide variants typical for the respective diagnosis, with shared gains in the name-giving region in 11q, gain of the *MYC* gene in 8q24 and trisomy 12. While we can show that the imbalances in 8q and 11q arise from different mechanisms in both tumors, trisomy 12 involved gain of the same parental chromosome. Our findings corroborate the existence of a subtype of immunodeficiency-related high-grade B-cell lymphomas with 11q aberrations, provide further insights into its molecular pathogenesis, and reveal potential pitfalls in the molecular diagnosis of simultaneous tumors based on the technology applied.

1. Introduction

Historically, Burkitt lymphoma (BL) has been a leading model disease in somatic cancer genetics and virus-driven tumorigenesis [1,2]. It is now widely accepted that the *IG*::*MYC* translocation is the genetic hallmark of BL [3]. Nevertheless, a small subset of high-grade B-cell lymphomas with features of BL remains which bona fide lacks a genomic event targeting the *MYC* gene. Recently, it has been shown that a substantial subset of these cases carries a distinct pattern of chromosome 11 aberration, specifically a gain in 11q23 and a more telomeric loss in 11q [4–6]. These cases were named "Burkitt like lymphoma with 11q aberration" in the 2016 edition of the WHO classification [7]. Despite their morphological similarities to BL, the genetic alterations of these cases with 11q aberration differ substantially from those of BL [4–6]. Thus, the recent WHO classification designated them as *MYC*-negative high-grade B-cell lymphomas with 11q aberrations (HGBCL-11q) [5,8].

IG::*MYC* carrying BL comprises three subgroups: sporadic, endemic, and immunodeficiency-related BL [7]. In the latter subgroup, the

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immunodeficiency can be inborn or acquired. Remarkably, already the first description of HGBCL-11q by our group reported the disease in a patient with an inborn error of immunity (IEI), specifically ataxia telangiectasia [5]. Subsequently, Ferreiro et al. provided evidence that the typical 11q aberration pattern is particularly frequent in BL in immunodeficient hosts. Indeed, these authors demonstrated that the typical 11q pattern is significantly more frequent in BL occurring in the context of transplantation and immunosuppression (43 % of all post-transplant and 60 % of EBV-negative cases) than in immunocompetent patients (3 %) [9].

Here, we report another case of a patient developing HGBCL-11q after a childhood kidney transplant, presenting with simultaneous papillary renal cell carcinoma (pRCC) of the kidney.

2. Case report

We report a case of a 25-year-old male renal transplant recipient with developmental delay and hypogonadism. He had suffered from lupus nephropathy since early childhood. Despite immunosuppressive therapy starting at age 8, his condition worsened over time, ultimately leading to renal insufficiency. At age 17, the patient received a deceased-donor kidney transplant (male), followed by standard triple immunosuppressive maintenance therapy (tacrolimus, mycophenolate mofetil, and steroids). The graft functionality was excellent, with average creatinine values of 1.2 mg/dl (ClCr 80 ml/min). At presentation, a routine abdominal echography, followed by a total body CT scan with contrast and PET scan, demonstrated a mass in the right native kidney and massive lymphadenopathy.

A biopsy of the kidney mass showed a type 2 pRCC. Additionally, a biopsy of a cervical lymph node was performed to exclude metastasis from the pRCC. Remarkably, histologic and cytogenetic analyses revealed the diagnosis of a HGBCL-11q. Bone marrow biopsy and cerebrospinal fluid analysis were negative for lymphomatous involvement. The clinical workup revealed Ann Arbor stage IIIA disease within the IPI 2 with an increased LDH risk group. The patient received for his lymphoma two cycles of rituximab, cyclophosphamide, vincristine, doxorubicin and methotrexate (R-CODOX-M regimen) and two cycles of rituximab, ifosfamide, etoposide and cytarabin (R-IVAC regimen). Control PET and CT imaging five month after diagnosis were negative. Seven months after diagnosis, robot-assisted right nephrectomy surgery was performed. At the time of writing (approximately four years after diagnosis), the lymphoma appears to be in remission, and the follow-up of the kidney cancer is negative. Kidney function was normal at the last follow-up (Fig. S1).

3. Materials and methods

3.1. Histopathology

Lymph node and renal tissues were fixed in formalin and embedded in paraffin. They were stained with HE and with Giemsa staining for lymphoma. Immunohistochemical staining for both lymphoma and pRCC diagnosis using a panel of antibodies listed in Table S1 and in situ hybridization for EBV-encoded small RNAs was performed on a Ventana BenchMark ULTRA (Roche Diagnostics) following the manufacturer's instructions.

