Brief Report

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A Pragmatic First-Line Screening Assay for *PDGFR* Rearrangements: A Real-World Clinical Validation

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Abstract

Myeloid/lymphoid neoplasms with tyrosine kinase rearrangements (MLN-TK) are rare clonal eosinophilias driven by *PDGFRA*, *PDGFRB* and other kinase fusions, highly sensitive to tyrosine kinase inhibitors. Their detection remains challenging, particularly for cryptic *PDGFRA* rearrangements. We performed a large multicenter real-world validation of the generic quantitative RT-PCR assay (gPDGFR), which detects 3′ *PDGFRA*/*PDGFRB* overexpression independently of fusion partner. A total of 231 consecutive patients with hypereosinophilia from 12 French centers were analyzed, and assay robustness was further assessed in an independent heterogeneous cohort of 102 TKI-treated patients. Twenty-two *PDGFR*-rearranged cases (14 *PDGFRA*-r, 8 *PDGFRB*-r) were identified. The assay demonstrated 100% sensitivity and 100% negative predictive value. For *PDGFRA*, positive predictive value and specificity reached 100%. In contrast, *PDGFRB* overexpression showed lower specificity due to borderline false-positive cases, underscoring the need for confirmatory testing. In selected patients, longitudinal gPDGFR kinetics paralleled fusion-specific RT-qPCR, supporting its use for molecular follow-up when dedicated assays are unavailable, although it does not provide quantitative measurable residual disease assessment. Overall, gPDGFR represents a robust, partner-independent first-line screening strategy that can be readily integrated into routine diagnostic workflows to enable timely identification of patients eligible for targeted therapy.

Keywords: *PDGFR* rearrangement; hypereosinophilia; generic quantitative reverse transcription-PCR; diagnosis and follow-up tool

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1. Introduction

Molecular biology has become central to the diagnosis and monitoring of malignant hematologic disorders, a role reinforced by the 2022 WHO Classification of Haematolymphoid Tumours[1]. Within this molecular framework, a new group of diseases has emerged: myeloid/lymphoid neoplasms with tyrosine kinase receptor rearrangements (MLN-TK), often associated with hypereosinophilia (HE). These clonal eosinophilias are driven by rearrangements involving one of five key genes: *PDGFRA*, *PDGFRB*, *FLT3*, *JAK2*, or *FGFR1* and seven other even rarer defined tyrosine kinase fusions. Their identification is crucial, given the remarkable sensitivity to tyrosine kinase inhibitors (TKIs), particularly imatinib for *PDGFR* rearrangements (*PDGFR*-r). However, detecting these rearrangements—particularly those involving *PDGFRA*—is challenging due to cryptic cytogenetic profiles, variable breakpoints, and multiple fusion partners[2]. To address this, Erben et al. developed in 2010[3] a generic quantitative RT-PCR assay (gPDGFR assay) targeting overexpression of the 3′ region of PDGFRA or PDGFRB, independent of fusion partner or breakpoint. Although analytically attractive and technically simple, this strategy has not been widely implemented in routine diagnostics.

The present study provides a large-scale, real-world clinical validation of the gPDGFR assay in patients with unexplained HE, assessing its diagnostic performance, clinical correlates, and utility for longitudinal monitoring.

2. Materials and Methods

2.1. Study Design and Population

We conducted a multicenter observational study including 231 consecutive peripheral blood samples from patients referred for evaluation of HE (Appendix A). Samples originated from 12 hospital centers across France between 2012 and 2024 and were analyzed at the Bordeaux hematology laboratory. HE was defined as eosinophil count >1.5 × 10^9/L. To further challenge the negative predictive value (NPV) of the assay, we retrospectively analyzed a separate cohort of 102 TKI-treated patients identified through the institutional clinical data warehouse (Entrepôt de Données de Santé, EDS cohort). This cohort was not designed to estimate sensitivity but to assess the risk of false negatives in a heterogeneous TKI-exposed population.

