Convegno interregionale SIE

Delegazioni Emilia Romagna e Toscana

Gli ematologi insieme contro le malattie rare

21 Aprile 2026

Bologna, Aula Prodi

ITP - le terapie: quali ed in quale sequenza

Francesca Palandri

IRCCS AOU di Bologna

Disclosures of Francesca Palandri

Company name	Research support	Employee	Consultant	Stockholder	Speakers bureau	Advisory board
Incyte			x			x
Novartis					x	x
AOP					x	x
Celgene/BMS					x	x
Grifols						x
Sobi						x
GSK					x	x
Kartos/Telios						x
Amgen					x	x
Sanofi						x
Takeda			x			x

Goals of treatment in adult ITP

✓ Decision making relies on efficacy, safety and **patient-dependent factors**

Bleeding risk

✓ **Safe platelet** count rather than normal count

Minimize treatment-related side effects

Maximize quality of life

✓ Minimize ITP-related symptoms, treatment burden, financial toxicity and **psychological distress**

Main pathogenic mechanisms of ITP

Multiple pathogenetic mechanisms also offer **multiple therapeutic opportunities**

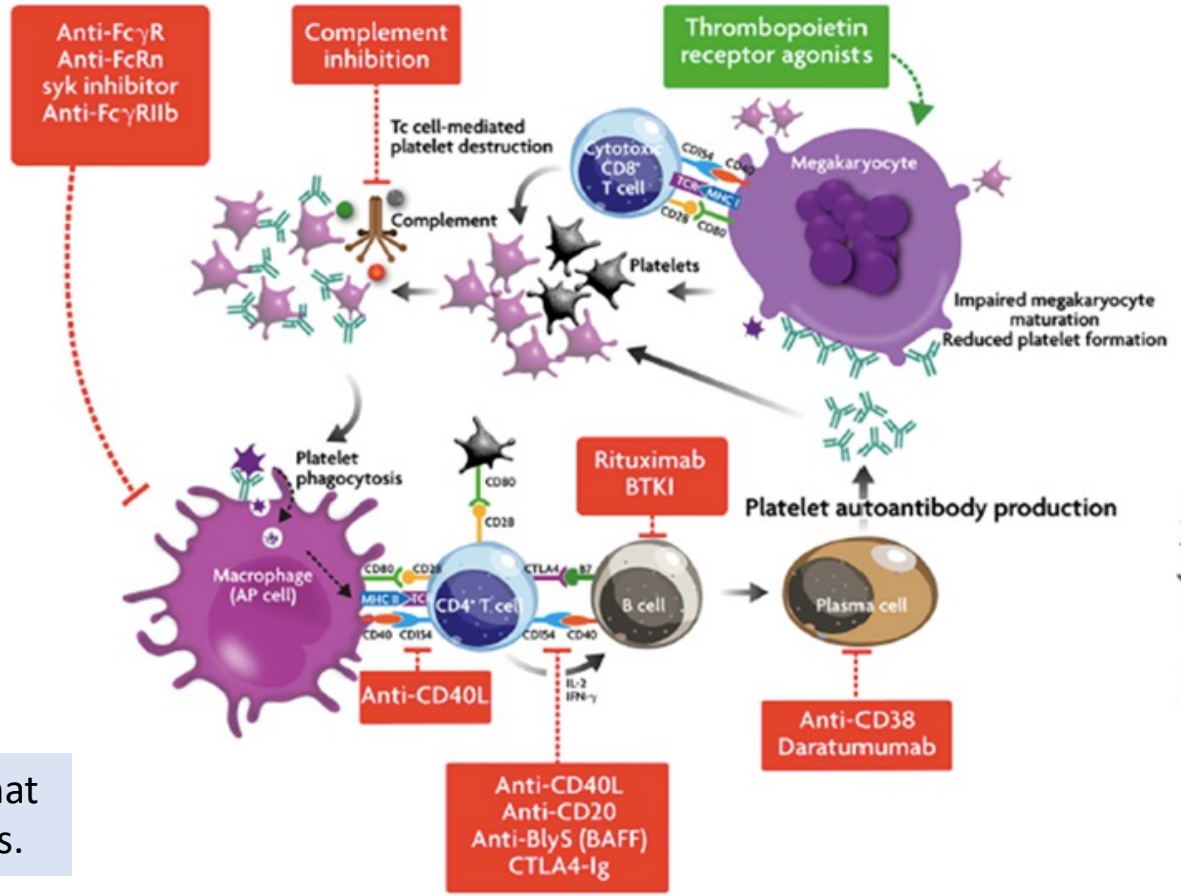
Antibody-mediated platelet destruction (spleen and liver via Fcγ receptors)

impaired platelet production (immune-mediated suppression of megakaryocytes in the marrow)

Direct T-cell toxicity to platelets and megakaryocytes

Complement-mediated destruction and platelet desialylation

Inflammation represents an underlying immune dysregulation that contributes to and amplifies the main pathogenetic mechanisms.



Houwerzijl EJ, et al. Blood. 2004;103:500-506. Reff ME, et al. Blood. 1994;83:435-445. Imbach P, et al. N Engl J Med. 2011;365:734-741. Newland A, et al. Future Med. 2017;10:9-25. Deutsch VR, et al. Br J Haematol. 2006;134:453-466. Kuter DJ, et al. Hematol Oncol Clin North Am. 2009;23:1193-1211. Grovovsky R, et al. Curr Opin Hematol. 2010;17:585-589. Lambert MP, et al. Blood. 2017;129:2829-2835

Glucocorticoids – 1st line therapy

- Glucocorticoids (GC) bind to cytosolic receptors and modulate a variety of genes.
- Main mechanisms of action in ITP include immunomodulation, reduced platelet destruction, and enhanced platelet production

Decrease of BAFF (B-cell activating factor)

- Serum BAFF levels are significantly elevated in patients with active ITP
- The GC receptor has a binding site in the BAFF promoter region, thus inhibiting its expression
- GC-mediated reduction of serum BAFF levels and BAFF mRNA expression, results in **reduced activation and proliferation of autoreactive B cells**

Decrease of immunoreactivity of dendritic cells

- GC **reduce the number of circulating dendritic cells** in ITP
- GC increases expression of tolerance-associated receptor (CD205) and **reduces immune activation receptors** (CD80/CD86) **in dendritic cells**

Decrease platelet destruction by inhibition of Fc receptor expression on monocytes

- GC decreases expression of activating receptors (FcγRI) and increase expression of inhibitory (FcγRIIb) receptors on monocytes
- This shift coincides with **reduced phagocytic capacity of monocytes**
- GC also reduces CD64 (FcγRI) expression on pro-inflammatory intermediate monocytes

Zufferey A J Clin Med. 2017 Feb 9;6(2):16. Provan D Blood Adv 2019; 3: pp. 3780-3817. Neunert C Blood Adv 2019; 3: pp. 3829-3866. Mingot Castellano ME J Clin Med 2023; pp. 12. Ma J Int J Hematol 2020; 112: pp. 773-779.

Glucocorticoids – 1st line therapy

Prednisone 1 mg/kg/day (not exceeding 80 mg/day)

Maintain for 3 weeks (2 weeks if no response)	Taper quickly (3-6 weeks)	Lower psycho-tropic effects	Higher long-lasting responses

High-dose Dexamethasone (HD-DEX) 40 mg/day for 4 days every 2 weeks

Fixed duration, no need for intercycle prednison Max 3 cycles	Faster response	Less mineral corticoid activity	Reduced compliance issues

Initial response rate of 50% to 80%

Long-term response rate ~30%

Neunert C, et al. American Society of Hematology 2019 Guidelines for Immune Thrombocytopenia. Blood Advances. 2019;3(23):3829-3866. **Wei Y, et al.** High-Dose Dexamethasone vs Prednisone for Treatment of Adult Immune Thrombocytopenia: A Prospective Multicenter Randomized Trial. *Blood*. 2016;127(3):296-302. **Cheng Y, et al.** Initial Treatment of Immune Thrombocytopenic Purpura With High-Dose Dexamethasone. *The New England Journal of Medicine*. 2003;349(9):831-836. **Mazzucconi MG,** et al. Therapy With High-Dose Dexamethasone (HD-DXM) in Previously Untreated Patients Affected by Idiopathic Thrombocytopenic Purpura: A GIMEMA Experience. *Blood*. 2007;109(4):1401-1407. **Mazzucconi MG,** et al. Prednisone vs High-Dose Dexamethasone in Newly Diagnosed Adult Primary Immune Thrombocytopenia: A Randomized Trial. *Blood Advances*. 2024;8(6):1529-1540.

