Heparin-induzierte Thrombozytopenie

Siemens Healthineers Hämostase-Workshop

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Hintergrund

Heparin-induzierte Thrombozytopenie (HIT)

- Immunologische unerwünschte Wirkung einer Heparintherapie

- Antikörpervermittelte Thrombozytenaktivierung → Thrombingeneration

- Paradox antikoagulantienbedingte Thrombozytopenie ↔

  Thrombogenität und hohes Risiko von Gefässverschlüssen

- Grosse Zahl von Heparingaben → whs. häufigste immunvermittelte

  medikamenteninduzierte Blutzellerkrankung

- Schwierigkeit unspezifische Leitbefunde

    - Thrombozytopenie

    - Thromboembolien

- Thrombozytopenie bei bestimmten Patientengruppen häufig

  (z. B. IPS, HLM, postoperativ u. a. m.)

- Thromboembolie $\rightarrow$ HIT oder Heparin-Versagen?

- Diagnostik: Suche nach PF4/Heparin-Antikörpern

- Antigen: PF4 gebunden an Heparin o. a. sulfatierte

  Glykosaminoglykane

- Antikörperbildung weitaus häufiger als klinische HIT

  (Warkentin, Blood 2000)

- Hoher negativer prädiktiver Wert (NPV) des Antikörpertests

- Positive Resultate $\rightarrow$ Bewertung nach klinischem Kontext

HIT-Risiko wesentlich bestimmt durch

<u>Patientenpopulation</u>

- Operative Fächer, extensives Trauma höheres Risiko (bis 5%),
  z. B. orthopädische Chirurgie, Kardiochirurgie
- Medizinische Pat., Geburtshilfe geringes Risiko (<1% bzw. <0,1%)

<u>Typ des Heparins</u>

- UFH > LMWH > Fondaparinux

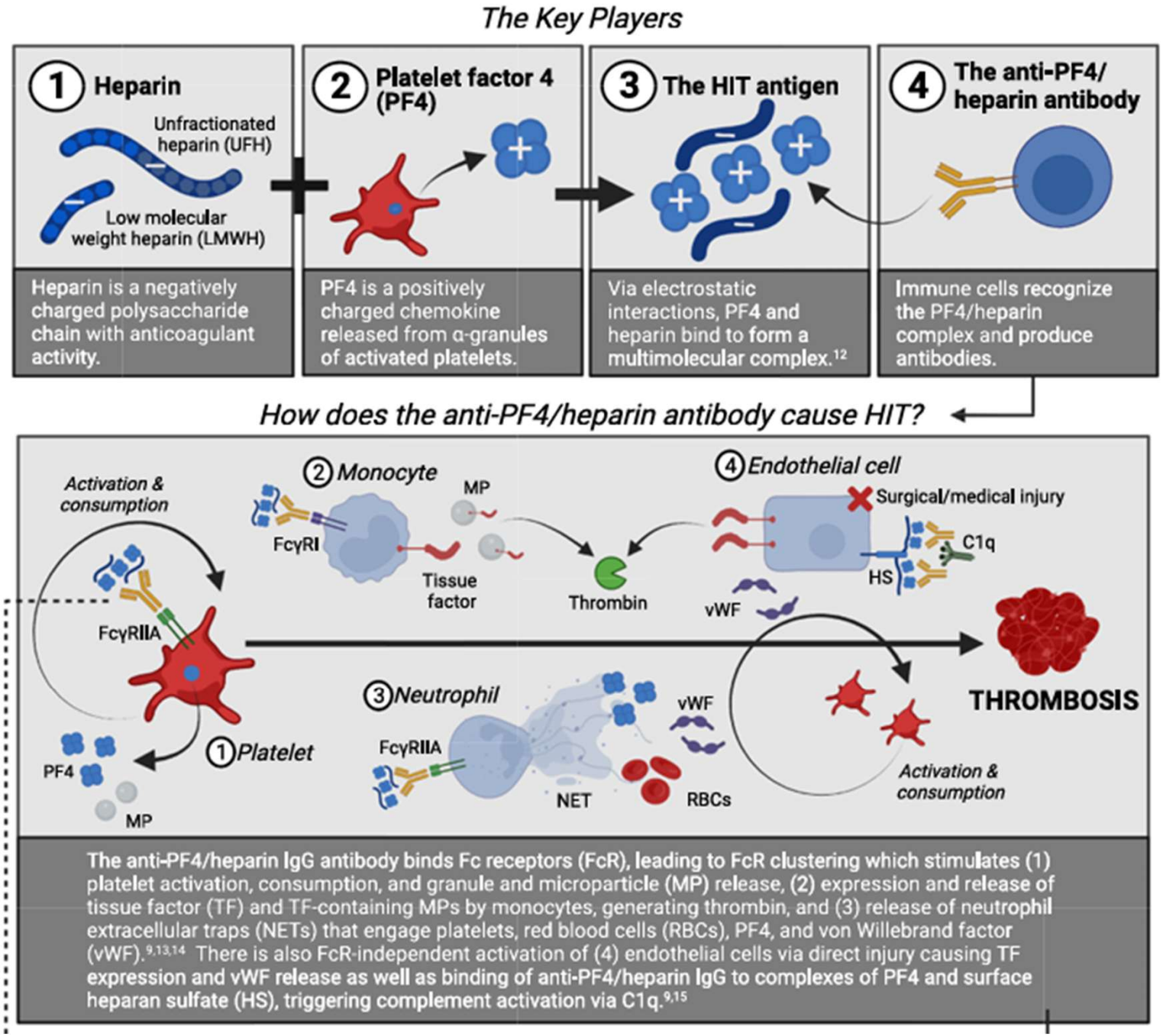
<u>Heparindosis und Stoechiometrie</u>

- Prophylaktische > therapeutische Dosis

(Warkentin, NEJM 1995; Blood 2000; J Thromb Haemost 2010; Greinacher, Thromb Res 2008; Lubenow, Blood 2010)

PF4/Heparin-Antikörper

- IgG pathogenetisch wirksamer Isotyp
- Immunkomplexe → *cross linking* des thrombozytären FcγIIa-Rezeptors
- Plättchenaktivierung, prokoagulatorische Mikropartikel; Aktivierung auch von Monozyten, Endothelzellen
- Gerinnungsaktivierung und starke Thrombingeneration
- Thrombozytopenie = Folge intravaskulärer Plättchenaktivierung



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Klinische Charakteristika

Thrombozytenabfall

- Typischerweise um > 50%
- Bezugspunkt höchster Wert ab Tag 4 der Heparingabe

  (chirugische Patienten u. U. reaktive Thrombozytose)
- Milde Thrombozytopenie (20-100 [meist 50-60] G/l,

  <15 G/l selten durch HIT)
- Typisch Tag 5-14 nach Heparinstart
- Innerh. 12-24 Std. bei kürzlicher Vorexposition (Sensibilisierung)

