Thrombocytopénie induite à l'héparine

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Congrès SSVQ, Québec, 23 novembre 2019

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Conflit d'intérêts

- Aucun conflit à déclarer

Objectif

- Revisiter l'algorithme du diagnotic de la thrombocytopénie à l'héparine (TIH)
- Voir les nouveatés en terme
  - Investigations
  - Traitements
  - Formes atypiques
- Traitement de la TIH par AOD

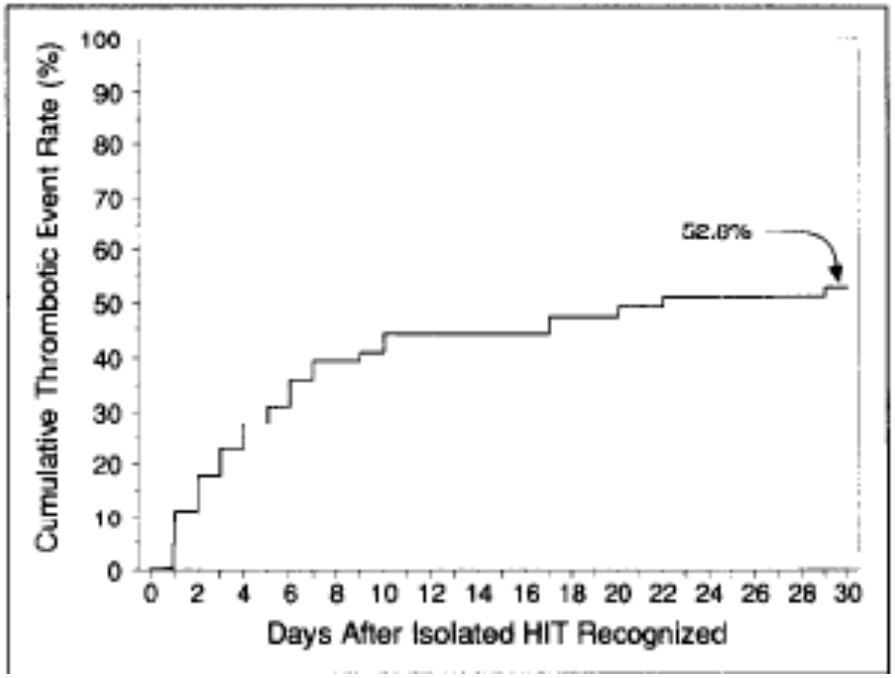
Héparine et TIH : historique

- 1950 : héparine comme traitement des thromboses
- 1957 : premiers cas décrits de thrombose et héparine
- 1979 : « White clot syndome »
- Fin des années 80 : type 1 et 2

Historique

- Formes historiques connues de TIH:
  - Type 1
    - Non immunologique
    - Habituellement vue au début du traitement sans exposition antérieure à l'héparine
    - Attribuée à une liaison directe entre l'héparine et les plaquettes
    - Résolution avec malgré la continuation de l'héparine sans autre traitement
  - Type 2
    - Immunologique
    - Associée à liaison de la partie variable d'une immunoglobuline IgG (Fab) avec le complexe PF4-héparine et de la partie cristallisable (Fc) avec le récepteur des immunoglogulines sur les plaquettes
    - Associée à des thromboses

Risque de thrombose



Risque élevé de thrombose si non traitée

November 1996 The American Journal of Medicine® Volume 101

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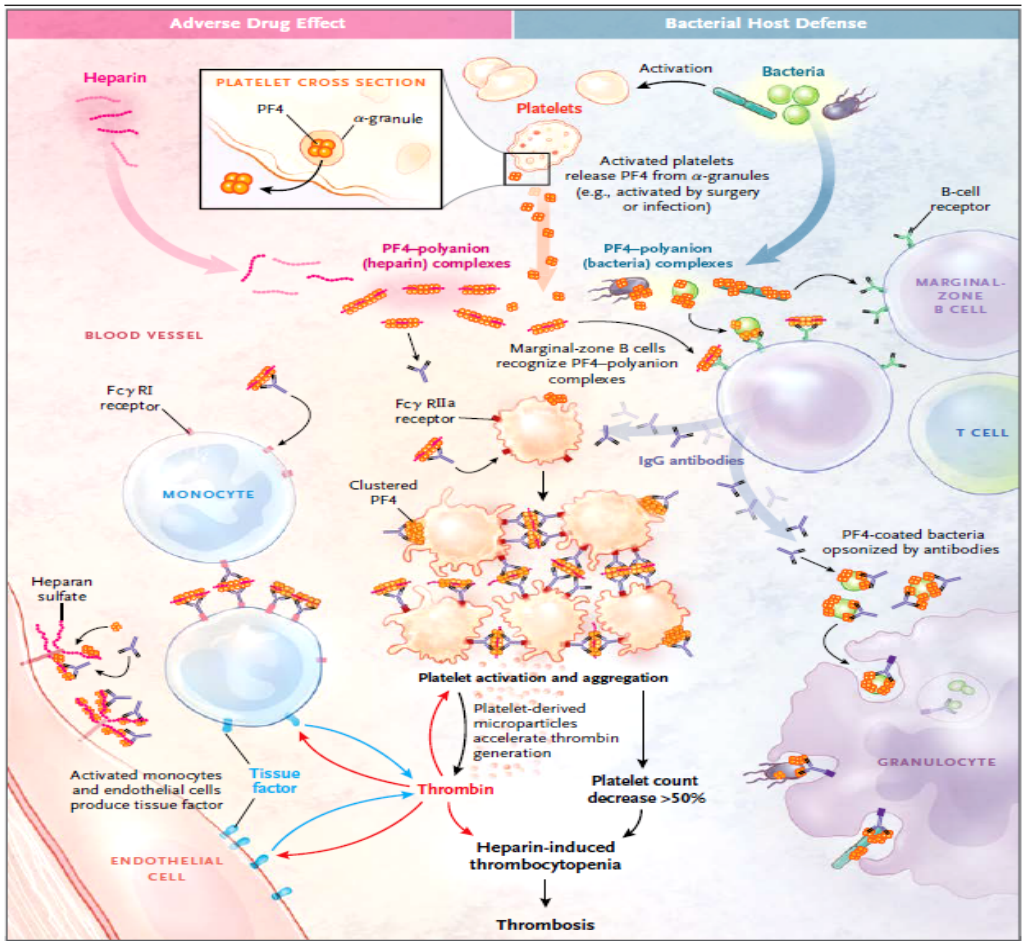