Glucocorticoids – practical considerations

hypertension

Monitor blood pressure, adaptation of therapy

hyperglycemia

Monitor glucose levels, start or adapt hypoglicemic agents

Gastric irritation

Recommend short-course PPI

Mood disturbances

Alert the patient and caregivers

Bone metabolism

Vitamin D intergration and DEXA

GCs contraindications or early signal of non-response/intolerance

Switch/Add-on to 2nd line agents as soon as possible

In case of bleeding or need of rapid platelet increase

Add IVIg

Initial response rate of 50% to 80%

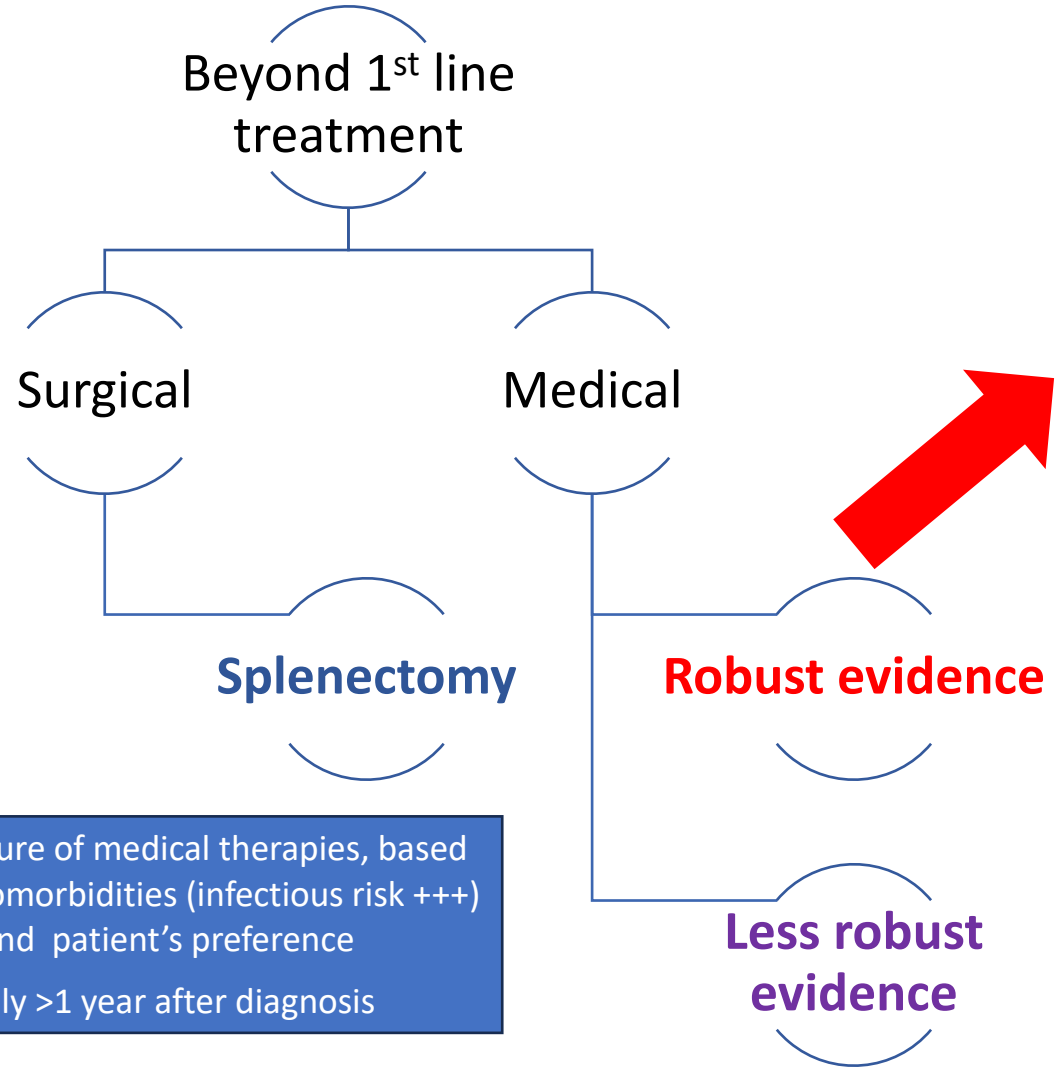
Consider inclusion into clinical trials!

Long-term response rate ~30%

Prepare for 2° line therapy

Speaker's personal opinion

Treatment options after GC failure



After failure of medical therapies, based on age, comorbidities (infectious risk +++) and patient's preference

Only >1 year after diagnosis

Eltrombopag
Avatrombopag
Romiplostim
Fostamatinib
Rituximab

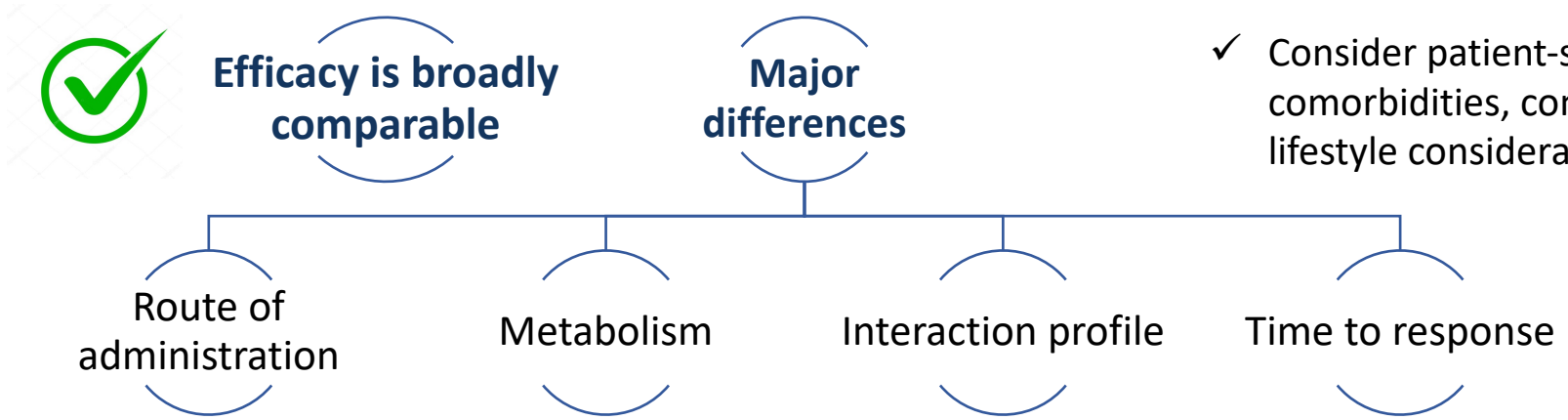
Rilzabrutinib

Azathioprine
Cyclosporin A
Cyclophosphamide
Danazol
Dapsone
Mycophenolate mofetil
Vinca alcaloids

- **No head-to-head trials**
- **Taylor patient treatment**
- Consider **thrombotic risk**, age, comorbidities, **patient's values and preferences**

Provan D et al. Blood Adv. 2019;3(22):3780-3817
Neunert C et al. Blood Advances. 2019;3(23),3829-3866
Ghanima W. et al, Blood. 2021;137(20):2736-2744

TPO-receptor agonists: which one



- ✓ Consider patient-specific factors such as comorbidities, concomitant treatments, and lifestyle considerations

Molecule	Class/route	Start dose	Major metabolism	Drug-drug Interactions (DDI)	Monitoring	Cautions
Romiplostim	Peptibody/ sc weekly	Weekly 1 µg/kg weekly, titrate to response (max 10 µg/kg)	Proteolytic & receptor mediated clearance (no hepatic CYP)	Low DDI risk	No routine LFT monitoring	Platelet fluctuations (avoid abrupt stop & PLT overshoot) Thrombotic risk
Eltrombopag	Small molecule Oral, daily	50 mg/day (25 mg Asian/hepatic impairment) titrate to response (max 75 mg/day)	Hepatic (UGT1A1, CYP1A2, others); biliary excretion	Chelates polyvalent Cations, separate from Ca^{2+}/Fe^{2+}/milk;	Baseline and periodic LFTs; Food requirements!	Platelet fluctuations (avoid abrupt stop & PLT overshoot) Thrombotic risk Hepatotoxicity risk
Avatrombopag	Small molecule Oral, daily	20 mg/die titrate to response (max 40 mg/day)	CYP2C9 / CYP3A (hepatic)	Fewer food/Ca interactions; potential CYP DDIs	LFT monitoring per clinical context; oral convenience improves adherence	Platelet fluctuations (avoid abrupt stop & PLT overshoot) Thrombotic risk

TPO-receptor agonists: practical considerations

Need for rapid platelet count increase (es. Surgery):	Hepatic alterations	Polypharmacy, or unreliable oral absorption:	Belonephobia:
Romiplostim avatrombopag	avoid eltrombopag	evaluate romiplostim for s.c. administration	Avoid romiplostim

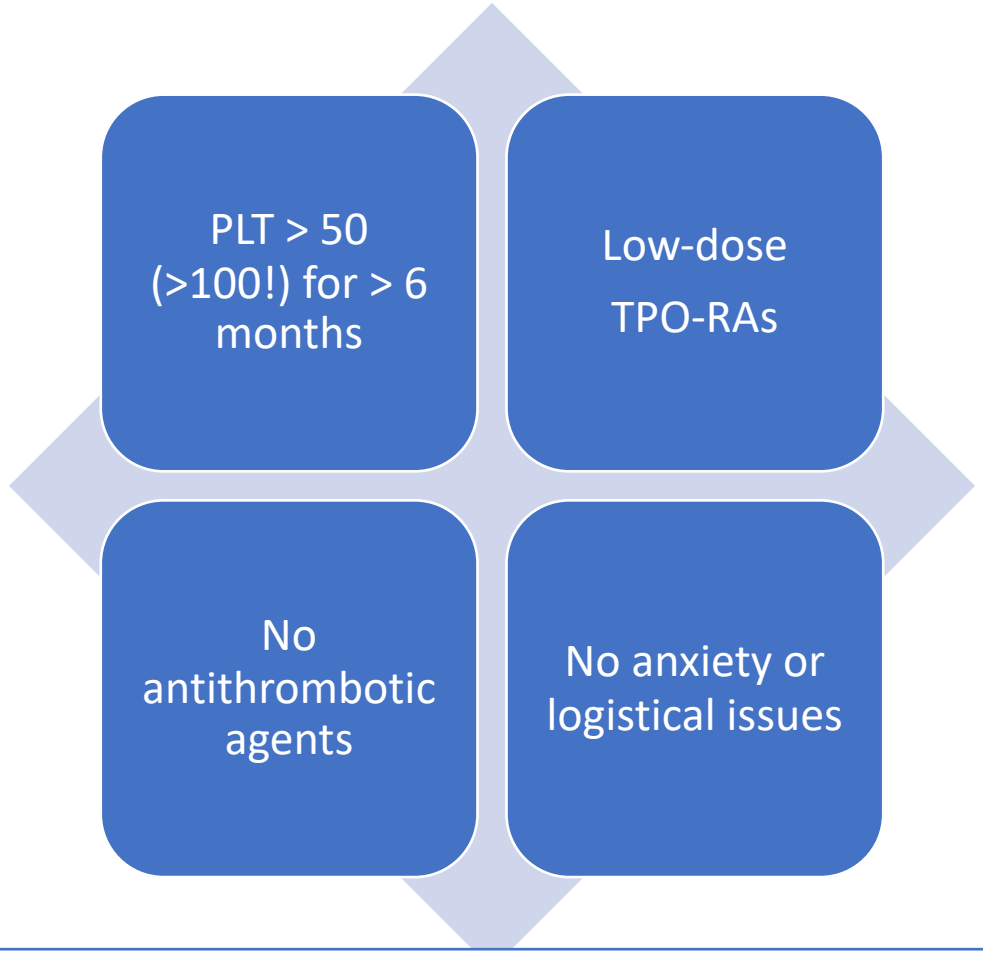
Agent/treatment	Reported dose range	Time to initial response*	Time to peak response*
Romiplostim	3-10 µg/kg weekly sc	5-14 d	14-60 d
Eltrombopag	50-75 mg po daily	7-28 d	14-90 d
Avatrombopag	20- 40 mg po daly	5-14 d (median 12 d)	10-14 d