2.2. gPDGFR Assay

This assay was performed as originally described by Erben et al. [3] using successive RT kits (Roche®, SuperScript™ III/IV VILO™). Expression levels were normalized to ABL1. Cut-offs were established locally using 31 healthy controls (mean + 3 SD): *PDGFRA*/*ABL1*: 1% and *PDGFRB*/*ABL1*: 40%. Historically, a 25% cut-off was initially applied for *PDGFRB*. Samples between 25% and 40% without confirmed rearrangement were retrospectively classified as false positives.

2.3. Definition of True Positive and True Negative Cases

True positive cases required molecular or cytogenetic evidence of *PDGFR* rearrangement, including karyotyping, FISH or transcript-specific RT-PCR. Clinical response to TKI was considered supportive but never used as a standalone diagnostic criterion.

True negative cases required: Absence of *PDGFR*-r by cytogenetic and molecular analyses (qualitative RT-PCR transcription negative[4], RT-multiplex ligation-dependent probe amplification (RT-MLPA) negative [5], combined with clinical, biological, or therapeutic features inconsistent with a clonal PDGFR-driven HE. Diagnostic performance parameters (sensitivity, specificity, **positive predictive value (**PPV), NPV) were calculated using standard 2×2 contingency tables.

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3. Results

3.1. Diagnostic Performance

Among the 231 samples, 194 were classified as negative with no overexpression of *PDGFRA*/B (mean of 0.02% [0-1.8%] and 5.66% [0.12-39.1%] respectively) and 37 as positive with overexpression of *PGDFRA* in 14 of them and *PDGFRB* in the other 23.

Notably, all samples exceeding the 1% *PDGFRA* expression cut-off (mean of 12.2% [2.53 – 116.6%]) had a confirmed *PDGFRA* rearrangement (*PDGFRA*-r), supporting a 100% PPV for this marker. In contrast, only 8 samples with *PDGFRB* overexpression (mean of 82% [40.7 – 107%]) had a confirmed *PDGFRB* rearrangement (*PDGFRB*-r), while 15 samples showed *PDGFRB* overexpression (mean of 32.81% [25.85 – 67.12%]) without any detectable rearrangement mentioned above (negative cytogenetic analysis, and if not performed, corticosteroid sensitivity or identification of another cause of HE). These borderline cases were classified as false positives, resulting in a PPV of 35% for *PDGFRB*. The overall sensitivity of the assay was 100%, with a specificity of 100 % for *PDGFRA*-r and 93% for *PDGFRB*-r and an exceptionally high negative predictive value of 100%. In the EDS cohort (n=102), none harboured *PDGFR-r* (gastro-intestinal stromal tumor (37%), chronic myeloid leukemia (31%), solid tumors (10%), and others such as *KIT*–mutated melanoma, systemic mastocytosis, chronic lymphoid leukemia or systemic sclerosis), confirming assay robustness with **no false positives** in heterogeneous TKI-treated populations.

3.2. Clinical and Biological Characteristics

We diagnosed 22 patients with *PDGFR*-r: *PDGFRA*-r (n=14) or *PDGFRB*-r (n=8) (Table 1) representing 1.75 new cases per year. This disorder predominantly affects elderly patients, with a median age of 67.5 years [range: 25–93 years]. A strong male predominance was observed for *PDGFRA*-r cases (sex ratio male / female of 2.5), and all male for *PDGFRB*-r. HE was higher in *PDGFRA-r* (mean: 6.42.10⁹/L [2.24–88.13 x 10⁹/L]) vs *PDGFRB-r* (mean: 2.05 x 10⁹/L [0.6–50.68 x 10⁹/L]). Cytopenias were present in all *PDGFRB-r* cases (100% anemia (mean: 10.8 g/dL [7.9 – 12.6 g/dL]), 83% thrombocytopenia (mean: 86 x 10⁹/L [34 – 269 x 10⁹/L]), but absent in *PDGFRA-r*. All *PDGFRA*-r patients showed elevated serum vitamin B12 (VitB12) and tryptase levels (corresponding data were not available for *PDGFRB*-r cases). Mean VitB12 concentration reached 2745.9 pg/mL [367–3500 pg/mL], and the mean tryptase level was 37.3 µg/L [22–51.9 µg/L]. Regarding associated hematologic diseases (Appendix B), *PDGFRA*-r linked mainly to MPN (86%) while *PDGFRB*-r showed a heterogeneous spectrum: 45% MPN, 22% acute myeloid leukemia (AML), 11% acute lymphoblastic leukemia (ALL), and 22% chronic myelomonocytic leukemia (CMML). This heterogeneity extended to the fusion partners (Appendix B). The *PDGFRA*-r predominantly involved *FIP1L1* (86%), whereas *PDGFRB*-r were more diverse: *ETV6 (*n=3), *CCDC6* (n=1), *CCDC88C* (n=1), and in 3 of cases, no fusion partner could be identified despite additional testing. Interestingly, the level of *PDGFR/ABL1* overexpression did not correlate with the eosinophil count, the fusion partner, or the associated hematologic malignancy.