Liaison héparine-PF4 et plaquettes via l'anticorps formé

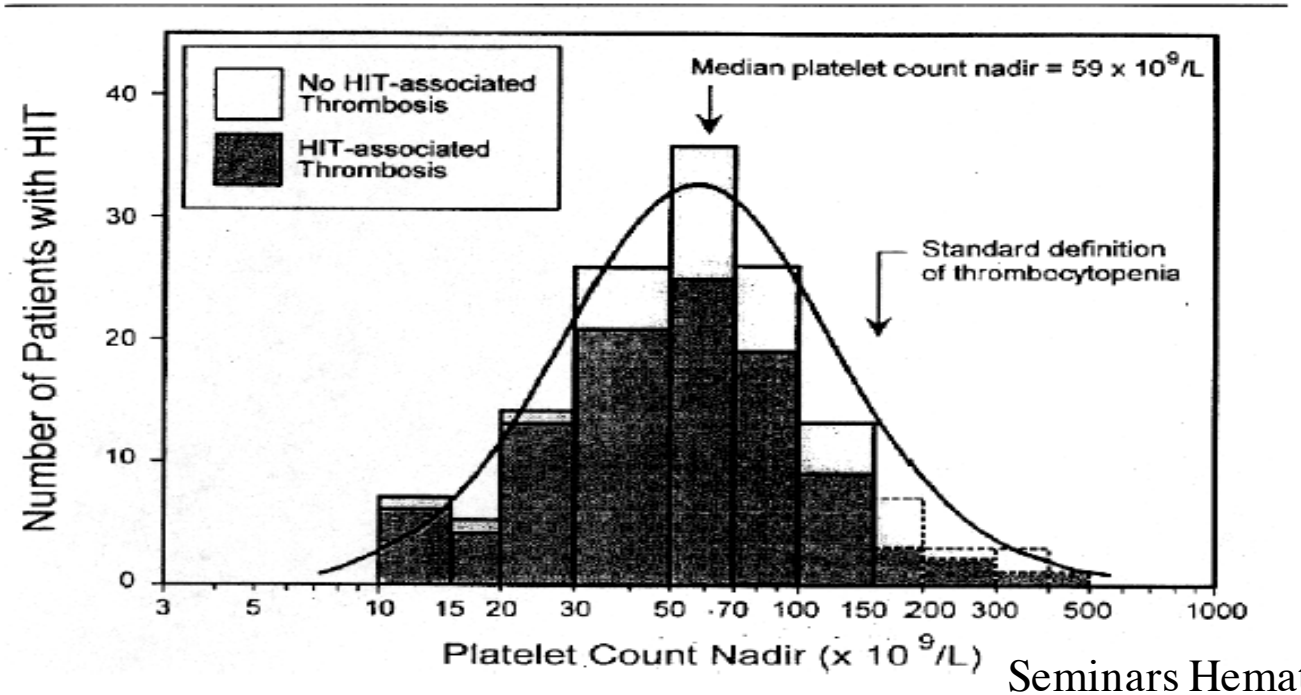
Génération de thrombine ++

N ENGL J MED 373;3 NEJM.ORG JULY 16, 2015

Populations à risque

- Patients de chirurgie cardiaque
  - Héparine pour la circulation extracorporelle
- Patients de chirurgie orthopédique
- Patients médicaux

Décompte plaquettaire usuel au diagnostic



Seminars Hematol, 1998

Chirurgie cardiaque : décompte plaquettaire usuel

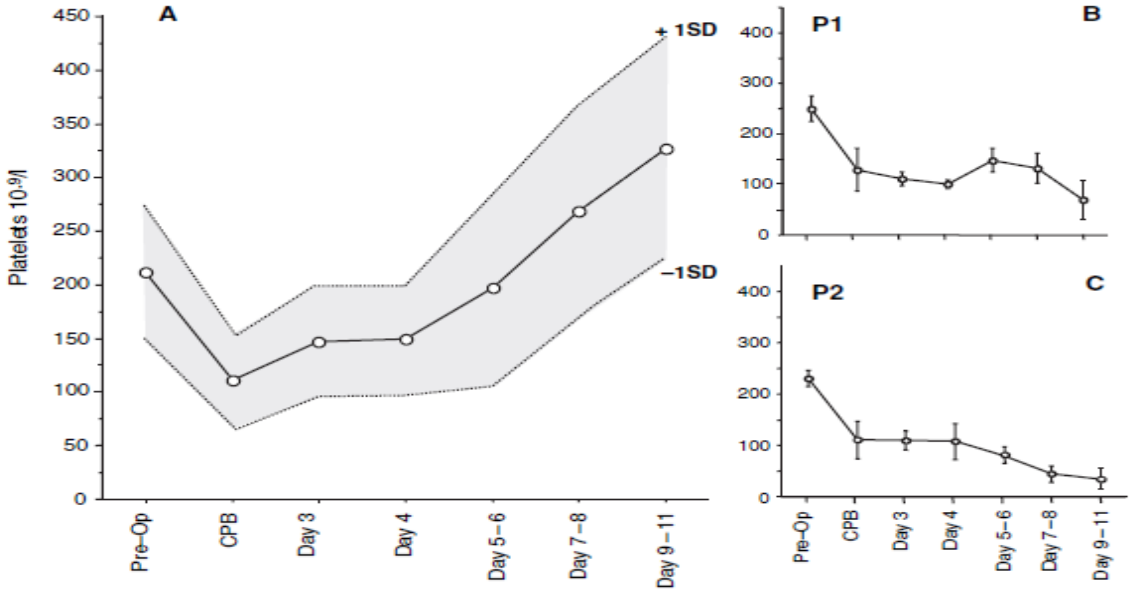


Fig 1. Retrospective cohort. (A) Normal changes in PC after CPB in patients without antibodies to H–PF4 ($n = 245$, mean ± 1 SD). (B, C) PC patterns associated with HIT. Patterns P1 and P2 were defined according to the evolution of PC (mean ± 1 SEM) in four and two HIT patients respectively.

British Journal of Haematology, **128**, 837–841

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Risque de développement : chirurgie cardique

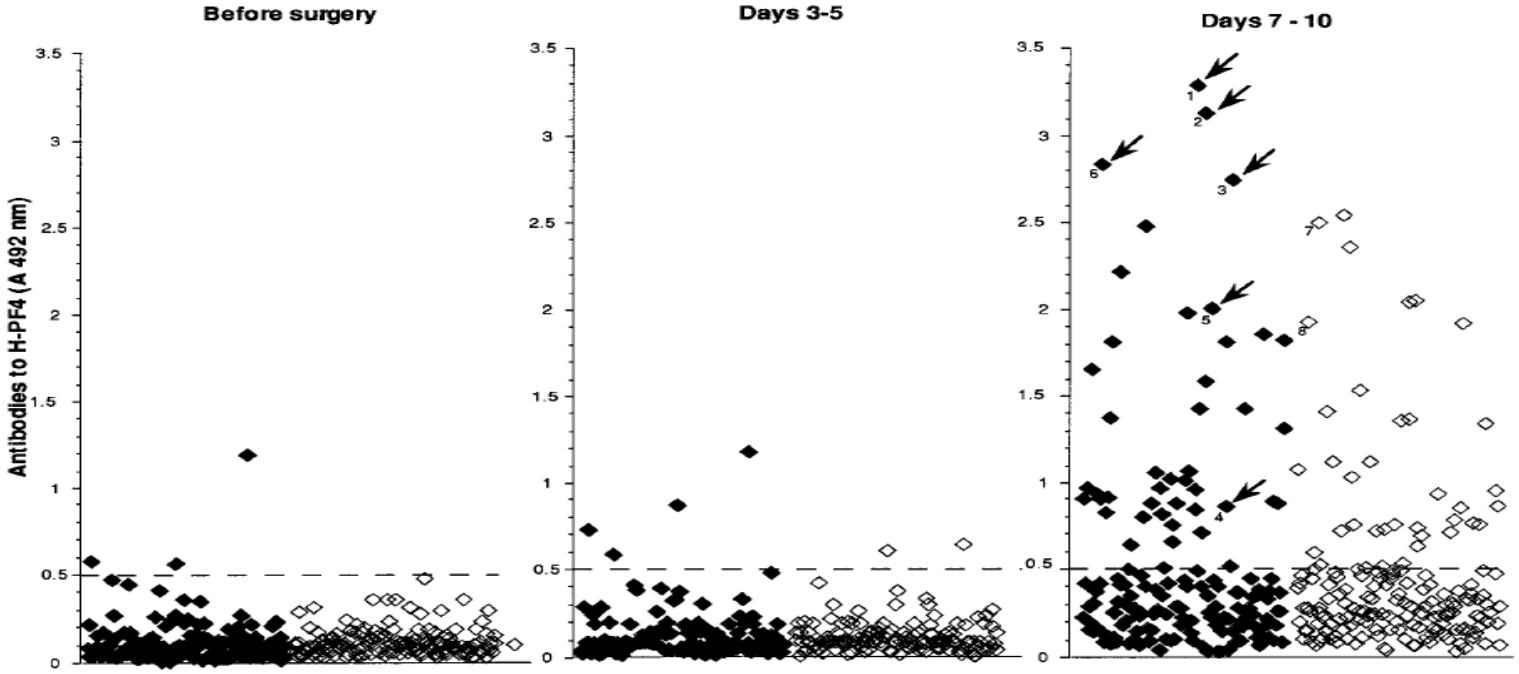

Figure 1. Development of antibodies to H-PF4 after CPB. Antibodies to H-PF4 before and after CPB in patients receiving UFH (◆) or LMWH (◇) postoperatively. $A_{492} \geq 0.5$ was taken into account (dotted line). The 8 patients with antibodies and positive SRA are numbered. Arrows indicate patients with HIT.