Rodeghiero F et al Blood. 2009 Mar 12;113(11):2386-93.
Labanca C Eur J Haematol, 114: 733-746.

TPO-RAs for ITP: Sustained Response Off Therapy (SROT)

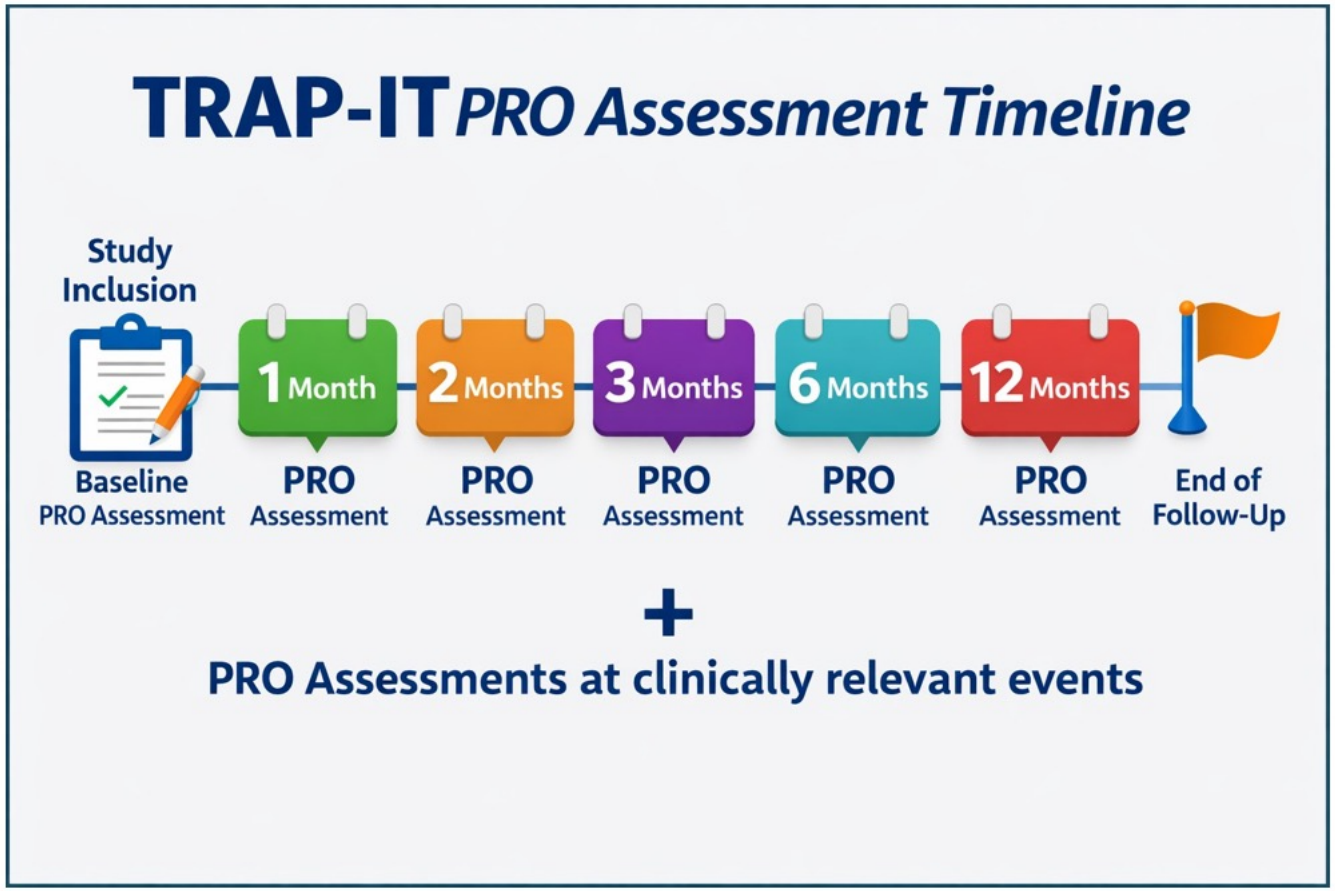
References	1. PLT > 50 (>100!) for > 6 months 2. Low-dose TPO-Ras 3. No antithrombotic agents 4. No anxiety 5. No logistical issues 6. TPO-RA rechallenge is generally effective!				
Agent					
Patients ≥18 yrs					
ITP duration before TPO-RA					
Line of treatment					
PLT count to start tapering					
Definition of SRoT					
Duration of TPO-RA					
SRoT	32%	25%	30.5%	56.2%	46.1%

TPO-RAs for ITP: SROT practical considerations



TPO-RA rechallenge is generally effective!

TPO-RA for ITP in the patient-centered era: TRAP-IT GIMEMA study



QUESTIONNAIRES

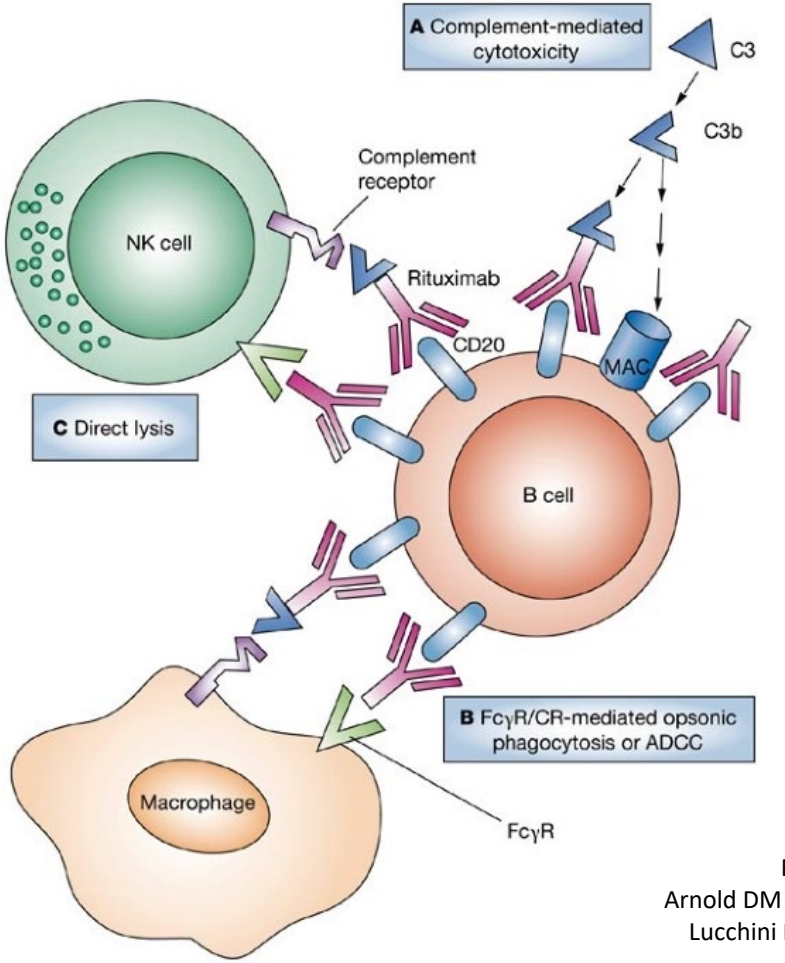
- **Adherence**
  - ARMS-7
- **Quality of Life**
  - SF-36
  - FACIT-Fatigue
- **Treatment Satisfaction**
  - TSQM-9
- **Financial Toxicity**
  - PROFFIT

Rituximab for ITP: targeting the immune system

Over the past 20 years, rituximab has been considered a valid second-line option in most guidelines (**Italy: L648**)

Rituximab is a type 1 IgG1-κ human-mouse chimeric mono clonal antibody directed against CD20, which acts through three mechanisms:

1. **complement-dependent cytotoxicity**
2. **antibody-dependent cellular cytotoxicity**
3. **induction of direct apoptosis** of the target cell



What to expect in ITP:

- Effective in 50–60% of cases
- Response duration at least 1 year in 40–60% of ITP patients
- At 5 years durable in 20–40% of patients
- Young women may benefit most
- Concomitant rheumatologic disease may benefit most

Lucchini E, Zaja F, Bussel J. Haematologica. 2019 Jun;104(6):1124-1135.
Image adapted from Taylor R Nat Rev Rheumatol **3**, 86–95 (2007).