Table 1. Clinical and biological data for patients with PDGFR rearrangements.

UPN	Sex	Age (years)	Hematologic diseases	Rearrangement	% overexpression	Eosinophils (10^9/L)	Tryptase (µg/L)	Viamin B12 (pg/mL)	Bone marrow	Karyotype	FISH	RT-PCR or RT-MLPA	TKI treatment	Time to remission
P1	M	49	MPN	FIP1L1::PDGFRA	6,85	4,4	unk	1902	HE > 20%	Normal	n.t	FIP1L1::PDGFRA	Imatinib 100 mg/d	unk
P2	M	85	MPN	FIP1L1::PDGFRA	10,1	6,42	51,9	890	unk	n.t	n.t	FIP1L1::PDGFRA	Imatinib 100 mg/d	unk
P3	M	84	MPN	FIP1L1::PDGFRA	4,23	3,25	n.t	367	unk	Normal	n.t	FIP1L1::PDGFRA	Imatinib 100 mg/d	3 months
P4	M	42	MPN	FIP1L1::PDGFRA	4,16	2,24	22	2160	Mild HE	Normal	CHIC2 deletion	FIP1L1::PDGFRA	Imatinib 100 mg/d	unk
P5	M	60	MPN	FIP1L1::PDGFRA	116,64	10,44	n.t	n.t	unk	Normal	n.t	FIP1L1::PDGFRA	unk	unk
P6	M	58	MPN	FIP1L1::PDGFRA	51,37	21	Normal	2500	HE	Normal	CHIC2 deletion	FIP1L1::PDGFRA	Imatinib 100 mg/d	unk
P7	M	54	MPN	FIP1L1::PDGFRA	17,93	4,4	unk	1902	unk	n.t	n.t	FIP1L1::PDGFRA	Imatinib 100 mg/d	unk
P8	M	75	CMML	FIP1L1::PDGFRA	13,29	14,5	n.t	8500	unk	Normal	CHIC2 deletion	FIP1L1::PDGFRA	Imatinib 100 mg/d	unk
P9	M	27	MPN	FIP1L1::PDGFRA	2,53	88,13	38	n.t	unsuitable	n.t	n.t	FIP1L1::PDGFRA	Imatinib 100 mg/d	unk
P10	F	77	MPN	FIP1L1::PDGFRA	9,81	6,22	unk	unk	HE=15%	Normal	CHIC2 deletion	FIP1L1::PDGFRA	Imatinib 100 mg/d	3 months
P11	F	48	MPN	FIP1L1::PDGFRA	12,17	1,43	unk	unk	HE=13%	Normal	CHIC2 deletion	FIP1L1::PDGFRA	« Watch and see »	unk
P12	M	76	MPN	FIP1L1::PDGFRA	10,78	unk	unk	unk	unk	unk	unk	FIP1L1::PDGFRA	unk	unk