Pouplard et al **May 18, 1999**

8 cas de TIH décrits
6 HNF
2 HBPM
Fréquence : 1-2 %

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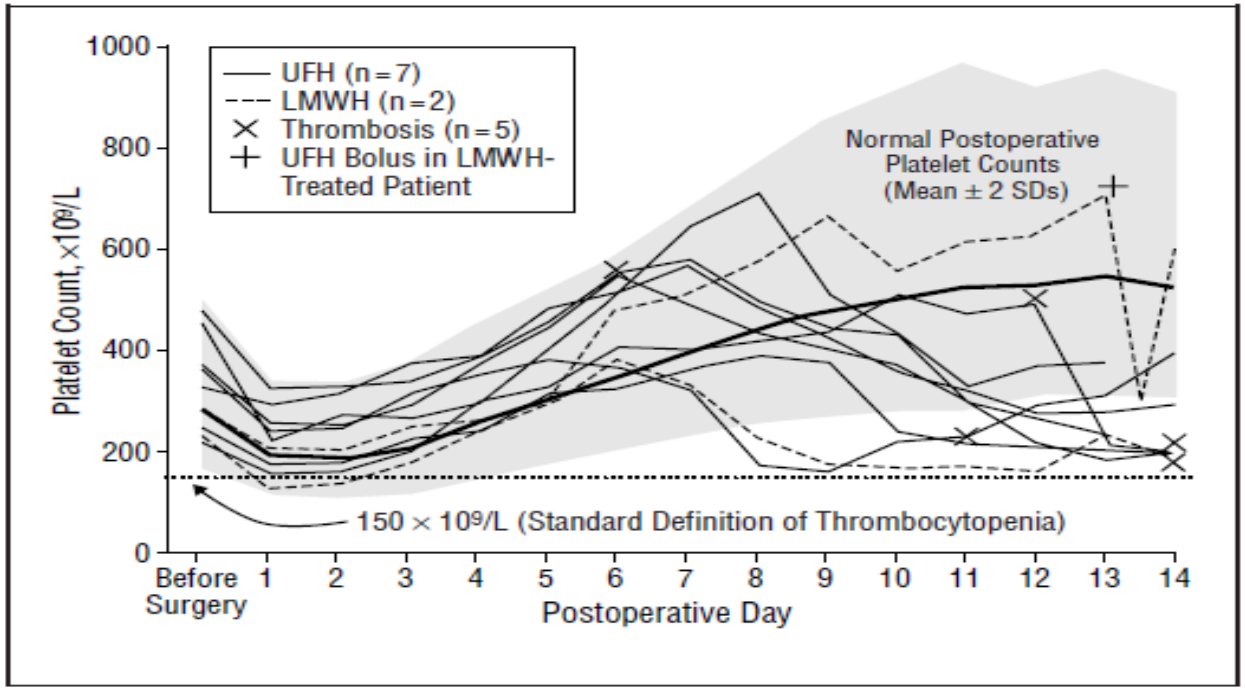
Chirurgie orthopédique : décompte plaquettaire usuel



Fréquence : 5 %

ARCH INTERN MED/ VOL 163, NOV 10, 2003

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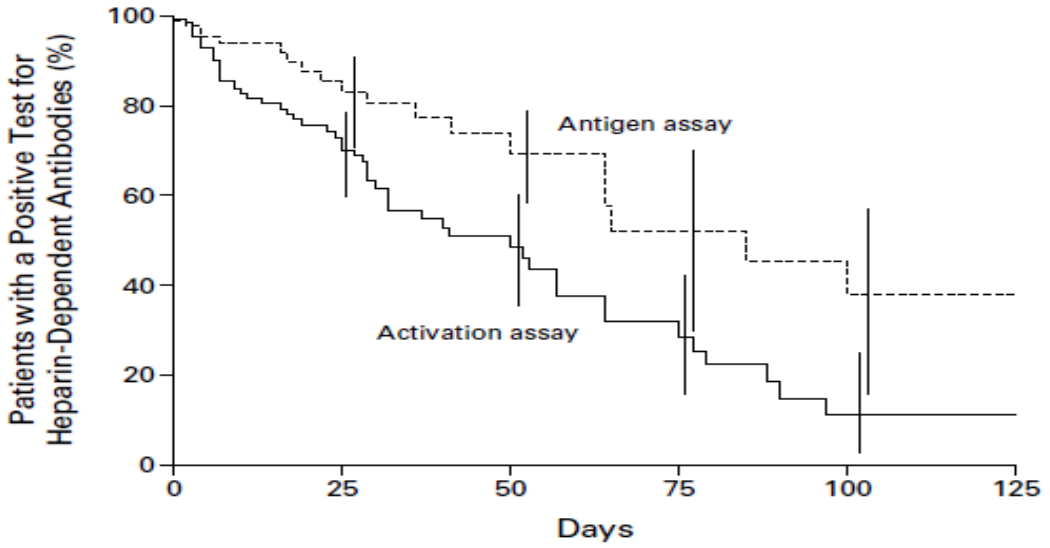
Histoire naturelle biologique des anticorps en TIH



No. at Risk					
Antigen assay	93	36	17	8	6
Activation assay	144	53	23	10	3

Figure 2. Kaplan–Meier Analysis of the Proportion of Patients with Heparin-Dependent Antibodies after an Episode of Heparin-Induced Thrombocytopenia.

N Engl J Med, Vol. 344, No. 17 · April 26, 2001 ·

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"Timing" de la thrombocytopénie

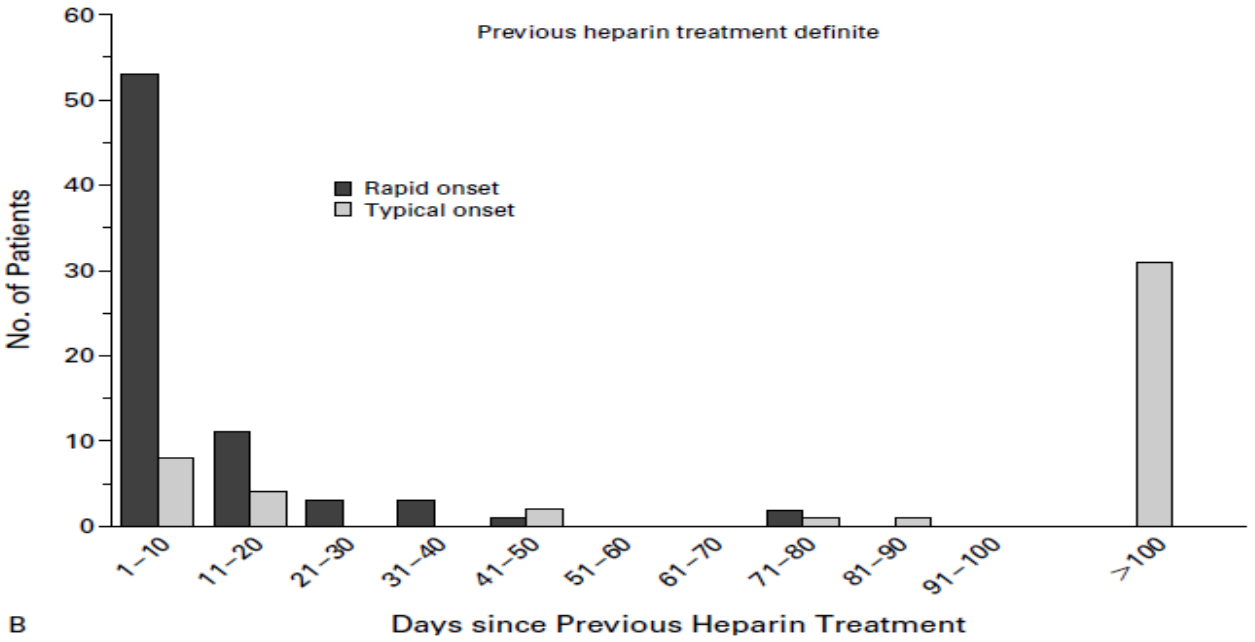

Figure 1. Temporal Patterns of Heparin-Induced Thrombocytopenia in Relation to Previous Treatment with Heparin.