Bussel JB Blood. 2009;113(10):2161–2171. doi: 10.1182/blood-2008-04-150078.
Arnold DM Ann Intern Med. 2007;146(1):25–33. doi: 10.7326/0003-4819-146-1-200701020
Lucchini E Haematologica. 2019;104(6):1124–1135. doi: 10.3324/haematol.2019.218883.
Marangon M Eur J Haematol. 2017;98(4):371–377. doi: 10.1111/ejh.12839

Rituximab for ITP: practical considerations

CONTRAINDICATIONS

Pregnancy

Active hepatitis B

Requires HBV screening; antiviral prophylaxis if HBsAg+ or anti-HBc+

Hepatitis B reactivation: Can be fatal during RTX

COMMON TOXICITIES

Infusion-related reactions: 38-62% of patients (fever, chills, rash, hypotension, bronchospasm); most common with 1st infusion; usually mild-moderate

Infections: 2.3-3.7% serious infections/100 patient-years; increased URTIs, bronchitis; rare PML

Serum sickness

Hypogammaglobulinemia: may persist >1 year

VACCINATION TIMING

Rituximab severely impairs responses (0-29% even 6-29 months post-dose);

Response to primary antigens more affected than recall antigens

Delay rituximab ≥2 weeks after vaccination;

if already on rituximab, vaccinate 6-12 months after last dose

VACCINATION RECOMMENDATION

Pneumococcal

Meningococcal (ACWY + B)

Haemophilus influenzae b

Influenza

COVID-19

Varicella-zoster (Shingrix)

Recommended before rituximab

Live vaccines are contra-indicated before, during, and after rituximab treatment

Lucchini E, Zaja F, Bussel J. Haematologica. 2019 Jun;104(6):1124-1135.
Image adapted from Taylor R Nat Rev Rheumatol **3**, 86–95 (2007).

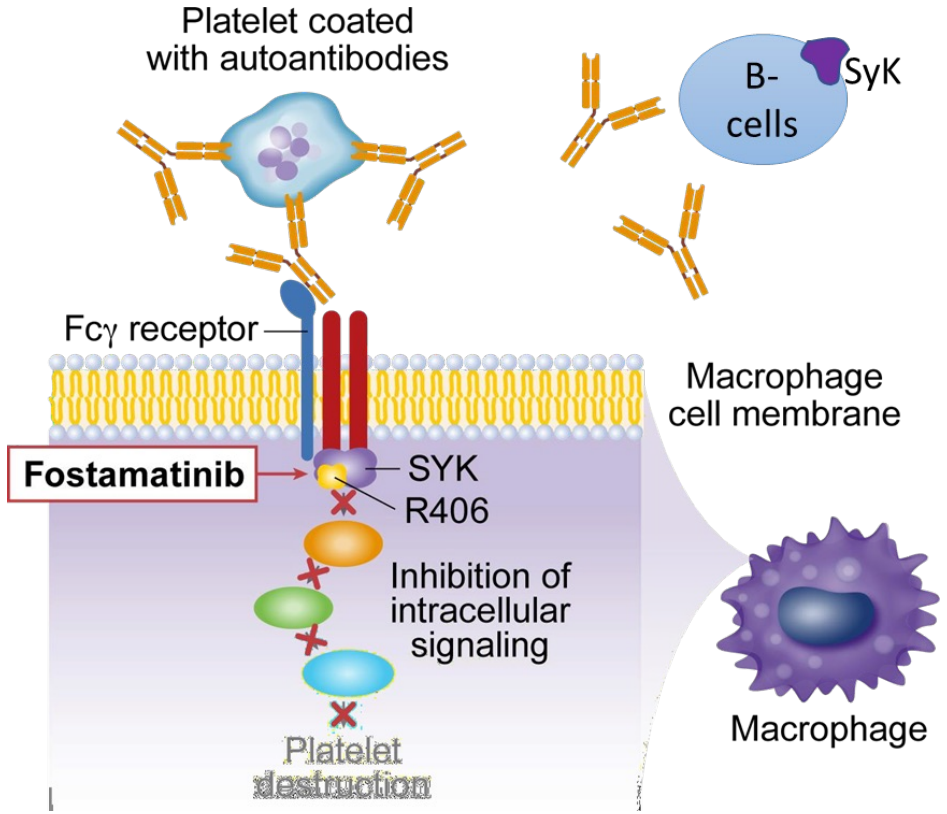
Bussel JB Blood. 2009;113(10):2161–2171. doi: 10.1182/blood-2008-04-150078.
Arnold DM Ann Intern Med. 2007;146(1):25–33. doi: 10.7326/0003-4819-146-1-200701020
Lucchini E Haematologica. 2019;104(6):1124–1135. doi: 10.3324/haematol.2019.218883.
Marangon M Eur J Haematol. 2017;98(4):371–377. doi: 10.1111/ejh.12839

Syk inhibition in ITP: mechanism of action

SYK is essential for B cell biology, regulating development, differentiation, maturation and antibody production through signaling cascades such as phosphoinositide 3-kinase (PI3K)–AKT, mitogen-activated protein kinase (MAPK) and nuclear factor-κB (NF-κB)

Decreased antibody-mediated destruction of platelets

(decreased opsonization of the complex Ab-PLT)



Decreased anti-PLT antibody production by B-cells

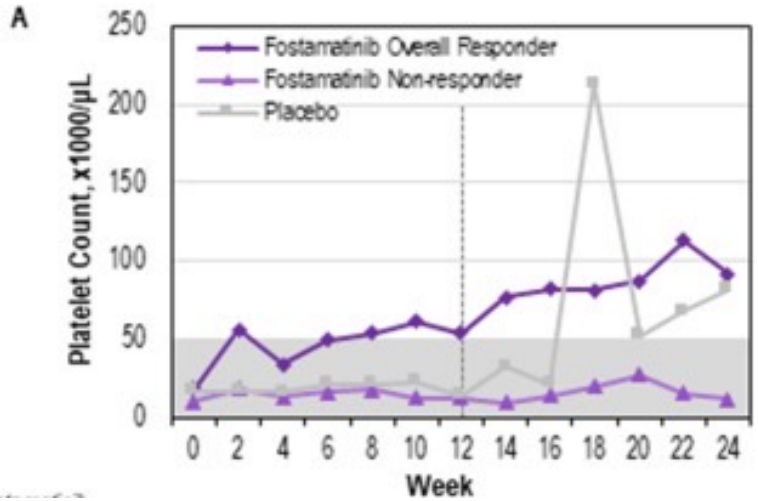
(decreased expression of the B-cell receptor)

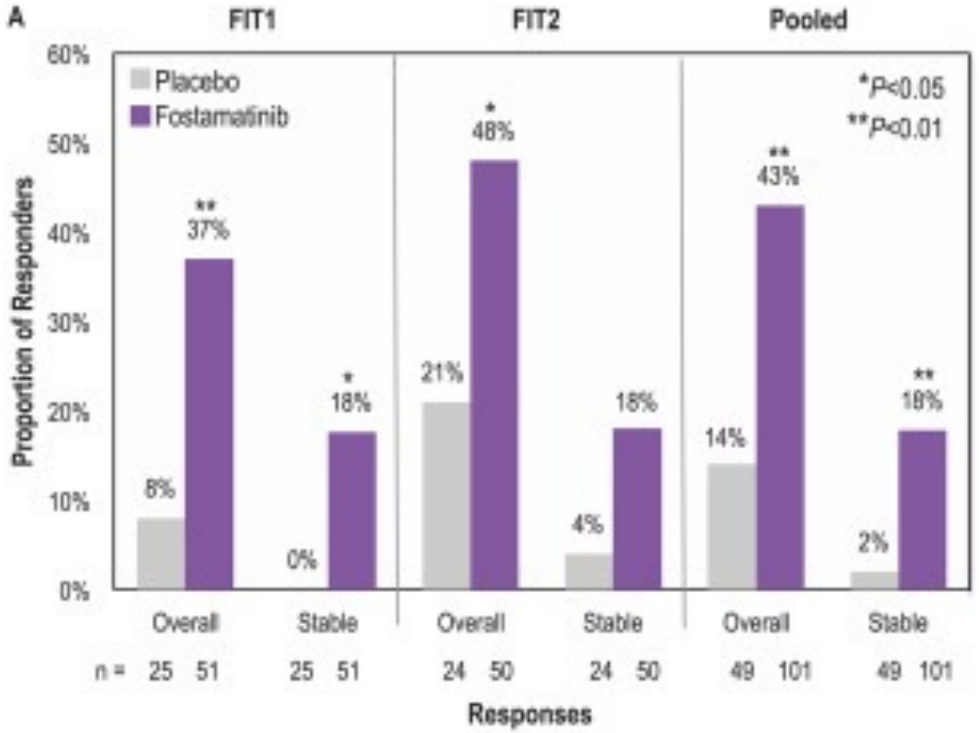
Zhang S Drug Discov Today. 2025 Jan;30(1):104257.