- PF4/Heparin-Antikörper rascher Titerabfall; nach 3 Monaten

  bei >90% nicht mehr detektierbar

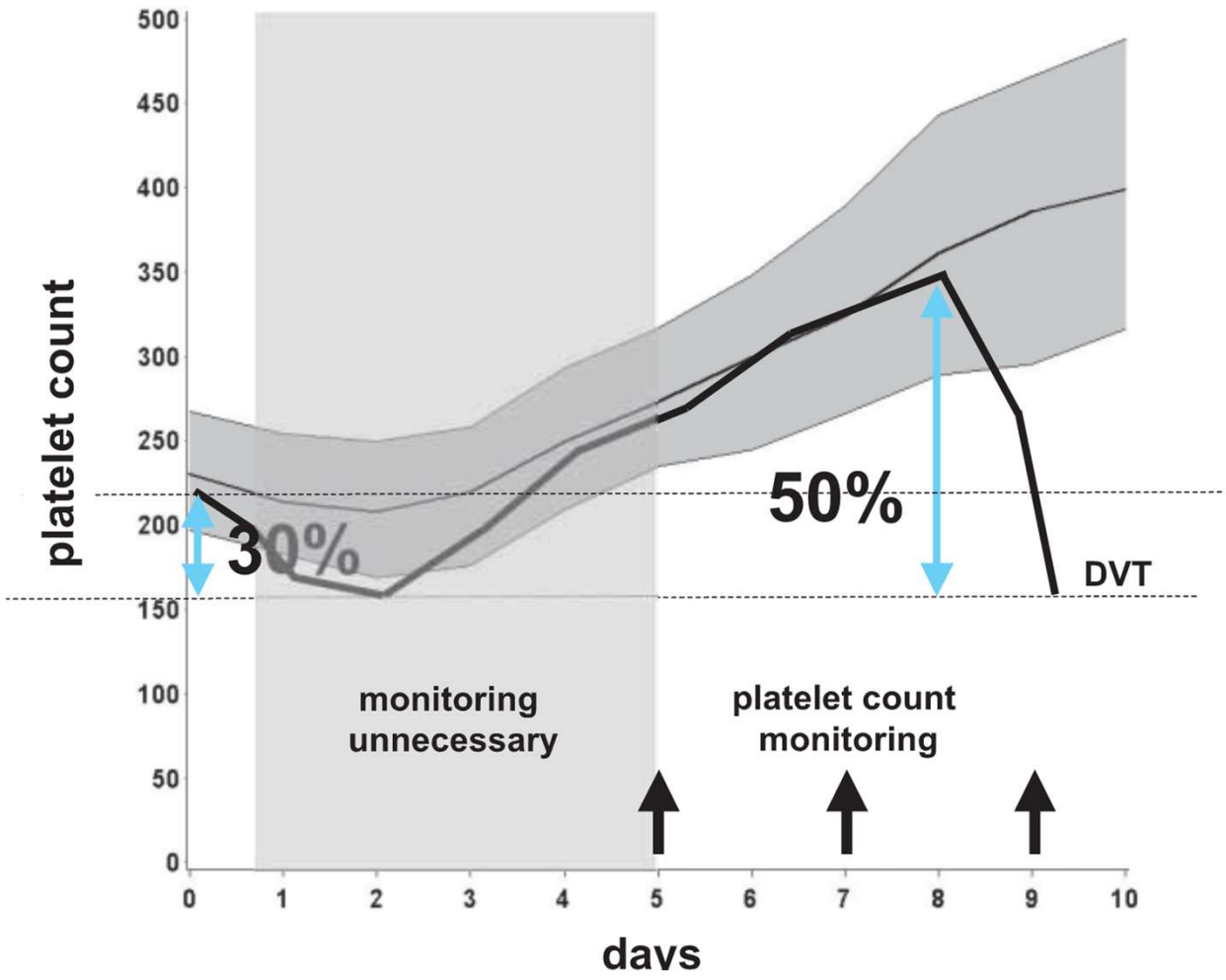
- Kinetik der Antikörper → Monitoring der Thrombozytenzahl

    - vor Heparingabe

    - bei Vorexposition nach 12-24 Std.