N Engl J Med, Vol. 344, No. 17 · April 26, 2001 ·

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Outils diagnostiques : score 4T

Table 1 Pretest scoring system for HIT: the 4 T's

4T's	2 points	1 point	0 point
Thrombocytopenia	Platelet count fall >50% and platelet nadir ≥20*	Platelet count fall 30–50% or platelet nadir 10–19	Platelet count fall <30% or platelet nadir <10
Timing of platelet count fall	Clear onset between days 5–10 or platelet fall ≤1 day (prior heparin exposure within 30 days)†	Consistent with days 5–10 fall, but not clear (e.g. missing platelet counts); onset after day 10‡; or fall ≤1 day (prior heparin exposure 30–100 days ago)	Platelet count fall <4 days without recent exposure
Thrombosis or other sequelae	New thrombosis (confirmed); skin necrosis§; acute systemic reaction postintravenous unfractionated heparin (UFH) bolus	Progressive or recurrent thrombosis¶; Non-necrotizing (erythematous) skin lesions§; Suspected thrombosis (not proven)**	None
Other causes for thrombocytopenia	None apparent	Possible††	Definite††

Journal of Thrombosis and Haemostasis, **4**: 759–765

Probabilité de TIH

Score faible 0-3 : 1,9%

Score intermédiare 4-5 : 6,7 %

Score élevé 6-8 : 36,6 %

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Outils diagnostiques : immunoessais anti-PF4

- Test quantitatifs
  - ELISA (*enzyme-linked immunoassays*)
    - Polyspécifique anti-PF4 IgG-IgA-IgM
    - IgG spécifique : plus spécifique que le test IgG-A-M
    - Utilité : très courant
    - Inconvénients :
      - fait en groupe (*batch*)
      - pauvre valeur prédictive positive

Anti-PF4 ELISA : Pauvre valeur prédictive positive

Table 1 Results of the serological investigation of 500 patients with suspicion of HIT in the heparin-induced platelet-activation (HIPA) test, a modified platelet factor 4/heparin (PF4/hep) IgG-specific enzyme-linked immunosorbant assay (IgG-ELISA), the commercially available PF4/hep IgM/A/G ELISA (Poly-ELISA) and in the particle gel immunoassay (PaGIA). The probability of HIT was evaluated using the Greifswald modification of the 4T's scoring system

		Additionally reactive				4T's scoring						
Test	Positive sera	HIPA	IgG-ELISA	Poly-ELISA	PaGIA	Sera with low probability	Sera with intermediate probability	Sera with high probability	Sensitivity (%)	Specificity (%)	NPV (%)	PPV (%)
IgG-ELISA (cut-off = 1.0)	56	32	–	56	45	0	11	45	91.4	94.8	99.3	57.1
IgG-ELISA (cut-off = 0.65)	69	34	–	69	55	0	19	50	97.1	92.4	99.7	49.2
IgG-ELISA (cut-off = 0.4)	86	35	–	86	59	0	36	50	100	89	100	40.6
Poly-ELISA (cut-off = 0.4)	124	35	86	–	73	5	65	54	100	80.8	100	28
PaGIA	90	33	59	73	–	26	24	40	94.2	87.8	99.5	36.6

Journal of Thrombosis and Haemostasis, 7: 1260–1265

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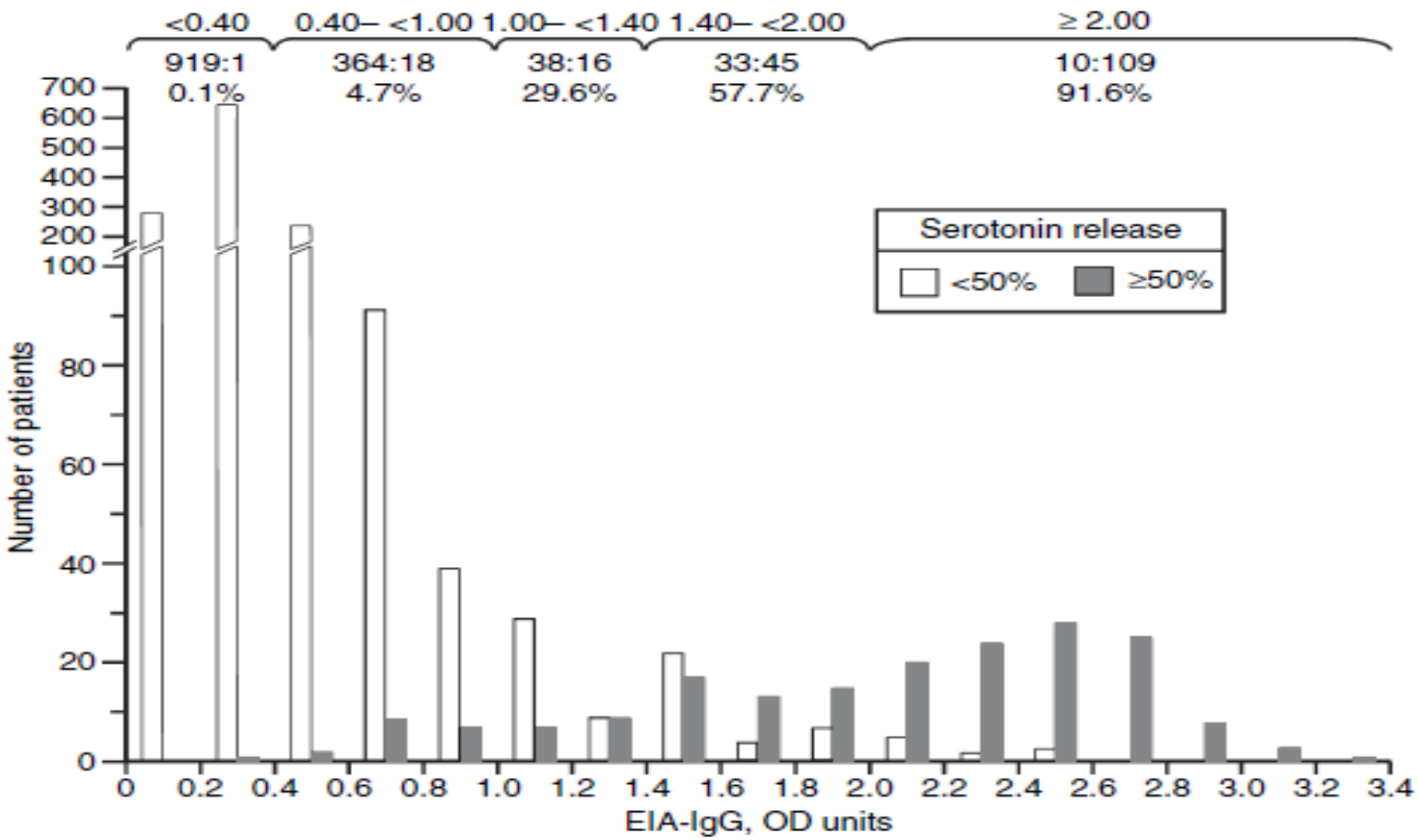