Fostamatinib (Syk inhibitor) in ITP: evidence from clinical trials

- Two randomized, double-blind trials (FIT1 and FI2 phase 3 trials)
- heavily pretreated patients with ITP
- median disease duration of 8.5 years
- **overall response rate 43% in the fostamatinib arm vs 14% in the placebo arm**

- **stable response rate was seen in 18% in the fostamatinib arm and 2% in the placebo arm.**





✓ Starting dose 100 mg BID, increase to 150 mg BID

The use of **fostamatinib as a 2nd line treatment for ITP is more effective** than when it is used in 3rd line or later

Bussel J Am J Hematol. 2018;93(7):921-930

Fostamatinib (Syk inhibitor) in ITP: safety

Drug-related AEs	Incidence	Comments
Hypertension	Fostamatinib group, 27.5% Placebo group, 12.5%	• Most reactions were mild or moderate in severity • Dose modification (reduction or interruption) was required for 4 patients receiving fostamatinib and no patients receiving placebo; study drug was discontinued in 1 patient receiving placebo and no patients receiving fostamatinib
ALT increase	Fostamatinib group, 11%	• All transaminase elevations were mild or moderate in se • Dose modification was required in 8 patients; st ntinued in 1 patient and the event resolved after discontinuation in that patient
AST increase	Fostamatinib group, 9%	
Noninfectious diarrhea	Fostamatinib group, 31%	• Most events were mild or m • Dose modification w nts receiving fostamatinib; study drug was discontinued in 1 patient recei
Neutropenia	Fostamatinib group, 7% Placebo group, 0	• M lated with infection and were mild or moderate in severity; all reactions except 1 of the study cation was required for 3 patients; study drug was discontinued in 1 patient
Infections	Fostamatin Placebo grou	espiratory tract infections accounted for 60% of the events in the fostamatinib group and 40% in the placebo group • No systemic opportunistic infections were reported with fostamatinib • Study drug was discontinued in 1 patient receiving fostamatinib

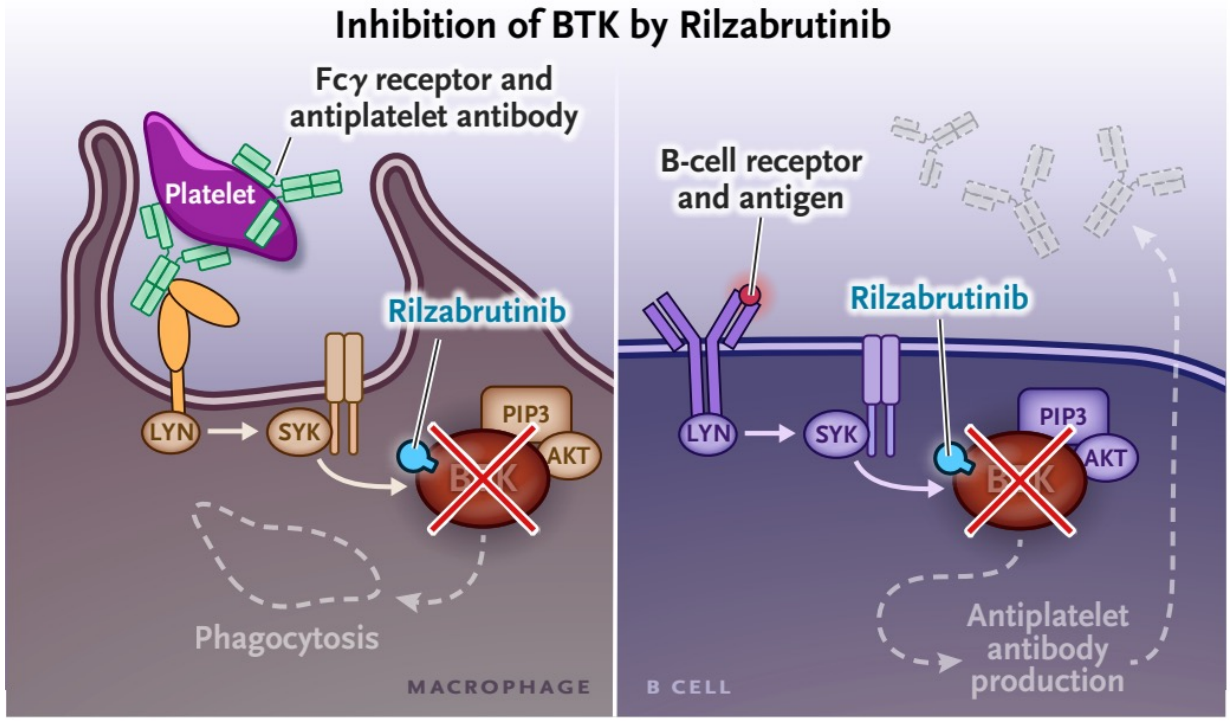
No novel toxicities developed during long-term treatment with fostamatinib

AEs rate did not increase with the increased duration of fostamatinib treatment during the extension trial (up to 62 months of treatment and 229 patient-years).

Bussel J Am J Hematol. 2018;93(7):921-930

Rilzabrutinib and BTK inhibition

Rilzabrutinib is an oral covalent, reversible Bruton tyrosine kinase inhibitor administered at dosage of 400 mg BID

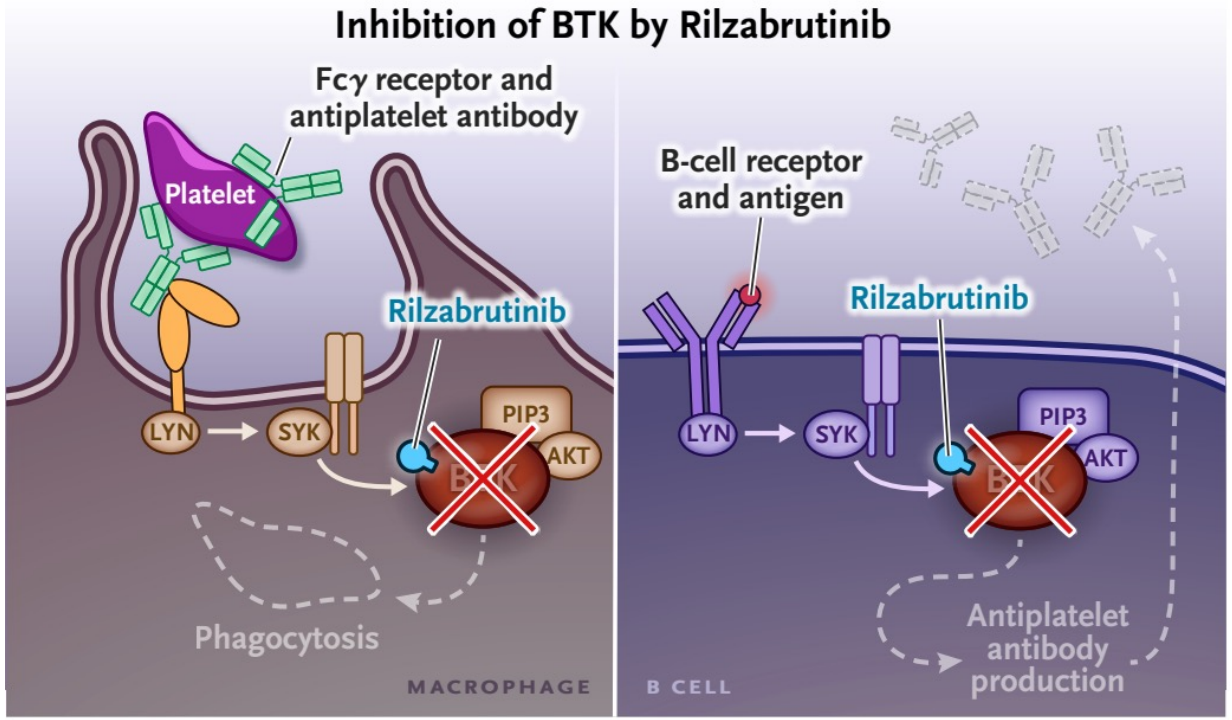


Rilzabrutinib mediates its therapeutic effect through multi-immune modulation mechanisms identified in ITP:

- inhibiting B-cell activation
- reducing pathogenic autoantibody production
- interrupting platelet phagocytosis by Fcγ receptor in the spleen and liver
- inhibiting inflammatory pathways

Rilzabrutinib and BTK inhibition

Rilzabrutinib is an oral covalent, reversible Bruton tyrosine kinase inhibitor administered at dosage of 400 mg BID



- **Reversible binding** – Unlike irreversible BTK inhibitors (e.g., ibrutinib), rilzabrutinib reversible covalent binding allows for **preservation of platelet aggregation** while reducing macrophage-mediated platelet clearance
- **High specificity** – Designed to **minimize off-target effects** through the PI3K-AKT pathway, reducing risks of atrial fibrillation and other adverse events associated with other BTK inhibitors
- **Rapid clearance** – Covalent binding provides **long BTK-target engagement** and durable inhibition with limited drug exposure, while **rapid systemic clearance reduces potential for off-target toxicity**

→ high sustained inhibition, minimal off target activity, and low drug levels

Rilzabrutinib pivotal phase 3 LUNA trial

LUNA3 was a phase 3, multicenter, placebo-controlled, parallel group study with open-label and long-term extensions

Patient enrolment:

- adults with persistent/chronic ITP

- 202 patients randomized
- median age was 47 years
- 63% female
- Median ITP duration 7.7 years
  - 28% prior splenectomy, 69% TPO-RAs
  - 45% of patients ≥ 5 prior unique ITP therapies → **heavily pretreated patient cohort**

Rilzabrutinib 400 mg BID for 24 weeks | R 2:1 | PBO

After 24 weeks PBO patients could cross-over to Rilzabrutinib

Platelet response defined as ≥50 × 10^9/L or 30 × 10^9/L to <50 × 10^9/L and doubled from baseline) was achieved in

64% for rilzabrutinib vs 32% for PBO

Primary end point: **durable platelet response** (platelet count ≥50 × 10^9/L for ≥two-thirds of ≥8 of the last 12 of 24 weeks without rescue therapy), was observed in:

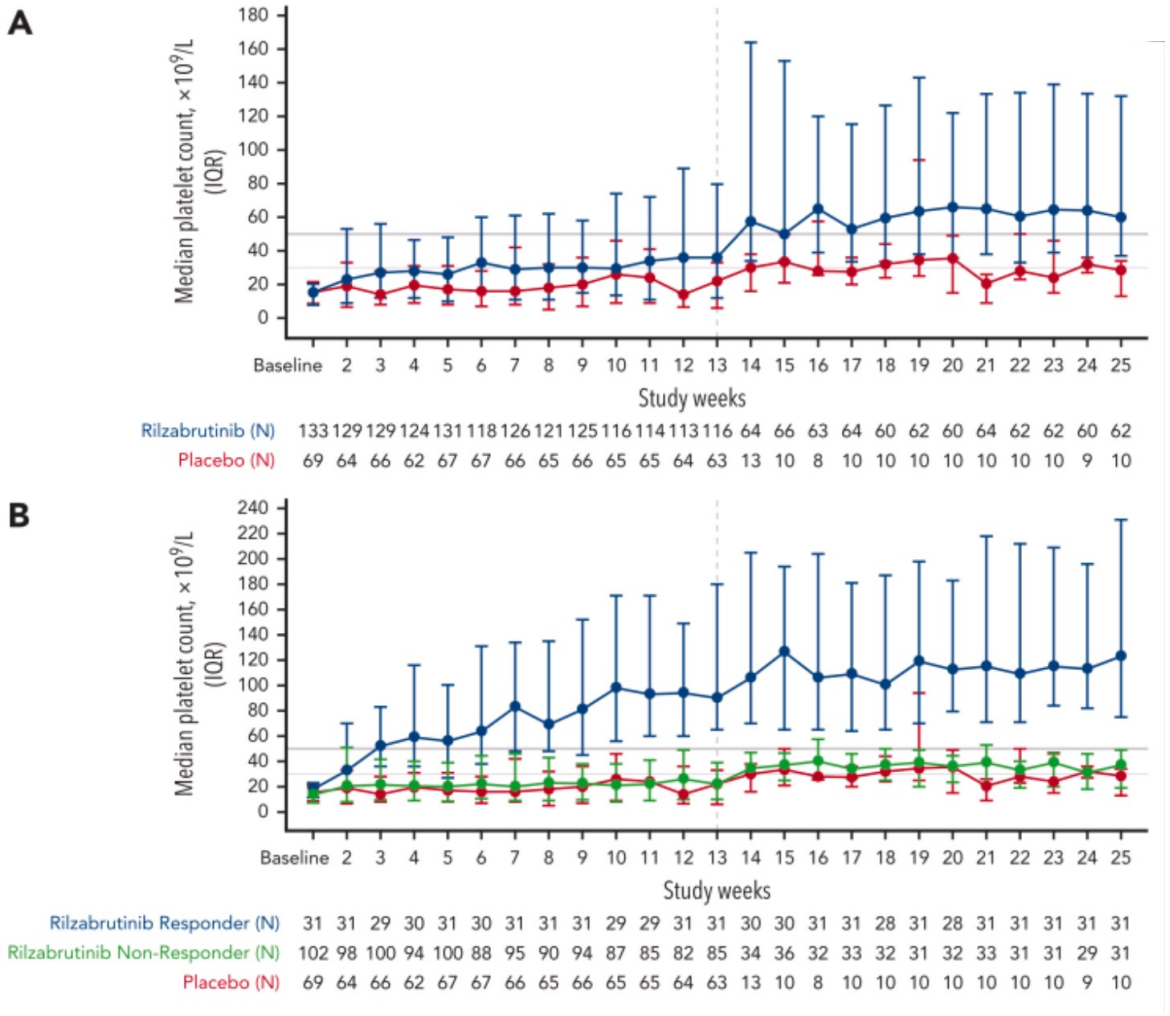
31 (23%) rilzabrutinib vs 0 placebo patients (*P* < .0001).

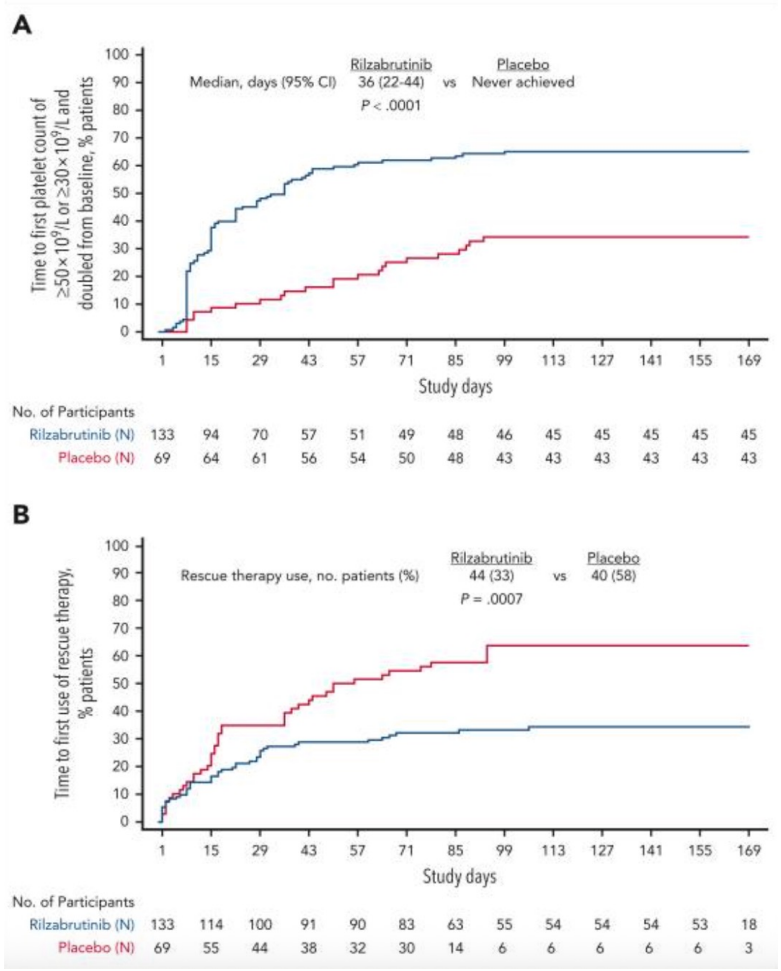
All secondary efficacy end points were significantly superior for rilzabrutinib (*P* < .05).

Median time to first platelet response was 15 days in rilzabrutinib responders.

Rilzabrutinib pivotal phase 3 LUNA trial: efficacy

Rilzabrutinib had rapid and durable platelet response, reduced rescue use and bleeding