3.2. Fluorescence in situ hybridization (FISH)

FISH on FFPE sections of the tumors was conducted according to standard procedures [10] using commercial probes listed in Table S1 as well as recently described double-color double-fusion probes for the detection of IGK::MYC and IGL::MYC fusions as well as for the typical 11q aberration pattern [5,11,12].

3.3. Genome-wide copy number and loss-of-heterozygosity mapping using OncoScan

DNA from tumor tissues was extracted using the GeneRead DNA FFPE Kit (Qiagen, Hilden, Germany) and from blood using MagCore Genomic DNA Whole Blood Kit (Diatech-Pharmacogenetics, Jesi, Italy). DNA was hybridized to an OncoScan CNV assay and analysis was performed using the Chromosome Analysis Suite Software version 4.0 (Thermo Fisher Scientific, Waltham, MA, USA). Only copy number alterations larger than 50 kb, encompassing at least 20 informative probes with a median log2ratio of > 0.2 or < -0.2 and copy number neutral losses of heterozygosity larger than 5 Mb were considered for further analyses. All variants were manually inspected and curated considering the B-allele-frequency and the sample quality (Table S2/3).

3.4. Tumor and germline exome sequencing

Exome sequencing was performed on 50 ng of genomic DNA from FFPE tissue (tumors) or whole blood using the NextSeq High Output Kit v2.5 (300 cycles; Illumina: Nextera™ Exome Kit) and sequenced on a NextSeq platform. Data was analyzed with Varvis-Software Version 1.18.4 (Limbus Medical Technologies GmbH, Rostock). Analysis was performed using the hg19 genome version.

All variants were filtered on read coverage of ≥ 20 reads and a minor allele frequency (MAF) of ≤ 5 %. Additionally, all synonymous variants and variants, which were reported as benign in ClinVar were excluded. Only coding exonic regions of the genes and 20 bp of the flanking intronic regions were evaluated.

Germline variants were additionally filtered on the quality index MedQ and excluded when present in homozygous state in gnomAD. Somatic variants were further filtered based on read quality (Quality score ≥ 150) and variant allele frequency (VAF) of ≥ 10 %.

All remaining variants were matched to Human Phenotype Ontology (HPO) terms using varvis, annotated using ANNOVAR [13] and are reported in Table S4 (germline) and Table S5 (somatic).

4. Results

4.1. Histopathology

The histological examination of the cervical lymph node biopsy revealed sheets of medium to large-sized tumor cells with fine or vesicular chromatin, one or multiple prominent nucleoli, frequent mitoses, and few tingible body macrophages with 'starry sky' appearance. Tumor cells were diffusely positive for CD20, CD10, BCL6, Ki67 (almost 100 %) and MYC protein expression but negative for TDT, BCL2, and Cyclin D1 (Fig. S2). Although more than 40 % of cells were positive for MYC immunostaining, the staining intensity was weaker than typical for BL, suggesting the diagnosis of HGBCL-11q. EBER ISH for EBV was negative (Fig. S2).

Gross and microscopic examination of the recipient-derived renal mass biopsy revealed a pigmented pRCC with type 2 papillary architecture (nuclear grade 3, pT3a TNM eighth edition)[14]. The immunophenotype was positive for CKAE1-AE3, CD10, CK7, AMACR, HMB45, MELAN-A, TFE3 and PAX8. The morphology and immunophenotype were suspicious for a Microphthalmia family of transcription factors associated RCC (MiT family) (Fig. S3).

4.2. Interphase cytogenetics

FISH analyses of the lymph node biopsy showed no evidence of a breakpoint in the *MYC* locus (8q24) or any kind of *IG*::*MYC* fusion, but indicated a gain at the *MYC* locus. Aberrations affecting the *BCL2* (18q21) or *BCL6* (3q27) loci were absent. Signal patterns indicating a gain in 11q23.3 and a loss in 11q24 were present in about 80 % of nuclei (Fig. 1a). In the papillary RCC, FISH analysis showed an average of three