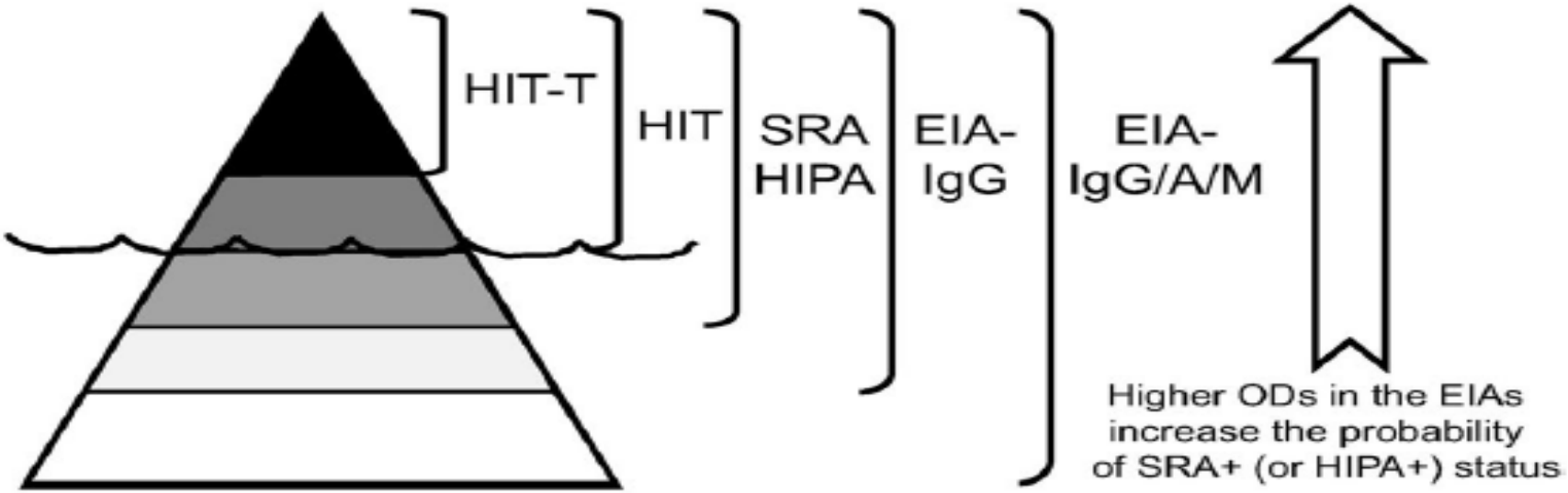
Journal of Thrombosis and Haemostasis, **6**: 1304–1312

Nouveaux tests disponibles

- Tests semi-quantitatifs
  - *LIA test* (turbidimétrique avec microbilles de latex)
    - Semble plus spécifique que le test ELISA
    - Pas encore répandu
    - Rapide, puits individuels
    - Étude de validation plus récente
      - Int J Lab Hematol 2019;41(Supll.1):15-25
    - Appels d'offre et achat de réactifs sous contrat

Tests fonctionnels : relâche de sérotonine et agrégation plaquettaire

- Aide à distinguer les cas pathologiques des cas non pathologiques
- Plus spécifique que les anti-PF4
- Malheureusement
  - Couteux
  - Peu accessible
  - Un mal nécessaire la plupart du temps



EIA-IgG/A/M result (OD units):	≤0.4	0.4-1.0	1.0-1.5	1.5-2.0	≥2.0
Probability of SRA+ status:	~0%	~5%	~25%	~50%	~90%

Int J Lab Hematol. 2019;41(Suppl.1):15–25

Algorithme : pourquoi faire le 4T et les autres tests

Table 4. Test accuracy per 1000 patients after each strategy for diagnose (11% prevalence of HIT)

	Gestalt	4Ts	Immunoassay (based on IgG ELISA low threshold)	95% CI	Functional assay
Sensitivity	0.969	0.921	0.98	0.95-0.99	1.0
Specificity	0.004	0.542	0.85	0.78-0.91	1.0

Strategy	1. Gestalt	2. Gestalt, then IA positive, then FA	3. 4Ts score (high or intermediate)	4. 4Ts score (high or intermediate), then IA	5. 4Ts score (high or intermediate), then IA positive, then FA (recommended strategy)
True positive. Patients will be appropriately treated and/or have more testing (reduced risk of thrombosis by 55%-70%).	107	105	101*	99 (high, 48; intermediate, 51)	100
False negative. Patients will be missed and may experience serious consequences of HIT (eg, thrombosis [300 of 1000 more people with HIT if not treated], amputation [60 of 1000 more people with HIT if not treated], death [60 of 1000 more people with HIT if not treated]).	3	5	9	11 (high, 1; intermediate, 1)	10
True negative. Patients will appropriately not have more testing and will appropriately not be treated for HIT.	4	890	482	829 (high, 34; intermediate, 313)	890
False positive. Patients will continue with unnecessary testing and/or may experience serious consequences of unnecessary treatment of HIT (eg, bleeding ~8% to 35% over treatment duration) and may be falsely labeled as having HIT over the long term.	886†	0	408*†	61 (high, 6; intermediate, 55)	0
No. of immunoassay tests performed					509
No. of functional assay tests performed					160

IA, immunoassay; IgG, immunoglobulin G; FA, functional assay.
*Eighty-nine of these patients would have a high 4Ts score, and 420 would have an intermediate 4Ts score.
†When gestalt or 4Ts score is followed by IA and FA (when IA is positive), these patients would receive non-heparin anticoagulants unnecessarily for varying periods of time depending on timing of the follow-up tests.

blood advances 27 NOVEMBER 2018 · VOLUME 2, NUMBER 22

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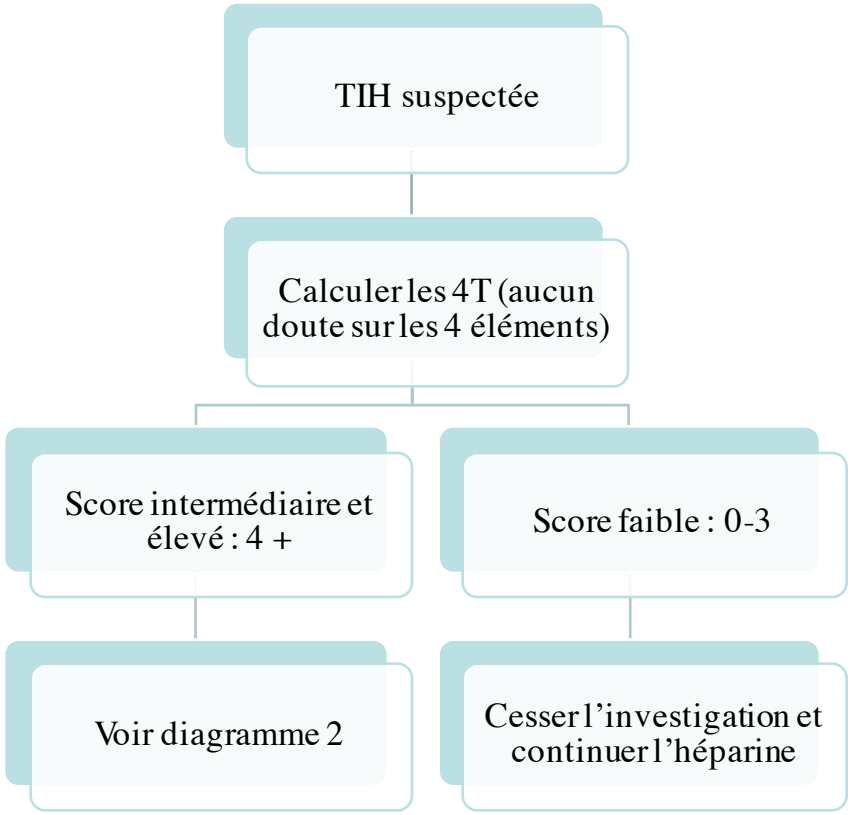
Algorithme décisionnel