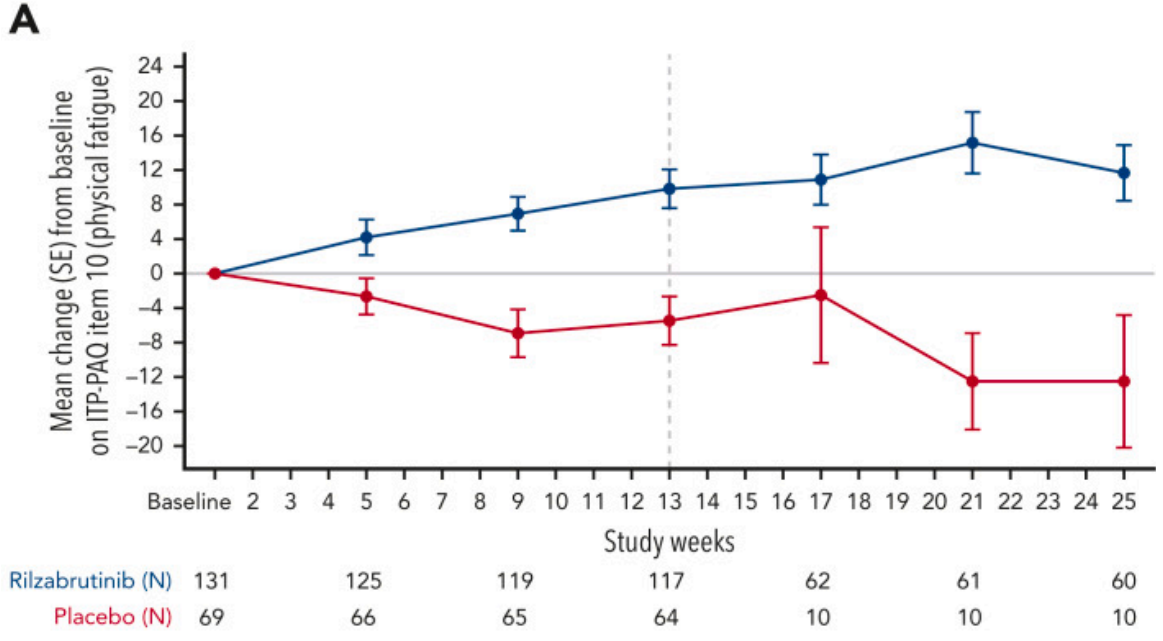


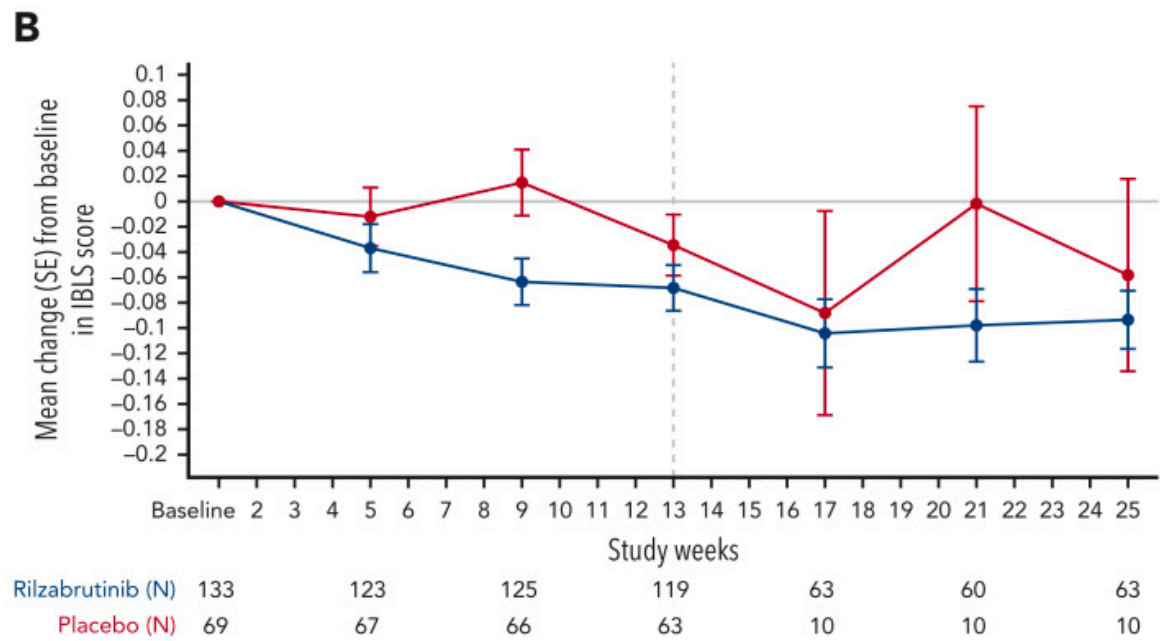


Kuter D Blood. 2025 Jun 12;145(24):2914-2926.

Rilzabrutinib pivotal phase 3 LUNA trial: PROs

Changes from baseline in fatigue score with rilzabrutinib were improved in durable responders and non-responders



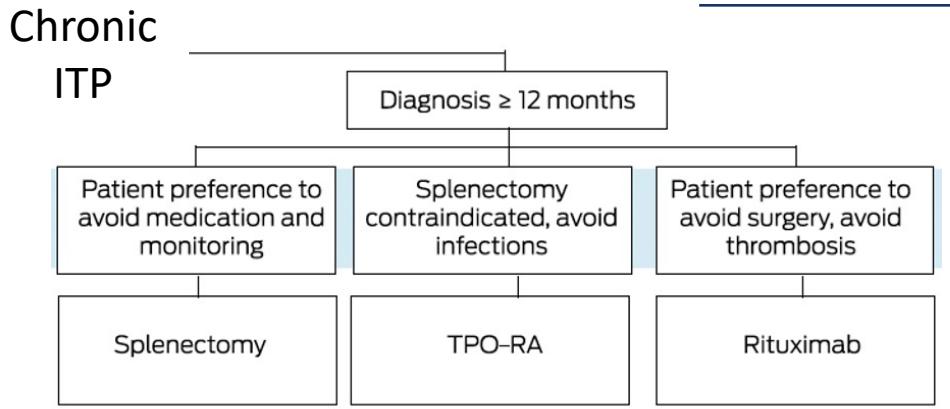


FDA and EMA
for persistent/chronic ITP
with insufficient prior response

MAP program open!

Kuter D Blood. 2025 Jun 12;145(24):2914-2926.

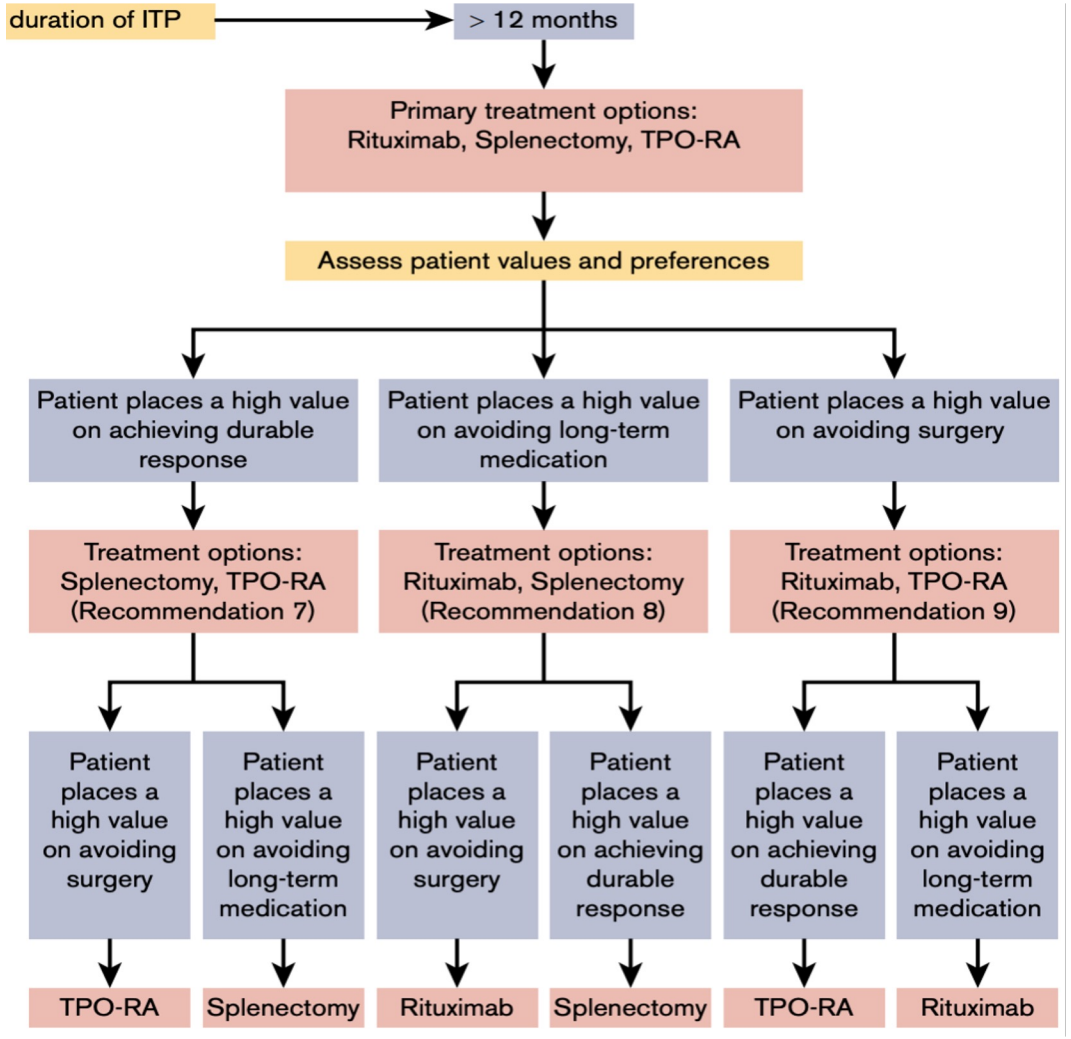
Splenectomy for ITP



- Delay splenectomy ≥ 1 year of ITP to avoid removing a healthy organ within range of spontaneous remission

- Patient's value and preferences:
  - To avoid long-term medication
  - To achieve durable response
  - Accepts surgery

Thrombotic + infectious risk assessment



Adapted from Neunert et al. 2019 Dec 10;3(23):3829-3866
Adapted from Choi PY Med J Aust. 2022 Jan 17;216(1):43-52. doi: 10.5694/mja2.51284.

Splenectomy for ITP: efficacy and predictors of response

	Johanna 2001	Schwarz 2003	Kojouri 2004	Vianelli 2005	Vianelli, Palandri 2013	Ahmed 2016	Guan 2016
No of patients	78	56	2623	345	233	254	174
Median f-up	9.4 y (2-22.6) from dx	7.5 y (5-10.5) from dx	29 mos (1-153) from dx	57 mos (1-498) from spl	20 y (10-43) from spl	54.3 mos (1-290)	100 mos
Complete Response		77%	66%	66%	77%	74%	72%
Overall Response	75%	86%	88%	86%	88%	90%	88%
Relapse	44%	20% (5y)	15% (2y)	23% (median time to relapse 8 mos)	29% (20 yrs)	30% (54mos)	20.8% (median time to relapse 24 mos)

On multivariable analysis, **lack of sustained response** after splenectomy was associated with **only older age** (60–75 years; > 75 years) and **use of at least 4 treatment lines** for ITP before splenectomy.

1) Johanna E. A., Blood. 2001;97:2549-2554; 2) Kojouri K., Blood. 2004; 104:2623-2634; 3) Schwartz J, American Journal of Hematology, 2003,72:94–98; 4) Vianelli N, Haematologica 2005; 90:72-77; 5) Vianelli N. Haematologica. 2013; 98(6):875-880; 6) Ahmed R. Ann Hematol. 2016;95(9):1429-34; 7) Guan Y. Eur J Haematol. 2016;98(3):235-241, 9) Mageau A, et al. Am J Hematol. 2022; 97(1): 10-17

Splenectomy for ITP in the modern era: GIMEMA study

Studio GIMEMA ITP1324

fondazione GIMEMA onlus
per la promozione e lo sviluppo della ricerca scientifica sulle malattie ematologiche FRANCO MANDELLI

Studio osservazionale sull'outcome dei pazienti con Piastrinopenia Immune primaria, sottoposti a splenectomia dopo l'01/01/2010

Disegno dello Studio

Studio **osservazionale, longitudinale , retrospettivo**, su pazienti affetti da ITP.
Verranno presi in considerazione i **pazienti affetti da ITP sottoposti a splenectomia a partire dall'01/01/2010 al 31/12/2022**
Tutti i dati utili saranno raccolti esclusivamente attraverso la consultazione delle cartelle cliniche ambulatoriali.
Il periodo di osservazione dei pazienti arruolati è di almeno 1 anno.