P13	F	78	MPN	BCR::PDGFRA	83,52	3,11	unk	unk	unk	t(4;22)	unk	BCR ::PDGFRA	Imatinib 400 mg/d	6 months
P14	F	91	MDS-IB2	ETV6::PDGFRA	21,4	0,03	unk	unk	MDS-IB2	t(4;12)	ETV6::PDGFRA rearrangement	n.t	Imatinib 100 mg/d	unk
P15	M	57	AML	ETV6::PDGFB	98,27	0,6	n.t	n.t	AML	t(5;12)	ETV6::PDGFRB rearrangement	n.t	Imatinib 400 mg/d	unk
P16	M	77	MPN	ETV6::PDGFRB	60,5	50,68	n.t	n.t	HE	t(5;12)	ETV6::PDGFRB rearrangement	Negative	Ruxolitinib	unk
P17	M	58	AML	ETV6 ::PDGFRB	98,36	unk	n.t	n.t	unk	unk	n.t	ETV6 ::PDGFRB	Imatinib 400 mg/d	1 months
P18	M	77	CMML	CCDC88C::PDGFRB	85,09	2,19	n.t	n.t	CMML	t(5;14)	PDGFRB rearrangement	n.t	Imatinib 100 mg/d	unk
P19	M	70	CML	CCDC6::PDGFRB	40,7	1,9	n.t	n.t	MPN	t(5;10)	CCDC6::PDGFRB rearrangement	n.t	Imatinib 200 mg/d	unk
P20	M	93	MPN	PDGFRB unknown partner	82	unk	n.t	n.t	unk	unk	PDGFRB rearrangement	n.t	unk	unk
P21	M	65	MPN	PDGFRB unknown partner	107	16,28	n.t	>1476	unk	n.t	PDGFRB rearrangement	n.t	Imatinib 100 mg/d	unk
P22	M	25	ALL T	PDGFRB unknown partner	65,37	1,3	n.t	n.t	ALL	t(5;12)	PDGFRB rearrangement	Negative	Imatinib 500 mg/d	unk

M : male, F : female, B12 : Vitamin B12, FISH : Fluorescence In Situ Hybridization), RT-PCR : reverse transcription polymerase Chain reaction, RT-MLPA : reverse transcription multiplex ligation-dependent probe amplification, HE: Hypereosinophilia, MPN : myeloproliferative neoplasm, MDS-IB2 : myelodysplastic neoplasm with increased blasts 2, AML : acute myeloblastic leukemia, CMML : chronic myelomonocytic leukemia, ALL T : acute lymphoblastic leukemia, CML : chronic myeloid leukemia, n.t : not tested, unk : unknown. Time to remission represents time until patients normalized their eosinophilia and *PDGFR* rearrangement was undetectable.

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3.3. Treatment Response and Molecular Monitoring

All *PDGFRA*-r patients treated with imatinib (n=14) achieved sustained complete hematologic remission, with no molecular or clinical relapse observed after a median follow-up of 5 years.

Among *PDGFRB*-r patients, 6 patients received imatinib and one received ruxolitinib. Follow-up data were available for 4 patients with a mean-time of 6 years.

In two patients with adequate longitudinal data, gPDGFR kinetics paralleled transcript-specific RT-qPCR (*FIP1L1::PDGFRA*, *BCR::PDGFRA*). While not suitable for quantitative measurable residual disease (MRD) assessment, the assay reliably reflected molecular clearance and stability (Figure 1).