Algorithme décisionnel

Score intermédiaire et élevé

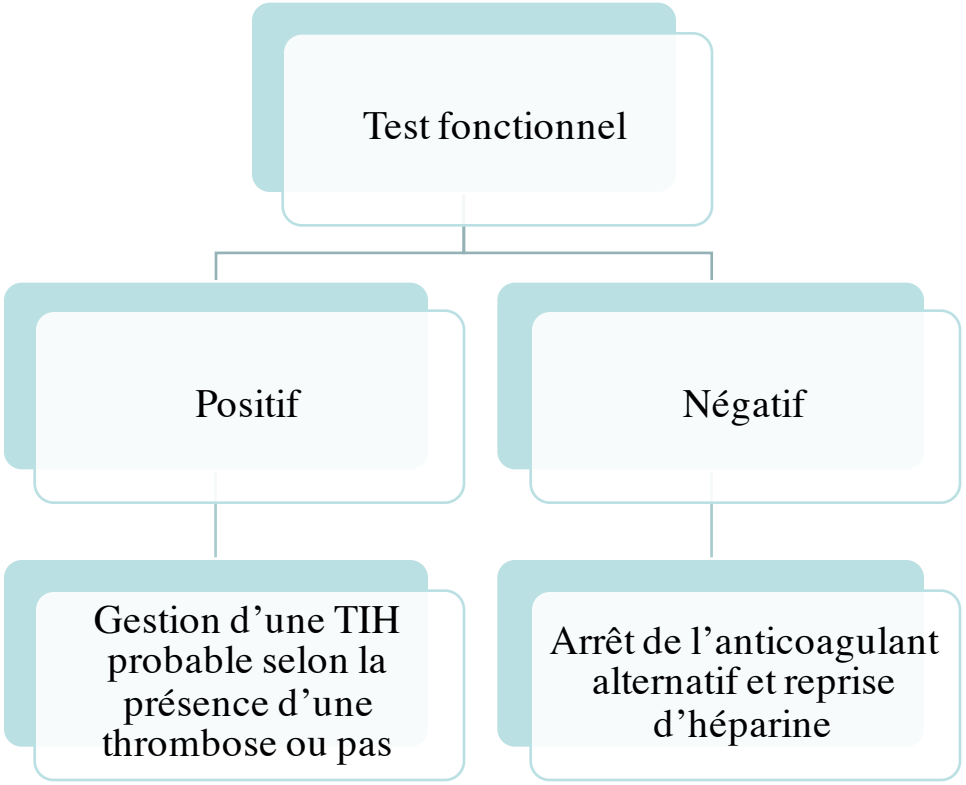
- Obtenir un immunoessai anti-PF4
- Cesser l'héparine et commencer un anticoagulant alternatif (idéalement un inhibiteur Xa)
  - Pour les autres situations, voir diagramme 3
  - Si score intermédiaire et risque de saignement élevé : prophylaxie acceptable si pas de thrombose

Algorithme décisionnel

Anti-PF4 (densité optique (DO))

Si DO < 0,4	Si DO 0,4-2,0	Si DO > 2,0
TIH peu probable : arrêt investigation et reprise héparine/arrêt anticoagulant alternatif	Test fonctionnel requis	Très forte probabilité de TIH (>90 %), test fonctionnel discutable

Algorithme décisionnel



Nouveautés en TIH : en clinique

- Nouvelle entité : TIH auto-immune
  - Excellent article de Greinacher dans le JTH 2017

REVIEW ARTICLE

Autoimmune heparin-induced thrombocytopenia

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Journal of Thrombosis and Haemostasis, **15**: 2099–2114 DOI: 10.1111/jth.13813

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TIH auto-immune

Table 1 Autoimmune heparin-induced thrombocytopenia (aHIT) syndromes

Clinical entity	Description
Delayed-onset HIT	HIT that begins or worsens after stopping of heparin
Persisting HIT	HIT that persists for > 1 week despite stopping of heparin
Spontaneous HIT syndrome	HIT without proximate heparin exposure
Flush heparin HIT	HIT induced by exposure to heparin flushes
Fondaparinux-associated HIT	HIT that is believed to be triggered by exposure to fondaparinux
Severe HIT (e.g. platelet count of $< 20 \times 10^9$ L^{-1}) with overt DIC	Overt HIT-associated DIC defined as proven HIT with one or more of the following: relative/absolute hypofibrinogenemia, elevated INR (without another explanation), and normoblastemia (circulating nucleated red blood cells)

DIC, disseminated intravascular coagulation; INR, International Normalized Ratio.

Journal of Thrombosis and Haemostasis, **15**: 2099–2114 DOI: 10.1111/jth.13813

TIH auto-immune

- Présence d'un anticorps anti-PF4 indépendant de l'héparine
  - Peut activer les plaquettes sans héparine
- Induction d'anticorps anti-PF4
  - ADN et ARN circulants
  - Paroi bactérienne
  - Polyanions autres que l'héparine

TIH auto-immune : particularité

- TIH auto-immune retardée :
  - 25 % de coagulation intravasculaire dissiminée
  - Fréquence élevée de thrombose (100 % vs 50 % en général) selon certaines références

Table 2 Laboratory features of heparin-induced thrombocytopenia (HIT) antibodies

Clinical presentation of HIT	PF4-dependent EIA	Functional (platelet activation) test using washed platelets (e.g. serotonin release assay or heparin-induced platelet activation test)				
		Buffer (0 IU mL^{-1} heparin)	UFH 0.1–0.3 IU mL^{-1}	Fondaparinux/ danaparoid	UFH 100 IU mL^{-1}	Heparin 0.1–0.3 IU mL^{-1} + IV.3
No HIT	Negative or (usually weakly) positive	Negative	Negative	Negative	Negative	Negative
Non-HIT platelet activation	Negative or (usually weakly) positive	Positive	Positive	Positive	Positive	Positive/negative
Typical (or rapid-onset) heparin-dependent HIT	Positive (usually strongly positive)	Negative	Positive	Negative	Negative	Negative
aHIT	Positive (usually very strong reactivity, e.g. OD > 2.0 units)	Positive*	Positive	Positive	Negative	Negative
	Serial diluted sera (amount of dilution required differs among sera)					
aHIT antibodies not cross-reacting with either fondaparinux or danaparoid	Positive (usually very strong reactivity, e.g. OD > 2.0 units)	Negative	Positive	Negative	Negative	Negative
	Diluted serum (required dilution grade differs between sera)					
aHIT antibodies cross-reacting with either fondaparinux or danaparoid	Positive (usually very strong reactivity, e.g. OD > 2.0 units)	Negative	Positive	Positive	Negative	Negative

Journal of Thrombosis and Haemostasis, **15**: 2099–2114 DOI: 10.1111/jth.13813

Traitement de la TIH par les AOD

- Peu d'études vu la rareté de la maladie
  - La plus grosse étude : celle de l'équipe de McMaster avec plusieurs séries de cas
  - La molécule la plus étudiée : rivaroxaban

CLINICAL TRIALS AND OBSERVATIONS

Direct oral anticoagulants for treatment of HIT: update of Hamilton experience and literature review

Theodore E. Warkentin,[1-3] Menaka Pai,[1-4] and Lori-Ann Linkins[2,4]

[1]Department of Pathology and Molecular Medicine and [2]Department of Medicine, Michael G. DeGroote School of Medicine, McMaster University, Hamilton, ON, Canada; [3]McMaster Centre for Transfusion Research, Hamilton, ON, Canada; and [4]Thrombosis and Atherosclerosis Research Institute, Hamilton, ON, Canada

Table 2. Literature review of rivaroxaban for probable HIT (including new patients reported in this article): primary or secondary treatment during acute HIT (groups A_1, A_2, and B)

Study author	Reference	No. of patients	Group			Median platelet count at rivaroxaban start	HIT-associated thrombosis*		Outcome			
			A_1	A_2	B		Thrombosis		Thrombosis		Bleed	
							No.	%	No.	%	No.	%
Rivaroxaban-Hamilton experience												
Linkins et al	17	12	3	2	7	56	6		1		0†	
This study		10	7	1	2	64	5		0		0	
Rivaroxaban-other (non-Hamilton) centers												
Kopolovic and Warkentin	28	1	0	0	1	30	0		0		0	
Ng et al, Ong et al‡	29, 36	9	9	0	0	64	9		0		0	
Sharifi et al§	30	9‡	0	0	9	90‡	4		0		0	
Hantson et al	31	1	0	0	1	30	1		0		0	
Abouchakra et al	32	1	1	0	0	25	1		0		0	
Sartori et al	33	1	0	1	0	150	1		0		0	
Casan et al	34	1	0	0	1	48	1		0		0	
Samoš et al	35	1	1	0	0	65	1		0		0	
Summary		46	21	4	21	73	29/46‖	63.0	1/46	2.2	0/46	0