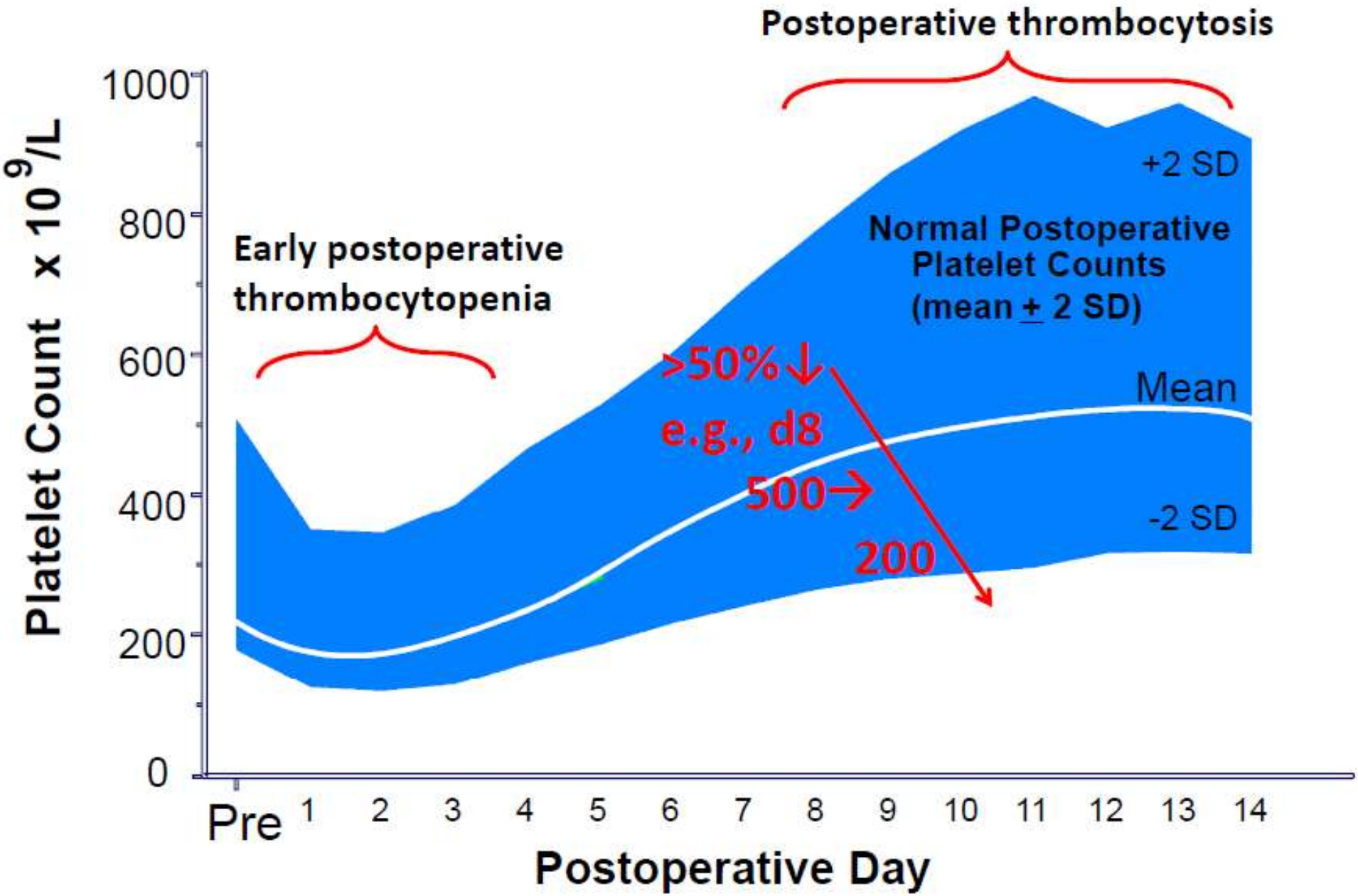
    - an Tag 5, 7, 9

    - erfasst grosse Mehrzahl der Patienten



(Greinacher A. Hämostaseologie 2010; 3017))

Platelet Counts After Surgery



Warkentin et al. *N Engl J Med* 1995;332:1330-5

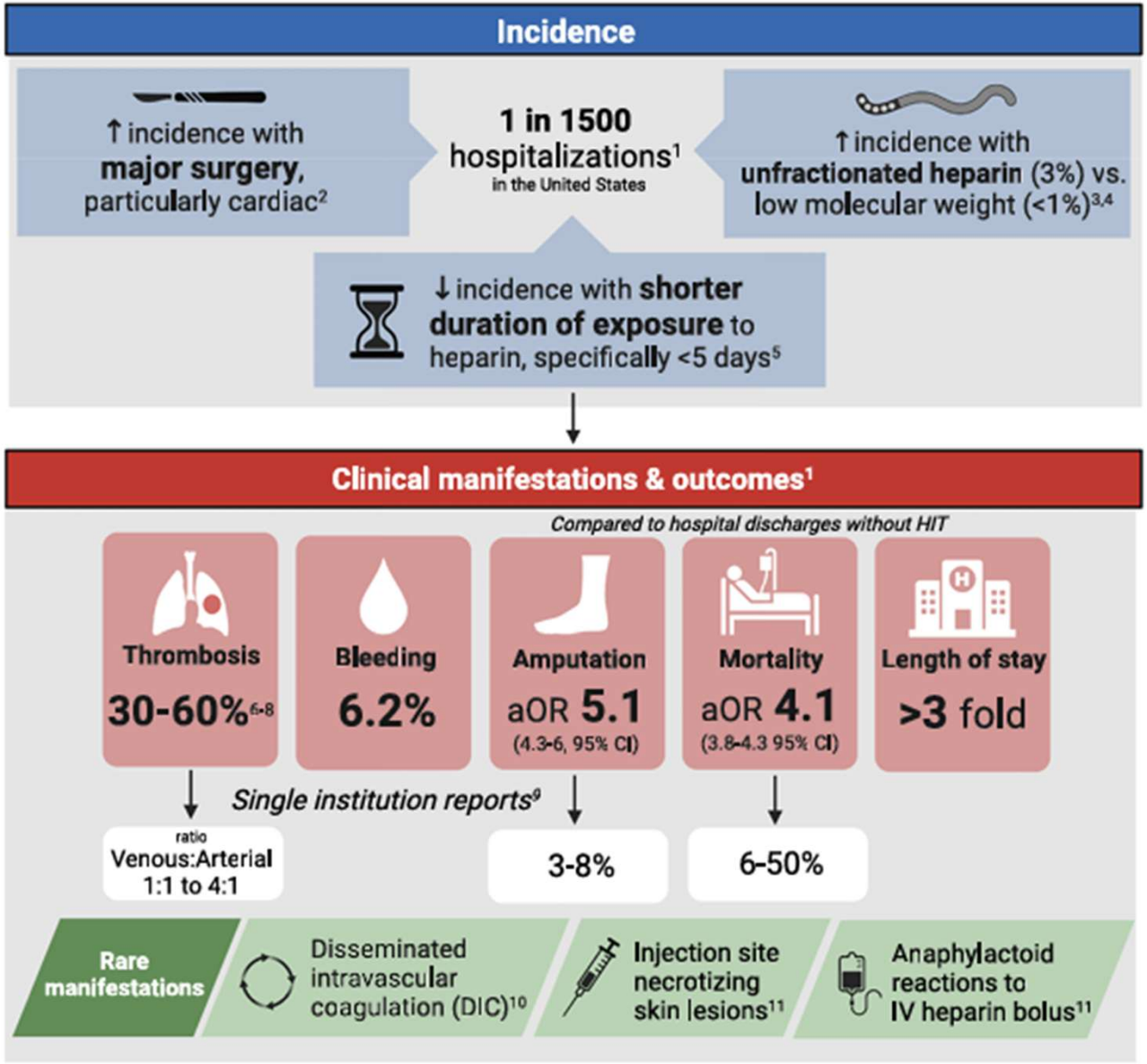
- Thromboembolien häufig und bevorzugt venös, auch arteriell
- Daneben Hautnekrosen, Nebennierenrinden-Nekrosen, anaphylaktoide Reaktionen nach i.v.-Heparinbolus
- Thromboembolien oft zeitnah zum Thrombozytenabfall, aber auch 1-2 Tage vor diesem
- Routinemässige Suche nach Thromboembolien ratsam

	Number of patients with TEC / number of evaluable patients (%)	Odds Ratio	95% confidence interval	p-value
Gender (406 patients)				
Male	80/167 (47.9%)	1.0		
Female	146/239 (61.1%)	1.08	0.63–1.87	0.77
Age (405 patients)				
0–39 years	16/34 (47.1%)	1.0		
40–59 years	57/103 (55.3%)	2.71	0.96–7.61	0.06
60–79 years	134/232 (57.8%)	1.94	0.76–4.96	0.16
≥ 80 years	19/36 (52.8%)	0.98	0.29–3.29	0.98
Underlying disease (400 patients)				
Internal medicine	62/143 (43.4%)	1.0		
General surgery	20/53 (37.7%)	0.72	0.34–1.51	0.38
Cardio-vascular Surgery	12/31 (38.7%)	0.62	0.23–1.68	0.35
Orthopedic and trauma surgery	98/125 (78.4%)	5.34	2.67–10.68	<0.001
Others	30/48 (62.5%)	1.70	0.76–3.84	0.20
Platelet count decrease (319 patients)				
≤ 30%	5/19 (26.3%)	1.0		
> 30–50%	9/29 (31.0%)	1.72	0.40–7.47	0.47
> 50–70%	27/57 (47.4%)	3.63	0.95–13.86	0.06
> 70–90%	101/158 (63.9%)	8.11	2.29–28.66	0.001
> 90%	38/56 (67.9%)	8.79	2.26–34.17	0.002

§ All predictors were simultaneously entered into the model. Hosmer-Lemeshow Chi-square=11.5 (8 df, p=0.18); Nagelkerke R Square=0.27.