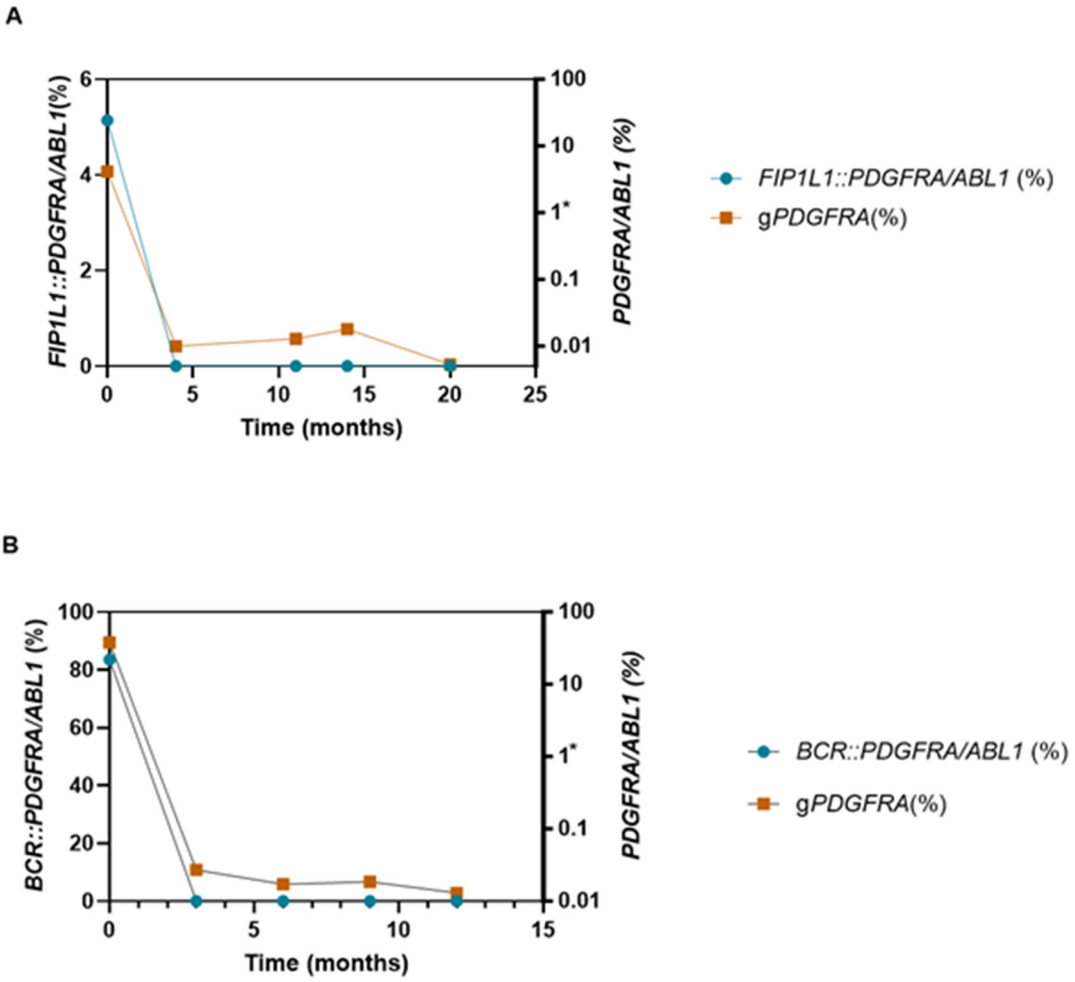

Figure 1. Two examples of follow-up by gPDGFR and specific quantitative PCR: (A): patient P4 with *FIP1L1::PDGFRA*, (B): patient P13 with *BCR::PDGFRA*. The cut-off point is indicated by the asterisk on both graphs.

4. Discussion

In this multicenter real-world study, we demonstrate that the generic gPDGFR assay is a robust and clinically relevant screening tool for the detection of PDGFRA rearrangements in patients with unexplained HE. The assay showed excellent diagnostic performance, with 100% sensitivity and negative predictive value in our cohort. Notably, *PDGFRA* overexpression above the predefined threshold was fully concordant with confirmed *PDGFRA* rearrangement, resulting in a positive predictive value and specificity of 100% in this setting.

In contrast, *PDGFRB* overexpression was associated with a lower positive predictive value due to borderline cases without detectable rearrangement. This finding likely reflects higher physiological

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baseline expression and greater biological heterogeneity of *PDGFRB*-driven neoplasms. These results indicate that, while gPDGFR is highly reliable for excluding *PDGFR* rearrangements, *PDGFRB*-positive or borderline cases require systematic confirmatory cytogenetic or molecular analyses.