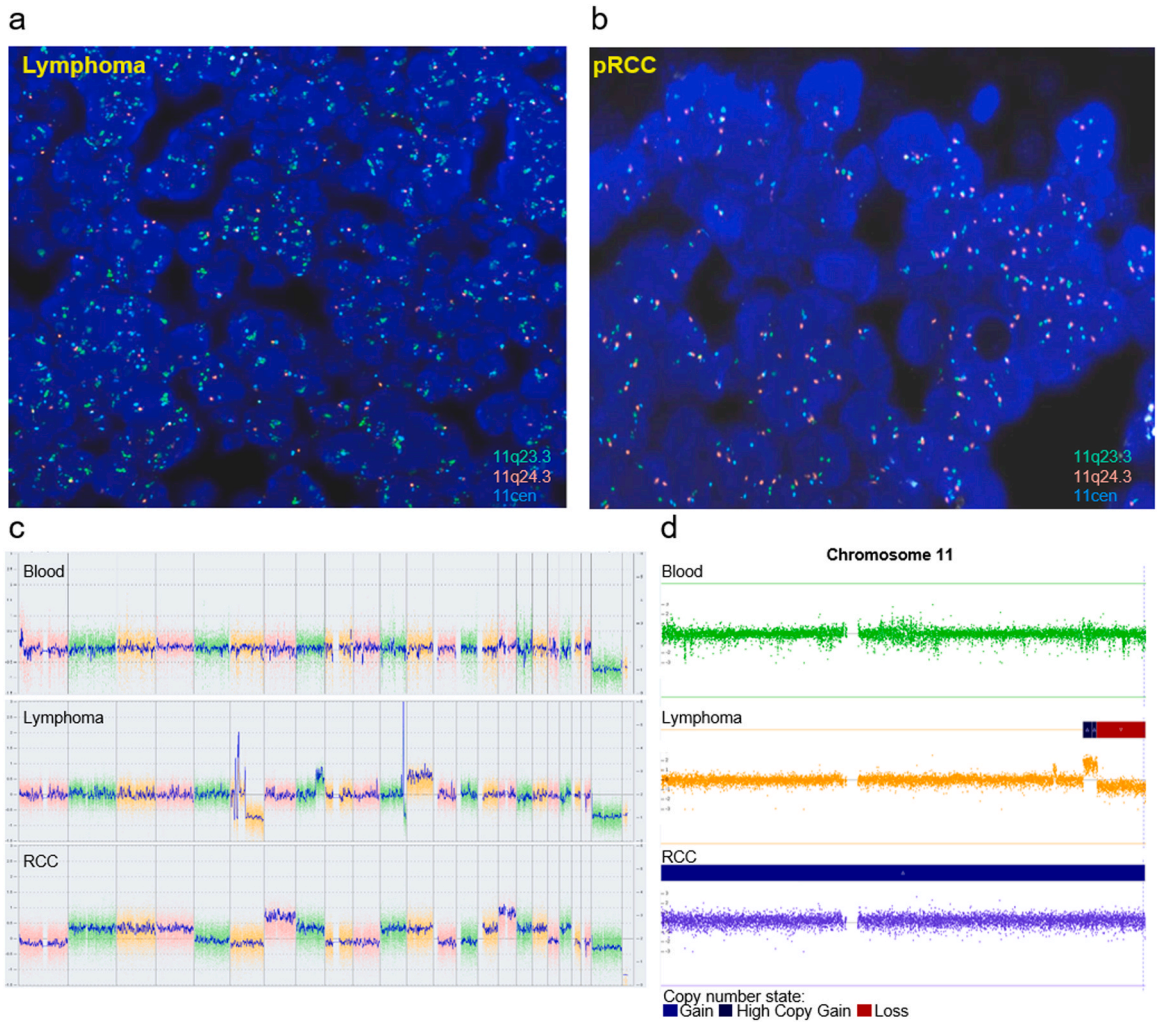

Fig. 1. Cytogenetic and copy-number analyses of the lymphoma and the pRCC. a, FISH analysis of the cervical lymph node biopsy using the 11q gain / loss triple color probe (Bio-Optica) showed clustered green signals in the 11q23.3 region but only one orange signal in the 11q24.3 region, confirming the presence of the typical loss pattern of gain 11q. The centromeric region of chromosome 11 is indicated by the blue signal. b, FISH analysis of the pRCC using 11q gain/loss triple color probe showed 2–3 signals for all regions on chromosome 11 investigated. c, OncoScan analysis showing whole genome views of copy-number alterations in the blood, the lymphoma and the pRCC sample. d, Copy number states of chromosome 11 of blood, lymphoma and pRCC sample indicating a balanced genotype in the blood, the typical gain/loss pattern in the lymphoma and trisomy of the complete chromosome 11 in the pRCC.

copies for all probes on chromosome 11 (Fig. 1b).