Thromboembolien können bereits vor >50%-Abfall der Plättchenzahl auftreten

(Greinacher A. Thromb Haemost 2005; 94: 132)



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Nebennieren-Einblutung (hämorrhagische Nekrosen,

NN-Venenthrombose mit sekundärer Hämorrhagie)

Wesentliche Differentialdiagnosen

- HIT: 2-3% aller HIT; 50% uni-, 50% bilateral ($\rightarrow$ NNR-Insuffizienz)

- Antiphospholipid-Antikörper-Syndrom (mögl. Merkmal eines CAPS)

- Sepsis (Waterhouse-Friderichsen-Syndrom bei fulminanter Meningokokkensepsis)

Diagnostisches Vorgehen

- PF4/Heparin-Antikörper >> HIT

- Erster Schritt: klinische Wahrscheinlichkeit einer HIT

  (valisierter Score, 4-T-Score)

- Metaanalyse zum 4-T-Score *(Cuker, Blood 2012)*

  - niedrig (1-3) hoher NPV 0,998 (niedrig entspricht ≤ 3 P)
  - intermediär und hoch begrenzter PPV (0,14 bzw. 0,64, kombiniert 0,22)

- Konsequenz

  - niedrig → Antikörpersuche nicht indiziert
  - intermediär oder hoch → Antikörpersuche indiziert
  - entscheidend: IgG-Komponente

	Punkte (0, 1 oder 2 für jede der 4 Kategorien: mögliches Maximum = 8 Punkte)		
	2	1	0
Thrombozytopenie	> 50% Thrombozyten-Abfall oder Nadir 20-100 x 10^9/l	30-50% Thrombozyten-Abfall **oder** Nadir 10-19 x 10^9/l	Thrombozyten-Abfall < 30% **oder** Nadir < 10 x 10^9/l
Zeitpunkt *) des Thrombozyten-Abfalls oder anderen möglichen Manifestationen einer HIT	Klarer Thrombozyten-Abfall zwischen Tag 5-10; oder weniger als 1 Tag (falls Heparin-Exposition innerhalb der letzten 100 Tage)	Bild vereinbar mit einer Immunisierung aber nicht sicher (z.B. fehlende Thrombozytenzahlen) oder Thrombozyten-Abfall nach 10 Tagen	Zu früher Thrombozyten-Abfall (ohne kürzliche Heparin-Vor-Exposition)
Thrombose oder andere mögliche Manifestationen einer HIT	Neue Thrombose; Hautnekrose; akute systemische Reaktion nach Heparin-Bolus	Fortschreitende oder rezidivierende Thrombose; erythematöse Hautläsionen; vermutete, noch nicht bestätigte Thrombose	Keine
Andere Ursachen für Thrombozytopenie	Keine andere offensichtliche Ursache für Thrombozyten-Abfall	Mögliche andere Ursache(n) ist (sind) präsent	Bestimmte andere Ursache(n) ist (sind) präsent

4-T-Score zur Ermittlung der Vortestwahrscheinlichkeit für eine HIT

1-3 = niedrig; 4-5 = mittel, 6-8 = hoch (*Warkentin, Curr Hematol Rep 2003)*

Diagnosis, Step 1: **The 4Ts Score**

4Ts Score[21] = Pre-test scoring system to assess the probability of HIT
Calculated as a sum of points from 4 components:

① Thrombocytopenia

Points:

- **2** — Platelet fall > 50% *and* platelet nadir ≥ 20 × 10⁹/L
- **1** — Platelet fall 30%-50% *or* Platelet nadir 10-19 × 10⁹/L
- **0** — Platelet fall < 30% *or* Platelet nadir < 10 × 10⁹/L

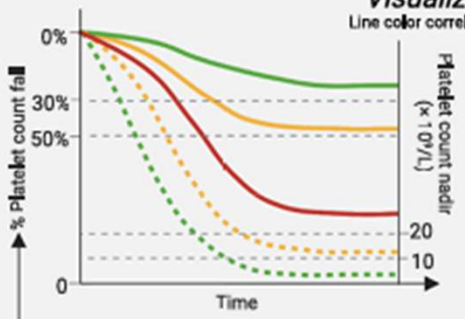
② Timing of platelet count fall

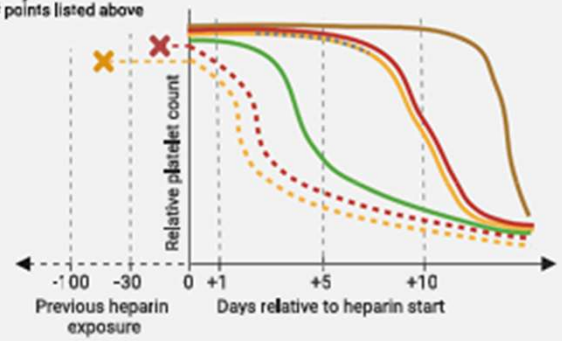
Points:

- **2** — Fall starts days 5-10 (timing is clear) *or* Fall starts ≤ 1 day + heparin exposure within 30 days
- **1** — Fall starts days 5-10 (timing is unclear) *or* Fall starts after day 10 *or* Fall starts ≤ 1 day + heparin exposure 30-100 days ago
- **0** — Fall starts ≤ 4 days without recent heparin exposure

Visualization of points:

Line color correlates with # points listed above





Calculate using *peak* platelet count *after* heparin started (i.e., not necessarily the day heparin started)

% decline is used, not an absolute platelet threshold, as HIT can occur without thrombocytopenia if the peak count is high (e.g., after surgery, myeloproliferative neoplasm).[21]

Calculated based on the *first day* of consistent platelet fall.

In many patients, platelet fall can be *biphasic*, with an initial decline due to a non-HIT insult (e.g. surgery), followed by a rebound and a second, HIT-induced decline.