Beyond diagnostic performance, our data further delineate the clinical differences between *PDGFRA*- and *PDGFRB*-rearranged neoplasms. *PDGFRA*-r cases were characterized by marked eosinophilia, absence of cytopenias, elevated serum vitamin B12 and tryptase levels, and a predominance of *FIP1L1* fusion partners. In contrast, *PDGFRB*-r cases displayed greater clinical and molecular heterogeneity, frequent cytopenias, and diverse associated hematologic malignancies. Importantly, the level of *PDGFR* overexpression did not correlate with eosinophil count, fusion partner, or underlying disease, supporting the concept that the assay functions as a qualitative screening tool rather than a quantitative surrogate of disease burden.

Therapeutically, our findings confirm the remarkable sensitivity of *PDGFRA*-rearranged neoplasms to imatinib, consistent with previously reported remission rates exceeding 90% [6,7]. All treated *PDGFRA*-r patients achieved sustained hematologic remission. Outcomes were more heterogeneous in *PDGFRB*-r cases, reflecting both biological diversity and differences in associated hematologic diseases.

Regarding longitudinal monitoring, we observed concordant kinetics between gPDGFR and fusion-specific RT-qPCR in selected patients. Although the assay does not provide the analytical precision required for measurable residual disease assessment, it reliably reflected molecular clearance and sustained response. Thus, gPDGFR may represent a practical alternative for molecular follow-up when fusion-specific assays are unavailable, particularly in cases with rare or unidentified partners.

This study has several strengths. It includes a relatively large consecutive multicenter cohort of patients with HE, reflecting real-world diagnostic practice. The independent evaluation in a heterogeneous TKI-treated population further supports the robustness of the assay and its high negative predictive value.

Limitations include the retrospective design, the limited number of *PDGFRB*-rearranged cases, and the absence of formal receiver operating characteristic analysis to refine cut-off thresholds. In addition, the monitoring data remain limited to a small number of patients and should be interpreted cautiously.

In conclusion, the gPDGFR assay represents a pragmatic, partner-independent first-line screening strategy for *PDGFRA* rearrangements and an effective tool for the preliminary exclusion of *PDGFR*-driven HE. Its integration into routine diagnostic workflows can facilitate timely identification of patients eligible for targeted therapy, while complementary cytogenetic and molecular analyses remain essential, particularly in *PDGFRB*-positive or borderline cases.

Author Contributions: Conceptualization, FL and AB.; methodology, FL and AB, software, FL and AB.; validation, FL and AB.; formal analysis, FL, LB, MM and CC.; investigation, FL, MMG, DL, CB, GE, WC, LM, JE, FL, JBG, NN, DR, JQ, PCL, EK, EL; data curation, FL.; writing—original draft preparation: FL and AB; writing—review and editing, FL, LB, MMG, DL, CB, GE, WC, LM, JE, FL, JBG, NN, DR, JQ, PCL, EK, EL, AB.; supervision, AB. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement: This study received a favorable opinion from the Research Ethics Committee of the Bordeaux University Hospital (CER-BDX 2024 – 44).

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

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Conflicts of Interest: The authors declare no conflicts of interest.