Array-based copy number analyses were consistent with the FISH analyses and showed a gain of whole chromosome 11 in the papillary RCC, in contrast to amplification (7–8 copies) in 11q23.3q24.1 and a loss in 11q24.1qter (1 copy) in the lymphoma sample (Fig. 1c,d, Fig. S4/5). The regions of imbalance in 11q in the lymphoma sample contained previously discussed candidate genes in HGBCL-11q [4,5] (Table S2). Thus, both tumors share a gain in 11q23.3q24.1, but due to different mechanisms. Whereas the pRCC displays multiple gains and losses of whole chromosomes, as is typical for this entity, the lymphoma exhibits a more restricted set of additional imbalances, including complex rearrangements of chromosome 6, a gain in 8q22q24 containing the *MYC* gene, trisomy 12, and a copy-neutral loss of heterozygosity encompassing major parts of chromosome 17, including *TP53*.

No clinically relevant imbalance was detected in the blood sample of the patient. Nevertheless, it is remarkable that all samples shared a long stretch of homozygosity of over 5 Mb in 20p11.23p12.2 (Genes in Table S3).

4.3. Exome sequencing

Combined exome analyses revealed 88 sequencing variants shared between both tumors and the blood sample. Three variants were detected in the blood and one of the tumors. All these variants were likely of germline origin (Table S4). Besides a heterozygous class 3 variant in the tumor predisposition gene *MUTYH*, the patient carried several class 3 variants in genes linked to IEI (*C8B, MS4A1, C1QB, DOCK8*) or kidney disease (*COL4A4, MST1*). No relevant germline variants were detected in genes located in the LOH region at 20p11.23p12.2.

Variants likely impacting protein function and specific to the lymphoma - and, thus, considered to be somatic - included genes recurrently mutated in HGBCL-11q, such as *PIK3R1* and *TP53* [15], as well as *SETD2* and *MTA3*, which encode a BCL6-associated transcriptional co-repressor in germinal center B cells (GCB) and lymphomas [16] (Table S5). Among the 40 variants exclusively found in the pRCC, a class 3 variant was identified in *EPHA5*, which has been associated with tumor stage in ccRCC [17].

As described above, the only chromosomal aberration truly in common between both malignancies was trisomy 12 (i.e. gain of whole

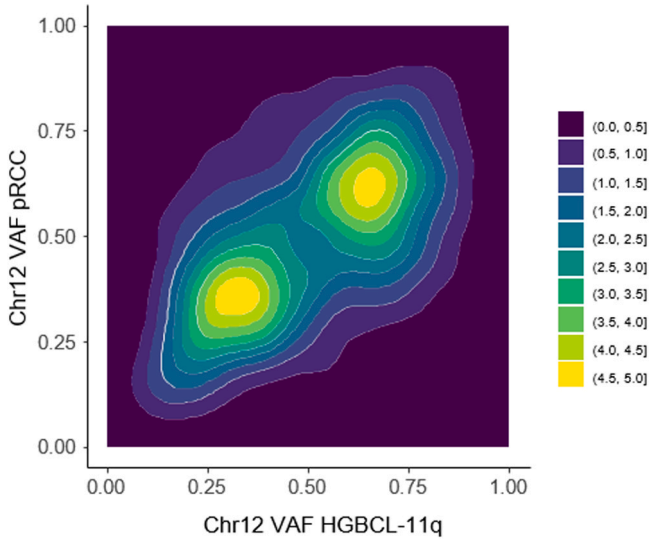

Fig. 2. Gain of same parental chromosome 12 allele in the lymphoma and the pRCC. Contour plot showing variant allele frequencies of all heterozygous variants found on chromosome 12 in the lymphoma and the pRCC sample. Trisomy 12 was found in both tumors and the direct correlation of the variant allele frequencies (VAFs) of heterozygous variants indicates the duplication of the same parental allele in both cases.

chromosome), which was not detected in the blood sample of the patient. Analysis of the allele frequencies of all heterozygous variants on chromosome 12 shared between both tumors indicated that the same allele was duplicated (Fig. 2). This copy number gain for chromosome 12 might affect the expression of multiple genes.