It takes 5-10 days to produce new IgG antibodies. Immediate HIT onset can occur in those with recent exposure due to presence of previously formed antibodies.[23,24]

Total 4Ts Score
= probability of HIT[22]

- **≥6** High (64%)
- **4-5** Intermediate (14%)
- **≤3** Low (0.2%)

③ Thrombosis or other sequelae

Points:

- **2** — New thrombosis; skin necrosis; acute systemic reaction post-intravenous UFH bolus
- **1** — Progressive or recurrent thrombosis; non-necrotizing (erythematous) skin lesions at heparin injection sites; suspected thrombosis (not proven)
- **0** — None

Includes thrombosis that occurs or progresses in the period of platelet count decline *after* heparin exposure (i.e., not thrombosis that occurred before heparin exposure)

④ OTher causes of thrombocytopenia

Points:

- **2** — None apparent
- **1** — Possible
- **0** — Definite

This variable can be challenging to determine and leads to variation between providers in score determination.[25]

HEP score[26] An alternate score with similar criteria but better performance with less experienced clinicians, likely due to inclusion of defined "other causes of thrombocytopenia":[27]

1. Chronic thrombocytopenia
2. Newly initiated non-heparin med known to cause thrombocytopenia
3. Severe infection
4. Severe disseminated intravascular coagulation (DIC)
5. Indwelling intra-arterial device
6. Cardiopulmonary bypass within 96 hours

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Labordiagnostik

Labordiagnostik nur bei hinreichendem klinischem Verdacht,

kein Routinescreening auf PF4/Heparin-Antikörper

Immunoassays (erster Schritt)

- ELISA (z. B. GTI, Stago, Hyphen)
    - quantitativ; Unterscheidung IgG und gesamt-Ig
    - hoher NPV, jedoch geringe Spezifität (40-80%)
- Automatisierter Chemolumineszenztest (AcuStar HemosIL)
    - quantitativ; Unterscheidung IgG und gesamt-Ig
- Partikel-Immunoassay (PaGIA, DiaMed/BioRad)
    - Agglutination von PF4/Heparin-Komplex-tragenden Partikeln
    - gering quantitativ
    - keine Unterscheidung IgG und gesamt-Ig

Diagnosis, Step 2a: **Laboratory diagnosis, *Immunoassays***

The laboratory diagnosis of HIT is classically divided into 2 steps:
(a) an **immunoassay** and (b) a **functional assay.**

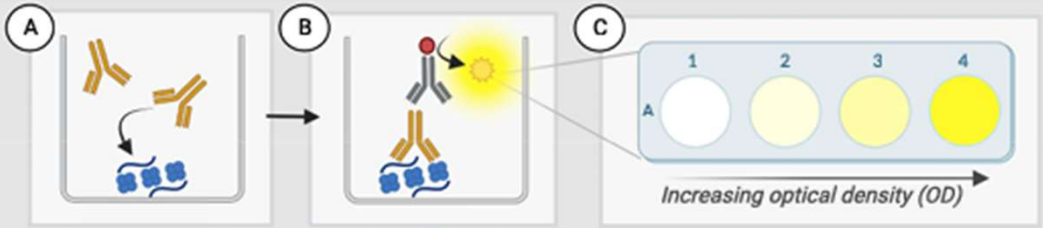
Enzyme Immunoassay (EIA)

The EIA is the prototypical immunoassay, and is used as the standard immunoassay throughout this manuscript

Purpose: Determine if anti-PF4/heparin antibodies are *present*, and if so, their abundance

Performance: High sensitivity (0.97, 95% CI 0.95-0.99), limited specificity (0.82, 95% CI 0.90-0.84)[28]

Specificity varies based on assay used, increasing with IgG-specific assays[28,29] and higher OD thresholds.[30]



Higher OD correlates with higher HIT likelihood[30] and thrombotic risk.[31]

How it works: (A) The plate well is coated with multimolecular complexes of PF4/heparin (or PF4/heparin-like polyanions). Patient serum or plasma is added and anti-PF4/heparin antibodies bind to the complex. (B) Tagged anti-human antibody is added and binds anti-PF4/heparin antibodies. A substrate is added to elicit color change. (C) Degree of color change (optical density, OD) increases with increasing concentration of anti-PF4/heparin antibodies.

***If EIA is positive**, proceed with functional assay (see next page, "Laboratory diagnosis, Functional assays").*
If EIA is negative, HIT is essentially excluded.

Additional immunoassay options: Rapid Immunoassays

Multiple types of immunoassays exist, all with different performance characteristics and different international availability. Selected examples of rapid immunoassays are included below.

Purpose: Similar to EIA but provide results in ≤30 minutes (EIA is often batched, takes hours to perform)

Performance: Varies by assay

		Sensitivity (95% CI)	Specificity (95% CI)	How it works: Method of anti-PF4/heparin detection	Ab class detected
Particle gel immunoassay[32]	PaGIA	0.98 (0.94-1.00)	0.88 (0.91-0.96)	Polymers coated with PF4/heparin complexes	IgG1
IgG-specific chemiluminescence assay[32]	IgG CIA	0.95 (0.90-1.00)	0.94 (0.89-0.99)	Magnetic particles coated with PF4/polyvinyl sulfonate	IgG
Lateral flow immunoassay[32]	LFIA	0.97 (0.92-1.00)	0.91 (0.86-0.96)	PF4-polyanion complexes	IgG
Latex immunoturbidimetric assay[33]	LIA	0.974	0.94	Competitive inhibition of nanoparticles coated with HIT-like antibodies	IgG, IgA, IgM

There is limited guidance on how rapid immunoassays should be incorporated into diagnostic algorithms, and whether additional confirmatory laboratory testing is needed.[32-33]

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Funktionelle Bestätigungstests

Arbeitsaufwändige Methoden zur Erfassung einer

Plättchenaktivierung in Gegenwart von Heparin

- HIPA (Heparin-induzierte Plättchenaggregation)

- SRA (Messung der thrombozytären Serotoninfreisetzung)

Diagnosis, Step 2b: **Laboratory diagnosis, *Functional assays***

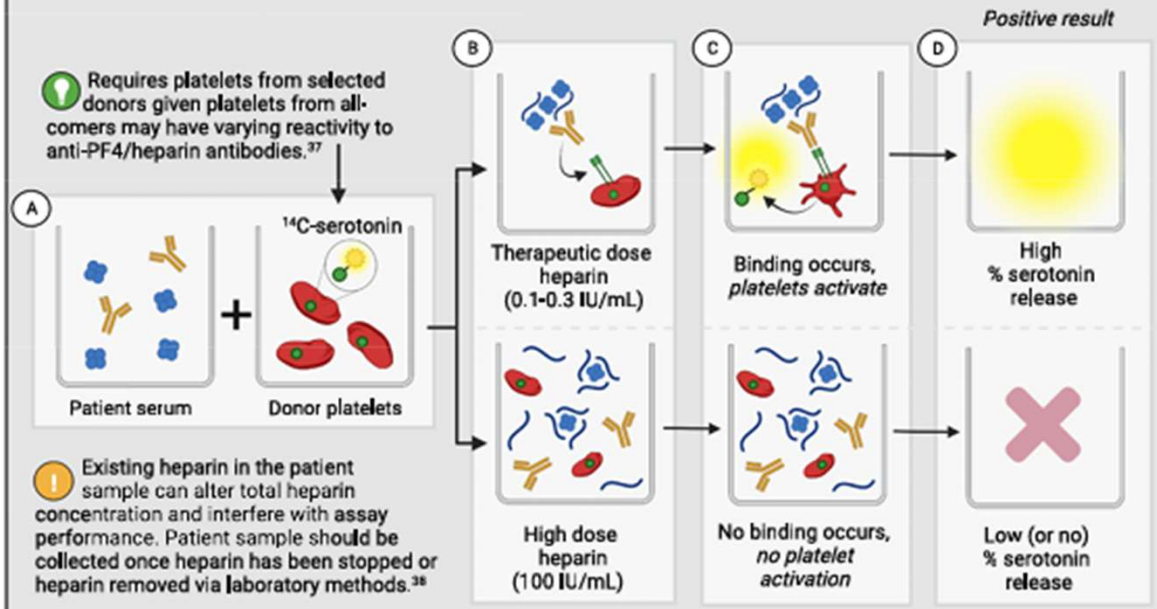
^{14}C-serotonin release assay (SRA)

The SRA is the "gold standard" functional assay[34,35] and is used as the standard functional assay throughout this manuscript

Purpose: Determine if anti-PF4/heparin antibodies are *pathogenic* (i.e. activate platelets)

Performance: High sensitivity (~0.95), high specificity (~0.95)[35]

Due to variation in assay performance and reporting practices,[36] real-world performance may vary from literature reports.