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Appendix A

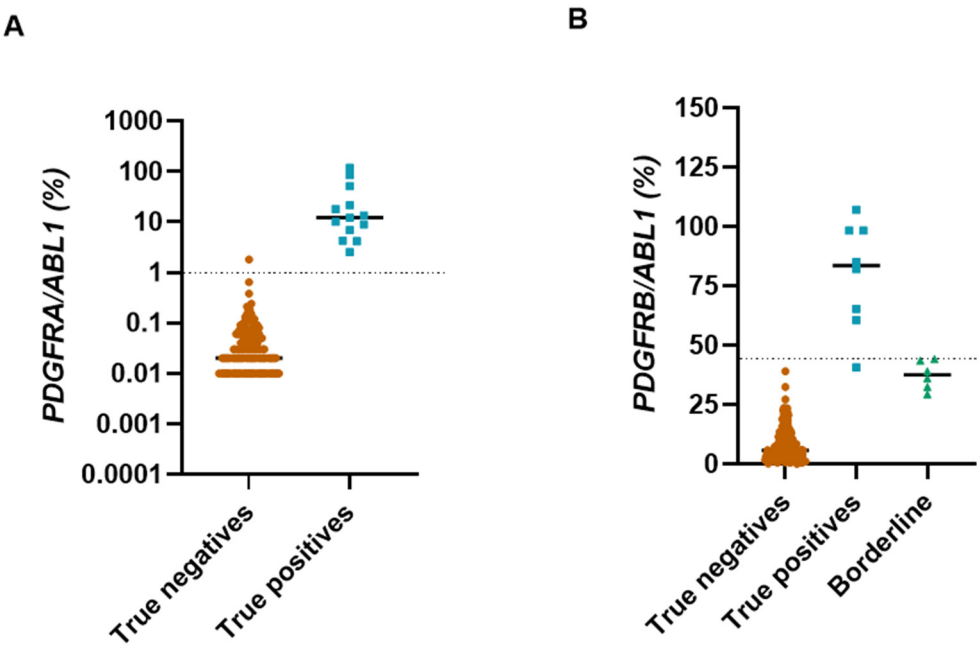

Figure A1. Diagnostic performance of gPDGFR: Percentage of *PDGFR/ABL1 (%)*. (A)*: PDGFRA* expression. (B)*: PDGFRB* expression. The cut-off point is indicated by dotted lines on both graphs (former cut-off, according to [3], is represented by the blue dotted line).

Appendix B

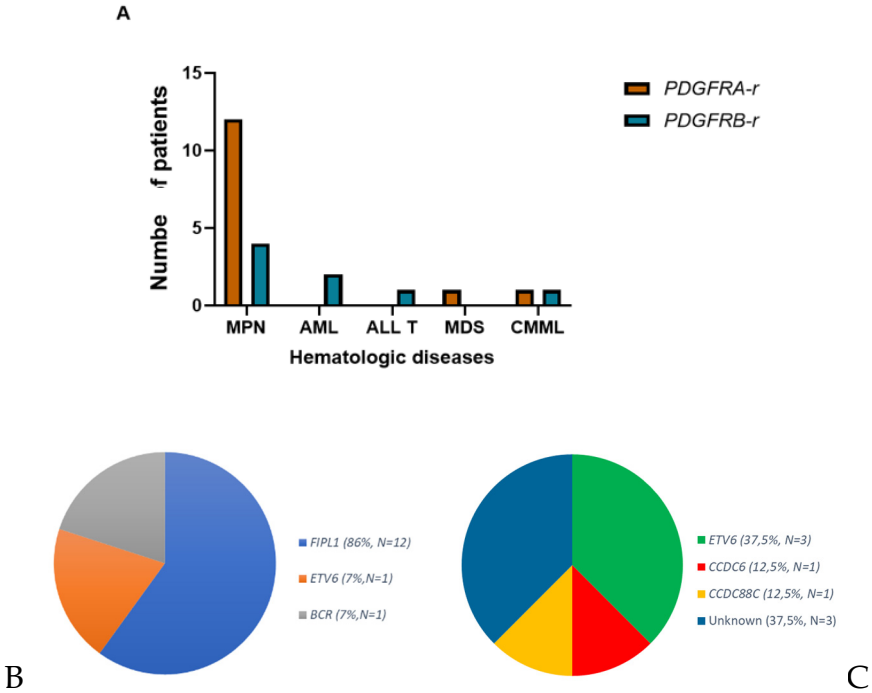

Figure A2. Hematologic diseases associated with *PDGFR* rearrangement and fusion partner. (A): Most of *PDGFRA* rearrangements are associated with MPN whereas *PDGFRB* rearrangements are associated with multiple neoplasms, such as MPN, AML, ALLT or CMML. (B): *PDGFRA* partner is mainly *FIP1L1*. (C): *PDGFRB* partner are multiple (*ETV6, CCDC6, CCDC88C*) and notably unknown in one third of the cases. MPN:

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myeloproliferative neoplasms, AML: acute myeloblastic leukemia, ALL T: acute lymphoblastic leukemia T, MDS: myelodysplastic neoplasms, CMML: chronic myelomonocytic leukemia.

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