Criteri di inclusione

- ✓ Pazienti di età ≥ 18 anni
- ✓ Pazienti affetti da ITP primaria secondo i criteri internazionali **sottoposti a splenectomia dall' 01/01/2010 al 31/12/2022.**
- ✓ Ottenimento del Consenso Informato.

Endpoint Primario

Determinare la percentuale e l'incidenza di complicanze trombotiche, emorragiche e infettive (valutati secondo il CTCAE v4.0) intercorse durante il follow-up del paziente.

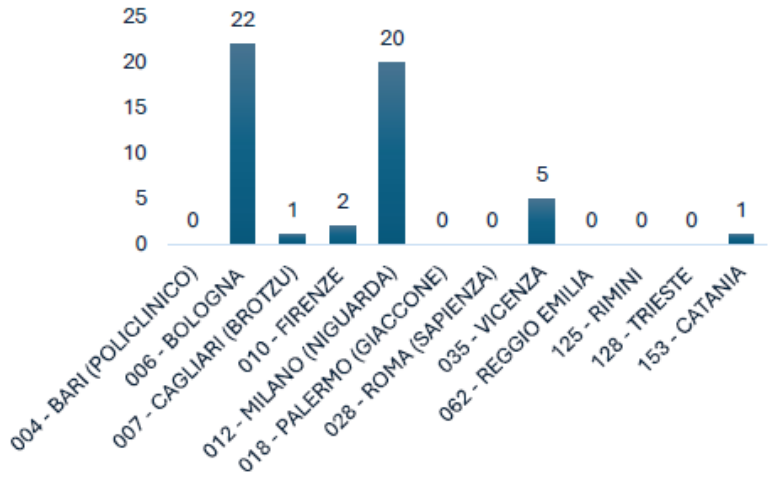
Endpoint Secondari

- **Valutazione della percentuale di pazienti che ottiene una risposta (R),** intesa come conta piastrinica superiore a 30.000/mmc e doppia rispetto al basale o una risposta completa (RC), intesa come conta piastrinica superiore a 100.000/mmc, a distanza di un mese dalla splenectomia
- **Valutazione della percentuale di pazienti che non rispondono o che ricadono dopo splenectomia**
- **Valutazione dell'impatto del numero e della tipologia dei trattamenti eseguiti pre intervento, sull'outcome**
- **Valutazione tipologia ed efficacia delle terapie mediche eseguite nei pazienti che non rispondono o che ricadono dopo splenectomia**
- **Valutazione della profilassi antinfettiva** effettuata mediante vaccinazione e/o terapia antibiotica
- **Valutazione del decorso di eventuali gravidanze post splenectomia** (ricaduta, necessità terapeutica, tipo terapia e risposta al trattamento, conta piastrinica neonato).

STATO DELLO STUDIO *Aggiornamento al 18.03.206*

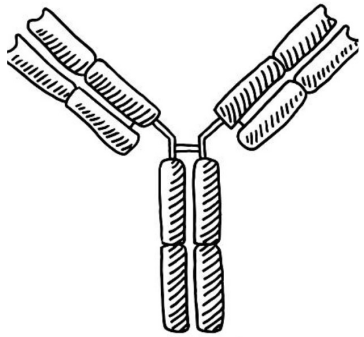
Centri coinvolti nello studio: **14**
Centri aperti all'arruolamento: **12**
Centri che hanno arruolato: **6**
Data apertura arruolamento dello studio: **03/02/2025**
Numero totale dei pazienti arruolati eleggibili: **51/157**

Centri aperti *Status arruolamento*



Future Agenda: (main) new agents

Anti-BAFF receptor antibody



Ianalumab (VAY736) is a glycoengineered (afucosylated) mAb which targets BAFF-R on plasmablasts, naïve and mature B cells.

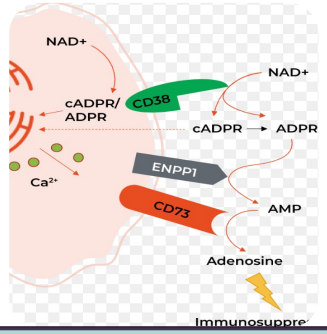
It has a dual MoA: decreased B-cell proliferation/Ab production & increased ADCC-mediated B-cell depletion

In 2L: 54% free from treatment failure at 12 months (vs 30% placebo); 62% stable response at 6 months (vs 39%)

Under investigation in 1L (with GC) and 2L (with eltrombopag)

Belimumab

Anti-CD38 antibody



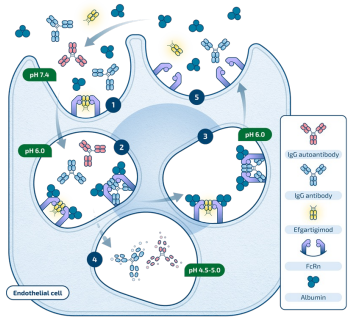
Mezagitamab targets long-lived plasma cells producing pathogenic autoantibodies

Phase 2 study: 91% platelet response at 600 mg dose vs 23% placebo

Phase 3 study now open in Italy! (NCT06722235)

Daratumumab

FcRn Antagonists



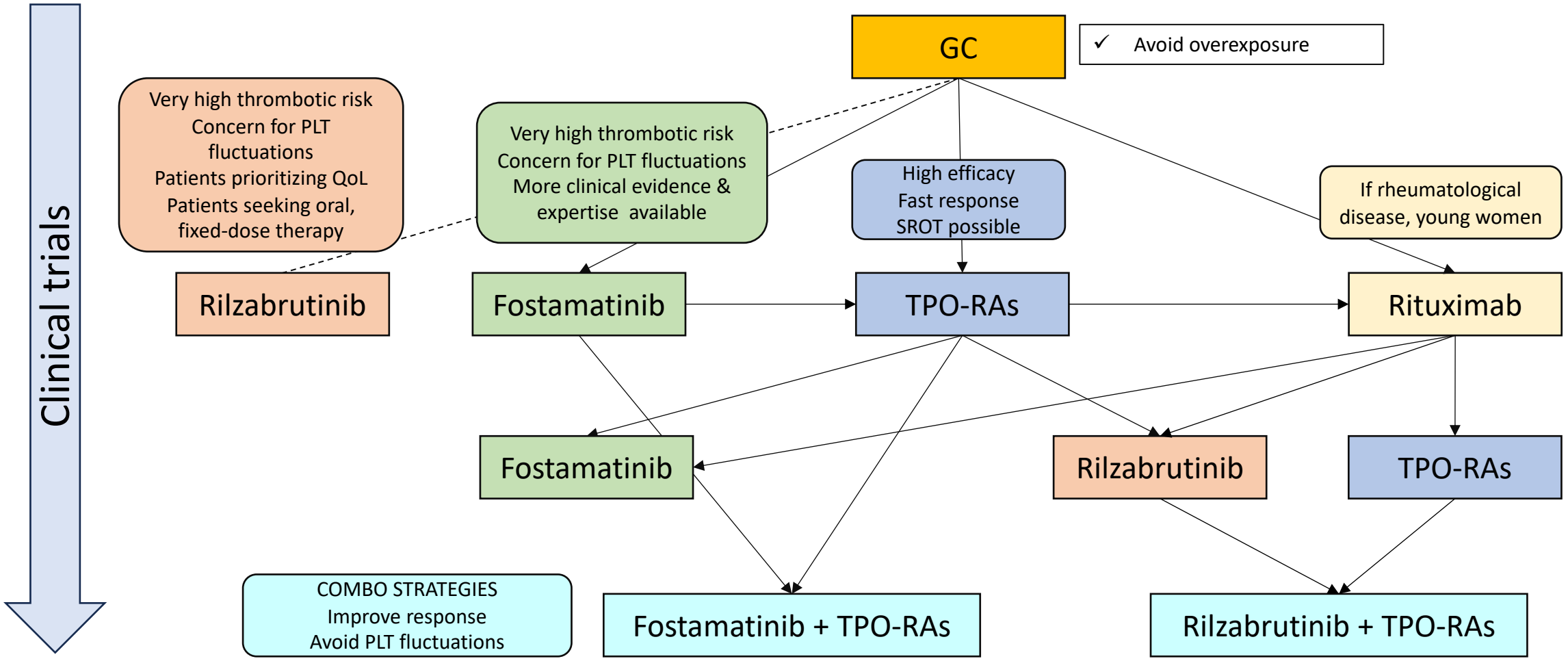
Efgartimod blocks neonatal Fc receptor → accelerates IgG degradation including autoantibodies

Phase 3 completed (ADVANCE IV); approved in Japan. 22% sustained response vs 5% placebo; ~60% IgG reduction; well tolerated

Rozanolixizumab

Broome CM, Lancet. 2023;402(10413):1648-1659. Cuker A, N Engl J Med. 2025. Kuter DJ, N Engl J Med. 2026;394(14):1388-1398. Al-Samkari H. Am J Hematol. 2024;99(11):2178-2190

Future Agenda: ITP «spider-man» therapy (beyond labels)



Speaker's personal opinion, modified from Mingot Castellano ME J Clin Med. 2026 Feb 20;15(4):1625.

Grazie!

francesca.palandri@unibo.it


SIE
Società Italiana di Ematologia
Convegno interregionale
SIE
Delegazioni Emilia Romagna e Toscana
Gli ematologi insieme contro le malattie rare
21 Aprile 2026
Bologna, Aula Prodi
Grazie!
francesca.palandri@unibo.it