How it works:[34,35] (A) Patient serum is combined with washed donor platelets radiolabeled with ^{14}C-serotonin. (B) At least two different doses of heparin are added. At therapeutic dose, the HIT antigen forms and is recognized by the anti-PF4/heparin antibody. At high dose, heparin disrupts the multimolecular HIT antigen[34,39] so the anti-PF4/heparin antibody cannot bind. (C) When the antigen/antibody complex binds to the platelet Fc receptor, platelets activate and release granules containing ^{14}C-serotonin. Radioactivity is measured and converted to a % ^{14}C-serotonin release. (D) A diagnosis of HIT (i.e. positive SRA) is generally characterized by strong activation (high % serotonin release) with therapeutic dose heparin *plus* inhibited activation (low % serotonin release) with high dose heparin. A negative SRA shows no activation at either concentration. An indeterminate SRA shows activation at both heparin concentrations.

Additional functional assays

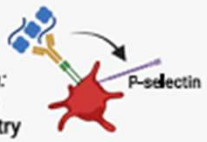
Multiple functional assays exist that differ by the method used for measuring platelet activation:

Heparin-induced platelet activation assay (HIPA)[40]

Measurement of activation: Visual assessment of decreased turbidity with platelet activation

P-selectin expression assay (PEA)[41-43]

Measurement of activation: Platelet P-selectin (CD62p) expression by flow cytometry



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Verbesserung der Spezifität → Vermeidung von Überdiagnosen

- Kombination Antikörpersuchtest (Immunoassay) und funktioneller Test
    - hohe Sensitivität der Immunoassays
      (negativ → HIT praktisch ausgeschlossen)
    - höhere Spezifität der funktionellen Tests
- Antikörpertiter im Immunoassay korreliert mit Wahrscheinlichkeit
  plättchenaktivierender Antikörper im funktionellen Test
    - ELISA: OD < 1,0 (schwach) nur 5-15% positiv
    - Chemolumineszenztest: > 4 U/ml (stark) > 85% positiv
    - PaGIA: ≥ 1:4 hohe HIT-Wahrscheinlichkeit (95%)
- Messung des IgG anstelle des unspezifischen gesamt-Ig
- Inhibitionstest (Heparin in hoher Konzentration)

(Warkentin, J Thromb Haemost 2011; Bakchoul, Int J Lab Hem 2015)

Fortschrittliche Algorithmen zur Verbesserung der Diagnostik:

TORADI-HIT (*Nilius H. E Clin Med 2022; 55:101745*)

TORADIHIT

INTENDED USE | ALGORITHM | EVIDENCE | PERFORMANCE | LIMITATIONS | LEGAL NOTICE

The TORADI-HIT algorithm is a validated diagnostic instrument to calculate the probability of HIT in individual patients.

Platelet count (nadir) [10^{-9}/L]

66

CRP (C-reactive protein; if available) [mg/L]

22

Type of immunoassay

Acustar HIT-IgG (CLIA) [U/mL]

Numeric immunoassay test result

3.6

For the Diamed ID-H/PF4 use Titer $^{-1}$. E.g. use 64 instead of 1:64

Did the patient receive unfractioned heparin during the current hospital stay?

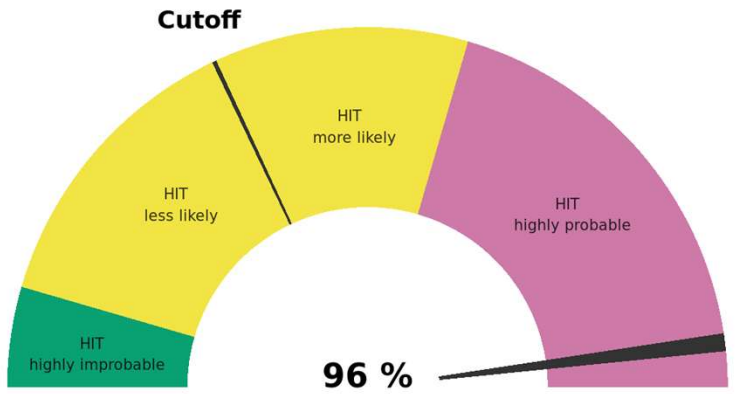
Yes

Timing of the thrombocytopenia

Day 5-10, or day 1 if recent heparin exposure

Other causes of thrombocytopenia

Results



Interpretation

The classification probability of HIT is 96 %. The probability of HIT is very high with values in the red area (90% of HIT-positive patients have values in the red area).

Personalized Clinical Diagnostics

von: *www.toradi-hit.dbmr.unibe.ch*

Diagnosis: **Putting it all together**

Why use risk scores before laboratory testing?

- Thrombocytopenia is common in hospitalized patients, and it is rarely due to HIT[44]
- The 4Ts Score has excellent *negative* predictive value (can exclude HIT)[22]
- EIA is sensitive, not specific. False positives are common.

Choosing Wisely recommendation to avoid laboratory testing in patients with low probability 4Ts Score[45,46]

Why not wait for the laboratory results to change anticoagulation?