5. Discussion

In the present report, we provide further evidence for the existence of an immunodeficiency-associated subtype of HGBCL-11q. Our findings corroborate prior systematic studies and case reports [5,9], summarized in Table S6 [15,18–21], suggesting an increased incidence of HGBCL-11q, particularly in patients with inborn or acquired errors of immunity.

A potential reason for this increased incidence of HGBCL-11q in these patients could be the presence of a specific cell-of-origin population at risk. Both BL and HGBCL-11q share a germinal center dark zone phenotype [5]. We have recently proposed for BL that the cell population at-risk could be a germinal center derived B-cell population poised to express IgA,which encounters a first microbial challenge in a primary immune response [22,23]. Inborn and acquired immunodeficiencies lead to the loss of immunologic memory, resulting in a higher rate of primary immune responses. This extends the cell population at-risk and increases the likelihood of developing BL and, likely, also HGBCL-11q. In contrast to BL, which often presents with mucosa-associated lymphoid tissue involvement, HGBCL-11q seems not to have a predilection for this tissue type. Thus, HGBCL-11q might arise from a misdirected primary B-cell response not poised for (mucosal) IgA expression, as typical for the gastrointestinal tract, but rather poised toward a systemic (e.g. IgG) response. This could explain the different body sites affected and the distinct pattern of somatic mutations in HGBCL-11q, which more resembles the mutational patterns typical for primary germinal center B-cell derived lymphomas not presenting in the gut, such as DLBCL [4,24–26]. The present case aligns with these observations regarding both clinical presentation and somatic mutation pattern, which, aside from the common *TP53* mutation, lacks mutations typical for BL.

In the lymphoma sample FISH and OncoScan results indicated a gain at the *MYC* locus in the absence of a rearrangement. We and others have previously shown that *MYC* gene expression is usually lower in HGBCL-11q than in *MYC* rearranged Burkitt lymphomas [5] even in cases with *MYC* copy number gains recurrently observed in HGBCL-11q [5,27,28]. Therefore, *MYC* copy number alterations might coexist with typical chromosome 11 aberrations, although the biological and clinical impact still remains unclear.

The current case is particularly noteworthy as it displays features of inborn errors and acquired disturbances of immunity, as well as two synchronous malignancies. The former could be associated with germline predisposition or extensive immunosuppressive treatment. The latter forces the hypothesis, that a (cancer stem) cell common to both malignancies may be involved. Nonetheless, the patterns of structural and small variations differed between the tumors, with the notable exception of the gain of chromosome 12, which had a 50 % probability of being identical.

In summary, the systematic genetic work-up of this case of simultaneous HGBCL-11q and pRCC in a young patient with treatment-induced immunodeficiency, but likely also germline predisposition to an IEI, supports the existence of an immunodeficiency-associated subgroup of HGBCL-11q. This case sheds further light on the pathogenesis of these unique tumors and reveals potential pitfalls in the molecular pathological diagnostic process when imbalances appear to be shared by targeted analyses in different tumor entities.

Authors' contributions

RG performed and evaluated FISH analysis; AFi analyzed exomes, integrated data, performed bioinformatics analyses and wrote the manuscript; MV supported the interpretation of data; AFa, GG and EC followed clinically the patient; MG wrote histological data; SAT and SL performed pathological analysis; KOH performed and evaluated OncoScan analyses; SB performed and evaluated FISH analyses; AM performed annotation and interpretation of genetic variants; RS designed the study, interpreted molecular studies and wrote the manuscript; LL designed the study, performed pathological analyses and edited the manuscript. All authors read and approved the manuscript.

CRediT authorship contribution statement

Guido Garosi: Resources, Investigation. **Kathrin Oehl-Huber:** Investigation. **Susanne Bens:** Investigation. **Massimo Granai:** Writing – original draft, Data curation. **Sergio Antonio Tripodi:** Investigation. **Stefano Lazzi:** Resources, Investigation. **Raffaella Guazzo:** Writing –

original draft, Investigation, Conceptualization. **Reiner Siebert:** Writing – original draft, Supervision, Conceptualization. **Anja Fischer:** Writing – original draft, Formal analysis, Data curation. **Abubakar Moawia:** Investigation. **Emanuele Cencini:** Resources, Investigation. **Lorenzo Leoncini:** Writing – original draft, Supervision, Funding acquisition, Conceptualization. **Margherita Vannucchi:** Investigation, Data curation. **Alberto Fabbri:** Resources, Investigation.

Declaration of Competing Interest

The authors declare that they have no competing interests.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.prp.2024.155777.

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