Table 3. Literature review of apixaban or dabigatran for probable acute HIT (including new patients reported in this article): primary or secondary treatment (groups A_1, A_2, and B)

Study author	Reference	No. of patients	Group			Median platelet count at DOAC start	HIT-associated thrombosis*		Outcome			
			A_1	A_2	B				Thrombosis		Bleed	
							No.	%	No.	%	No.	%
Apixaban												
Sharifi et al†	30	5	0	0	5	90‡	1		0		0	
Larsen et al	37	1	1	0	0	112	0		0		0	
Delgado-García et al§	38, 39	1	1	0	0	25	1		0		0	
Kunk et al	40	5	0	0	5	111	3		0		0	
Total		12	2	0	10	90‡	5/12‖	41.7	0/12	0	0/12	0
Dabigatran												
Sharifi et al†	30	6	0	0	6	90‡	2		0		0	
Anniccherico et al	41, 42	1	0	0	1	120	1		0		0	
Mirdamadi§	43	1	1	0	0	32	1		0		0	
Tardy-Poncet et al	44	1	0	0	1	56	0		0		0	
Noel et al	45	1	0	1	0	216	1		1¶		0	
Bircan and Alanoglu§	46	1	1	0	0	52	1		0		0	
Total		11	2	1	8	58	6/11‖	54.5	1/11	9.1	0/11	0

Les patients de Hamilton

Patient clinical setting
CA-DVT
CA-PE
Multiple myeloma flush‡
Multiple myeloma flush‡
Hip fracture thromboprophylaxis§
CA-DVT
General surgery thromboprophylaxis
Hip fracture thromboprophylaxis§
CA-DVT/PE
Post–coronary artery bypass graftingǁ

Patient clinical setting
Post–abdominal aortic aneurysm thromboprophylaxis
General surgery thromboprophylaxis
Medical prophylaxis
Hip fracture thromboprophylaxis§
Post–coronary artery bypass graftingǁ
Post–aortic valve replacementǁ

BLOOD, 31 AUGUST 2017 • VOLUME 130, NUMBER 9

- En général, pas de thrombose critique
- 5/16 patients avec thrombose associée à la TIH (TIH-T)
- Quelques cas de TIH auto-immunes *(“heparin flush”)*

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2e revue de littérature

PHARMACOTHERAPY

REVIEW OF THERAPEUTICS

Potential Role of Direct Oral Anticoagulants in the Management of Heparin-induced Thrombocytopenia

Ashley Barlow[1,*] Brooke Barlow,[1] Travis Reinaker,[2] and Justin Harris[3]

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PHARMACOTHERAPY Volume 39, Number 8, 2019

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TIH et AOD

Table 6. Reported DOAC Dosing Regimens for HIT (n=98)[a]

DOAC	Dosing Regimen	Total Recipientsn (%)	Pretreatment with Parenteral Anticoagulation n(%)
Rivaroxaban (n=59)	15 mg BID × 21 days[b] then 20 mg daily	34 (57)	10 (29)
	20 mg daily	16 (27)	13 (87)
	10 mg daily	9 (16)	3 (33)
Apixaban (n=27)	10 mg BID × 7 days then 5 mg BID	11 (41)	0
	5 mg BID	14 (52)	8 (57)
	2.5 mg BID	2 (7)	1 (50)
Dabigatran (n=12)	150 mg BID	9 (75)	7 (78)
	110 mg BID	3 (25)	1 (33)

BID = twice daily; DOAC = direct acting oral anticoagulant; n = number of patients.

[a]Six studies excluded.[24–26, 35, 37, 39]

[b]Some variability in median duration of rivaroxaban 15 mg BID.

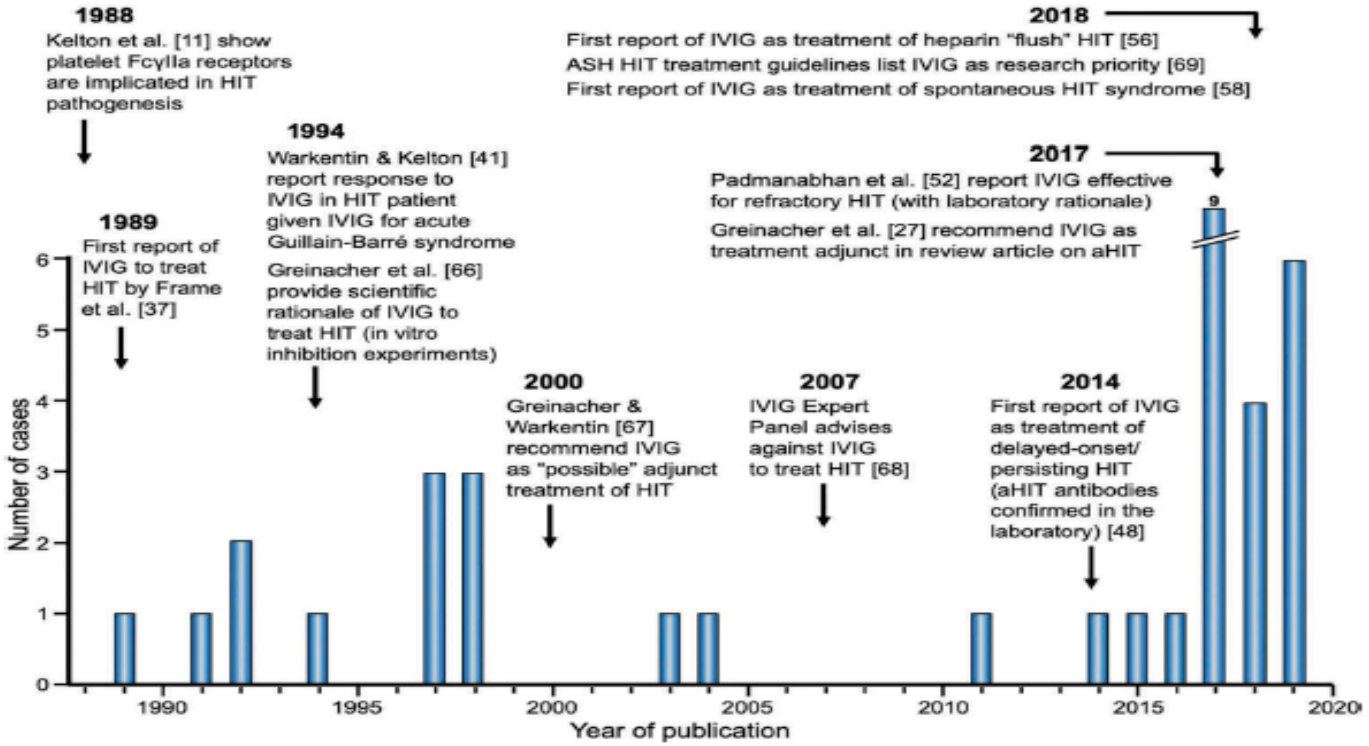
TIH et AOD : à retenir

- D'un point de vue clinique
  - Peu de récidives de thrombose
  - La majorité avait un décompte plaquettaire plus grand que 50 x 9/L
  - Le rivaroxaban a le plus de données
  - AOD utilisé chez les patients médicalement stables
  - La dose de rivaroxaban la plus étudiée
    - 15 mg BID x 3 semaines suivie de 20 mg die

Immunoglobuline IV (IgIV) et TIH

- OUI ! Ça existe !
- Malgré le risque de thrombose associée au IgIV
- Longue histoire...