Without appropriate therapy in a patient with HIT, the daily risk of complications (combined risk of thromboembolism, amputation, and death) is 6.1%.[47]

Risk of HIT can be further stratified based on EIA optical density (OD) and 4Ts Score:

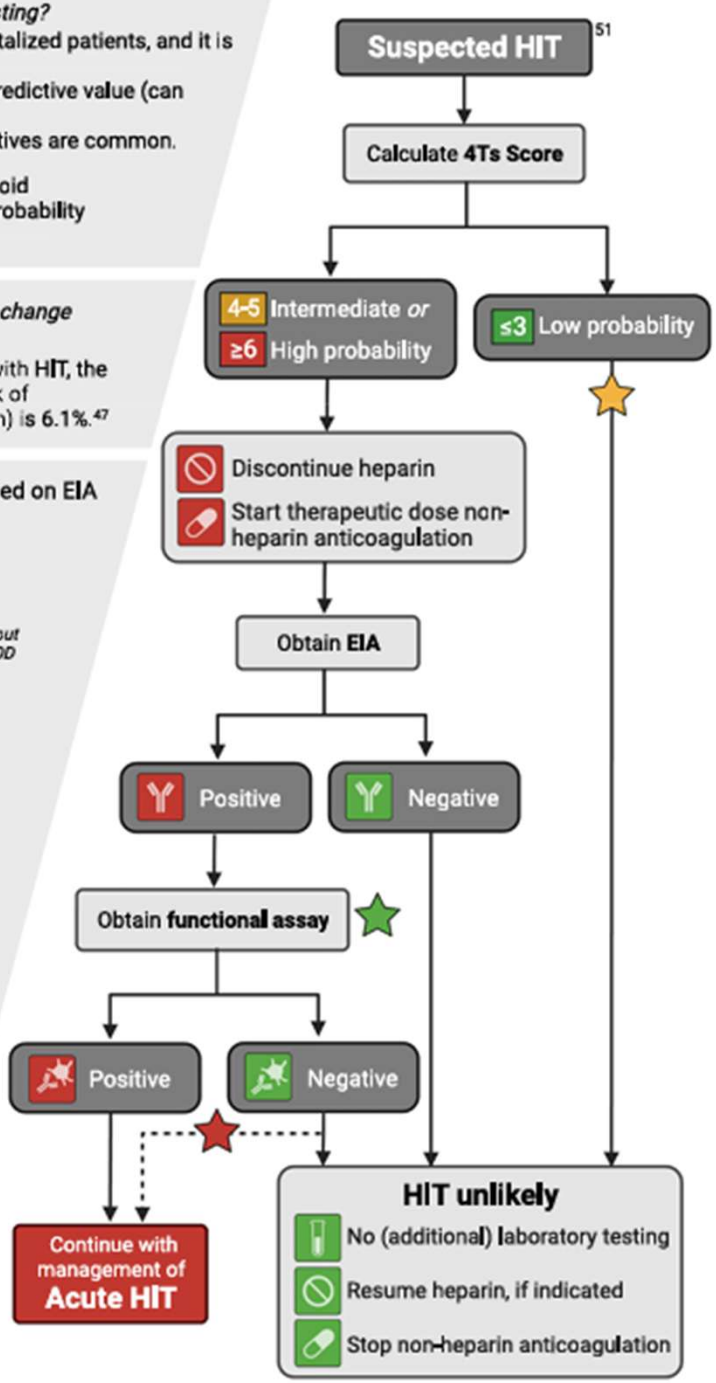
4Ts Score

EIA OD	4–5	6–8
	14%	64%
	without EIA OD	
<0.6	0%	0%	0%
0.6–1.49	5–25%	16%	68%
1.5–1.99	50%	53%	93%
2+	90%	92%	99%
	without 4Ts Score		

Lighter shades indicate probability of the individual component (4Ts Score without EIA OD,[22] EIA OD without 4Ts Score[48]). Darker shades indicate probability accounting for combination of 4Ts Score and EIA OD.[49,50] *Exact probabilities will vary with different EIA assays.*

In patients with a high probability 4Ts score and a strongly positive EIA, a functional assay may not be necessary.[51]

Rarely, functional assays are falsely negative.[52] In patients with a high 4Ts score and high EIA OD, treatment for HIT can be considered despite a negative functional assay.[51]



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Therapie

- Absetzen von Heparin in jeglicher Form

- Alternatives Antikoagulans, meist direkter Thrombininhibitor

  (unabhängig davon, ob manifeste Thromboembolien)

- Keine Vitamin K-Antagonisten in der Initialphase einer HIT

    - Gefahr mikrovaskulärer Verschlüsse und Nekrosen

    - meiste Amputationen bei HIT wg. VKA und DIC

- Umstellung auf OAK erst im Verlauf

    - stabile Normalisierung der TC-Zahl (>150 G/l)

    - niedrige VKA-Aufsättigungsdosis

    - 5-tägige Überlappungsphase mit alternativem Antikoagulans

Verfügbare Substanzen zur Initialtherapie der HIT

- Direkte Thrombininhibitoren (DTI)
  - Argatroban = Argatra®
  - (Bivalirudin = Angiox®)
- Antithrombin-abhängige Faktor Xa-Antagonisten
  - Danaparoid = Orgaran®
  - Fondaparinux = Arixtra®
- *on label*: Danaparoid, Argatroban
- *off label*: Bivalirudin, Fondaparinux

(Alberio L. Swiss Med Wkly 2020; 150: w20210)

Monitoring

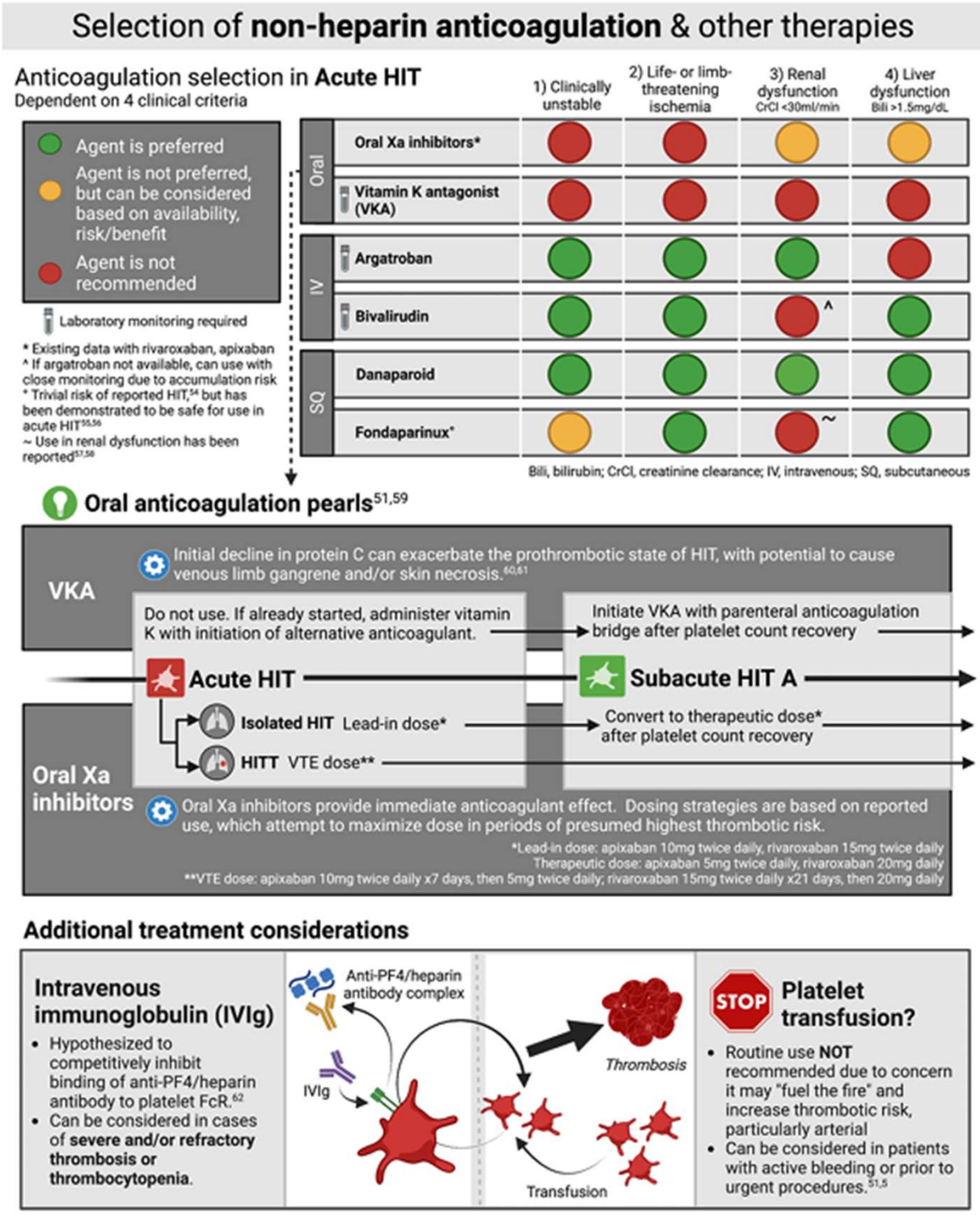
Substanz	Handelsname	1. Wahl	Hilfsparameter
Argatroban	Argatra®	anti-FIIa	aPTT, TZ (*)
Bivalirudin	Angiox®	anti-FIIa	aPTT, TZ (*)
Danaparoid	Orgaran®	anti-FXa	
Fondaparinux	Arixtra®	anti-Fxa	