IgIV et TIH : historique



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IgIV et TIH

- Quelques revues

High-dose intravenous immunoglobulin for the treatment and prevention of heparin-induced thrombocytopenia: a review

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Intravenous Immune Globulin to Prevent Heparin-Induced Thrombocytopenia

N ENGL J MED 378;19 NEJM.ORG MAY 10, 2018

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IgIV et TIH

- Pour des cas particuliers
  - La TIH auto-immune
    - Pont à une anticoagulation alternative ou adjointe
  - La réexposition à l'héparine malgré un test fonctionnel positif
    - Possibilité de réexposer de courte durée à l'héparine pour les interventions agressives : chirurgie cardiaque

IgIV et TIH :
activité est dose dépendante

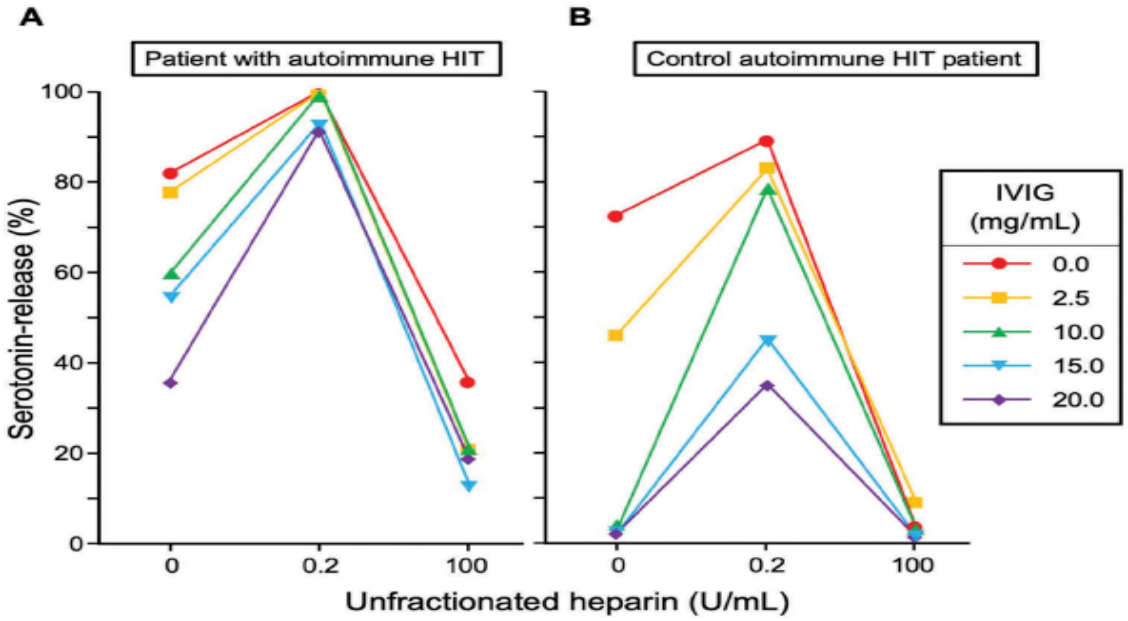

Figure 3. Dose-dependent inhibition by IVIG of aHIT serum-induced platelet activation (percent serotonin-release). (a) Results using serum obtained from a patient who subsequently received IVIG to treat aHIT [61]. (b) Results using serum from a control patient with aHIT. Both experiments show that it is eas activation at 0 U/mL heparin (buffer control) than in the presence of heparin (0.2 U/mL UFH). Reprinted, with permission, from [61].

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IgIV et TIH: cas rapportés

Table 1. Reported cases of HIT treated with IVIG from 2014 to 2019.

Report	Age/Sex	aHIT syndrome	Nadir platelet count, $\times 10^9$/L	HIT thrombotic and hemorrhagic sequelae	Concomitant anti-coagulation	IVIg dose, including need for repeat (rpt) dose	Response to IVIG, including response to repeat (rpt) dose
[48]Warkentin 2014	68M	Delayed/persisting, lab	20	DVT	Fonda	1g/kg×1; 1g/kg (rpt)	Good/transient; good/transient (rpt); no thrombosis
[49]Tvito 2015	85F	Persisting, fonda-XR, lab	3	DVT	Biv	1g/kg×2	Excellent; no thrombosis
[50]Lei 2017	47M	Delayed/persisting	8	Limb artery, DVT, PE, atrial thrombus	Biv	1g/kg×2	Excellent; no thrombosis
[51]Doucette 2017	49F	Delayed/persisting	5	Limb artery	Arg	1g/kg×2; 1g/kg (rpt)	Good/transient; good (rpt); thrombosis*
[51]Doucette 2017	84F	Delayed/persisting	16	DVTs (including UL-DVT)	Arg	1g/kg×5	Poor; no thrombosis
[52]Padmanaban 2017	47M	Delayed/persisting; lab**	10	DVT, limb artery, right atrial, PE	Biv	1g/kg×2	Excellent; no thrombosis
[52]Padmanaban 2017	73M	Delayed/persisting; lab**	15	PE	Biv	1g/kg×2	Excellent; no thrombosis
[52]Padmanaban 2017	72M	Delayed/persisting; lab**	5	Bilateral DVT	Arg	1g/kg×2; 0.5g/kg (rpt)	Good/transient; good/transient (rpt); no thrombosis
[53]Azimov 2017	58F	Delayed/persisting	26	Nil	Fonda	1g/kg×1, 0.45g/kg×1	Excellent; no thrombosis
[54]Ibrahim 2017	78F	Persisting	15	UL-DVT	Arg	0.4g/kg×4	Good; no thrombosis
[55]Gleichgerrcht 2017	52F	Persisting	*25*	CVST	Arg	2g/kg over 4d***	Good; no thrombosis
[56]McKenzie 2018	48M	"Flush" HIT; persisting	14	STEMI	Arg	0.5g/kg×5	Good; no thrombosis
[57]Park 2018	82M	Persisting	19	UL-DVT	Fonda	1g/kg×1	Poor; no thrombosis
[57]Park 2018	38M	Delayed/persisting	18	DVT	Fonda	1g/kg×2	Excellent; no thrombosis
[58]Irani 2018	30M	Spontaneous HIT; lab**	41	CVA (arterial), limb artery	Fonda	1g/kg×2	Excellent; no thrombosis
[59]Mohanty 2019	52F	Spontaneous HIT; lab	21	Mesenteric vein thrombosis	Fonda	1g/kg×2	Excellent; no thrombosis
[60]Ramachandran 2019	55M	Persisting	16	DVT	Arg	1g/kg×2****	Poor; no thrombosis
[61]Arcinas 2019	78M	Delayed/persisting; lab	9	DVT (venous limb gangrene)	Biv	0.4g/kg×5	Excellent*****; no thrombosis
[62]Gonzales2019	46F	Persisting	<10	CVST	Arg; biv	0.7g/kg×2 (arg); 0.4g/kg×7 (biv) (rpt)	Poor; excellent (rpt); no thrombosis
[63]Huang 2019	80F	Persisting	1	DVT; severe GI bleeding	Nil (riv later)	0.4g/kg×5	Excellent; no thrombosis
New case (Figure 4)	67F	Laboratory******	61	CVA (arterial), limb artery, PE, adrenal veins	Danap	1g/kg×2	Excellent; no thrombosis

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IgIV et TIH : à retenir

Seulement la dose de 1g/kg de IgIV die x 2 jours est efficace

La dose de 0,4 g/kg x 5 jours = nettement moins efficace.

N.B. 1 g IgIV = 10 mL

Messages clé

- Diagnostic et prise en charge sur plusieurs éléments
  - Clinique avec le score 4T
  - Immunoessais anti-PF4 : décision rapide
  - Test fonctionnel : décision finale
- Nouvelles entités cliniques : TIH auto-immune
- Utilisation grandissante des AOD pour la TIH médicalement stable
- Utilisation des IgIV pour les rares cas de TIH auto-immunes et de persistance de TIH aiguë/subaiguë

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Questions ?

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