(*) Quick/INR werden ebenfalls beeinflusst

Halbwertszeiten

Substanz	Handelsname	HWZ	Exkretion
Argatroban	Argatra®	50 Min.	Hepatobiliär
Bivalirudin	Angiox®	25 Min.	Enzymatisch
Danaparoid	Orgaran®	19-25 Std.	Renal
Fondaparinux	Arixtra®	17-21 Std.	Renal

Spezifisches Antidot für keine der o. g. Substanzen → kurze HWZ vorteilhaft



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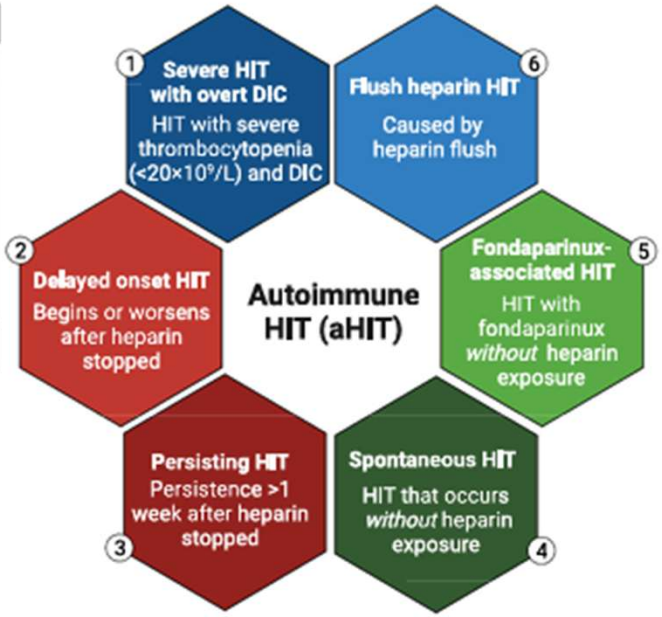
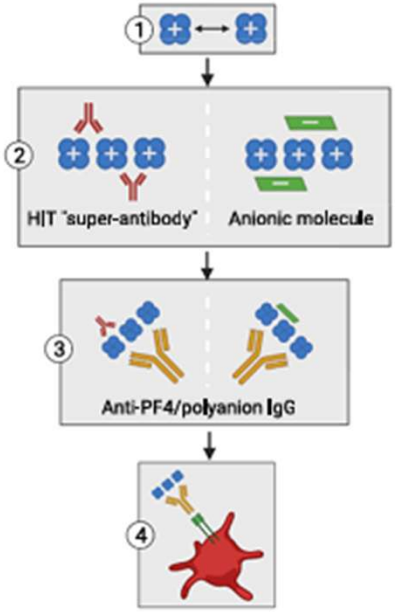
Autoimmune HIT (aHIT)

What is autoimmune HIT?

- Rarely, the clinical manifestations of HIT can develop via **heparin-independent platelet activation**.
- Autoimmune HIT (aHIT) refers to any HIT-related platelet count fall that begins or persists in the absence of heparin.[70,71]
- Six subtypes of aHIT have been described.
- Notably, spontaneous HIT has been reported to occur most commonly after knee replacement or infection.[71]



Pathophysiology



(1) Positively charged PF4 tetramers repel one another. (2) In aHIT, a non-heparin molecule overcomes this electrostatic repulsion, bringing individual PF4 tetramers together to form large multimolecular PF4 complexes. The exact non-heparin molecule able to perform this action is debated and may vary based on aHIT subtype, but proposed examples include HIT "super-antibodies" that bridge two PF4 tetramers and/or highly anionic molecules which could include chondroitin sulfate (released from joint cartilage in joint surgery), polyphosphate, and/or nucleotides such as DNA and RNA.[70,71] (3) The PF4 complexes then serve as antigens bound by anti-PF4/polyanion IgG, which (4) trigger activation of platelets and other cell types.

Principles of diagnosis & management of aHIT syndromes are ***the same as classical HIT,*** with some unique considerations:

When to suspect?

- Thrombosis *and*
- Thrombocytopenia

that is otherwise unexplained *even in the absence of heparin exposure*

Diagnosis

- Immunoassay +
- Functional assay +

Functional assay may show platelet activation in the absence of heparin, but a non-heparin control is not performed in all laboratories.

Management

- Avoid heparin
- Use non-heparin anticoagulation

Experts recommend consideration of high-dose IVIg in severe cases.[70,71]

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Zusammenfassung

- HIT = schwerwiegende prothrombotische immunologische Medikamentennebenwirkung
- Risiko je nach klinischem Kontext (Patientenkollektiv, Heparinart, Heparindosis)
- Wichtiger Stellenwert der Vortestwahrscheinlichkeit
- Verfügbarkeit sensitiver und spezifischer Labormethoden zum Nachweis von PF4/Heparin-Antikörpern (IgG)
- Therapie alternatives Antikoagulans, alleiniges Sistieren der Heparintherapie reicht nicht aus
- Rasches Verschwinden der Antikörper (meist nach 3 Monaten)
- HIT-artige Entitäten (Autoimmun-HIT, VITT)