KOAGULOPATIE V NEUROINTENZIVNÍ PÉČI

20.9.2024

MUDr. Kamil Vrbica

Klinika anesteziologie, resuscitace a intenzivní medicíny

Koagulopatie a neurointenzivní péče

- V neurointenzivní péči se můžeme setkat s mnoha chorobami, které mohou být způsobeny, ovlivněny nebo samy způsobují zvýšenou krvácivost, trombembolii a jejich patofyziologický podklad je značně odlišný

- zhoršení neurologického stavu

- Krvácivé choroby a komplikace
  - TBI, hCMP, rebleeding u SAK, perioperační krvácení, epidurální hematom, atd.

- Trombembolické choroby a komplikace
  - iCMP, infarkt myokardu, hluboká žilní trombóza, plicní embolie, atd.

- **Jednou ze základních výzev pro neurointenzivistu je vyřešit krvácivý či trombotický stav a poté preventovat trombembolické komplikace a zároveň minimalizovat krvácivé komplikace**

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Definice koagulopatie

- Koagulopatie může bý
  fibrinolytickými proces
  nebo k abnormalitám

ze

TABLE 1. Prevalences of Coagulopathy in iTBI Including Outcome in the Presence of Coagulopathy

Study	No. of patients	Definition of TBI	Definition of coagulopathy	Prevalence of coagulopathy (%)	Mortality with coagulopathy (%)
Harhangi[34]*	5357	Heterogeneous	Heterogeneous	32.7 (10-97.5)	51 (25-93)
Epstein[82]**	7037	Heterogeneous	Heterogeneous	35.2 (7-86.1)	17-86
Zehtabchi[83]	224	$AIS_{head} > 2$ *and/or* any intracranial hematoma on CT	INR > 1.3 *or* PTT > 34 s	17 (8-30)	–
Talving[84]	387	$AIS_{head} \geq 3$ *and* extracranial AIS < 3	Platelets < 100 000 mm^3 *or* INR > 1.1 *or* aPTT > 36 s	34	34.7
Lustenberger[22]	278	$AIS_{head} \geq 3$ *and* extracranial AIS < 3	Platelets < 100 000 mm^3 *and/or* INR > 1.4 *and/or* aPTT > 36 s	45.7	40.9
Lustenberger[85]	132	$AIS_{head} \geq 3$ *and* extracranial AIS < 3	Platelets < 100 000 mm^3 *or* INR > 1.2 *or* aPTT > 36 s	36.4	32.5
Wafaisade[86]	3114	$AIS_{head} \geq 3$ *and* extracranial AIS < 3	Quick (PT_R) < 70% *and/or* platelets < 100 000/ml	22.7	50.4
Chhabra[87]	100	GCS < 13	Fibrinogen < 200 mg/dL	7	–
Greuters[20]	107	Brain tissue injury on CT *and* extracranial AIS < 3	aPTT > 40s *and/or* INR > 1.2 *and/or* platelets < 120 × 10^9/l	24 (54#)	41
Shehata[88]	101	iTBI on admission brain CT	INR ≥ 1.2, PT > 13s, d-dimer positive, platelets < 100 × 10^3/CC	63	36
Schöchl[58]	88	$AIS_{head} \geq 3$ *and* extracranial AIS < 3	Quick (PT_R) < 70% *and/or* aPTT > 35 s *and/or* fibrinogen < 150 mg/dL *and/or* platelets < 100 × 10^9/l	15,8	50
Franschman[89]	226	iTBI on CT *and* extracranial AIS < 3	aPTT > 40 s *and/or* PT > 1.2 *and/or* platelets < 120 × 10^9/l	25 (44#)	33
Genet[90]	23	$AIS_{head} \geq 3$ *and* extracranial AIS < 3	aPTT > 35 s *and/or* INR > 1.2	13	22
Alexiou[91]	149	iTBI *with* exclusion of multisystem trauma	aPTT > 40s *and/or* INR > 1.2 *and/or* platelets < 120 × 10^9/l	14.8 (22.8#)	–
Joseph[7]	591	$AIS_{head} \geq 3$ *and* extracranial AIS < 3	INR ≥ 1.5 *and/or* PTT ≥ 35s *and/or* platelets ≤ 100 × 10^3/ml	13.3	23
Epstein[92]	1718	$AIS_{head} \geq 3$ *and* extracranial AIS < 3	INR ≥ 1.3	7.7	45.1
De Oliveira Manoel[93]	48	$AIS_{head} \geq 3$ *and* extracranial AIS < 3	INR ≥ 1.5 *and/or* aPTT ≥ 60s *and/or* platelets < 100 × $10^3/mm^3$§	12.5	66
Dekker[50]	52	$AIS_{head} \geq 3$	INR > 1.2 *and/or* aPTT > 40s *and/or* platelets < 120 × 10^9/l	42	45.5
Yuan[94]	2319	Intracranial injury on CT *and* extracranial AIS < 3	INR > 1.25 *and/or* PT >14 s *and/or* aPTT >36 s and/or platelets < 100 × 10^9/l	18.6	17.6
Albert[47]	561	iTBI on admission brain CT	INR ≥ 1.27 *and/or* PT ≥ 16.7 s *and/or* aPTT > 28.8 s	41.6%	61.1%
Böhm[8]	598	iTBI on CT *and* no extracranial injuries	INR > 1.2 *and/or* aPTT > 35 s *and/or* fibrinogen < 150 mg/dL and/or platelets < 100 × 10^3/nL	19.6	-

Review > Neurosurgery. 2021 Nov 18;89(6):954-966. doi: 10.1093/neuros/nyab358.

Coagulopathy and Progression of Intracranial Hemorrhage in Traumatic Brain Injury: Mechanisms, Impact, and Therapeutic Considerations

Marc Maegele [1 2 3]

Klinika anesteziologie,
resuscitace a intenzivní medicíny
Fakultní nemocnice Brno
Lékařská fakulta Masarykovy univerzity

Stanovení koagulopatie

RESEARCH Open Access

The European guideline on management of major bleeding and coagulopathy following trauma: fourth edition

Rolf Rossaint[1], Bertil Bouillon[2], Vladimir Cerny[3,4,5,6], Timothy J. Coats[7], Jacques Duranteau[8],

portance of fibrinogen and platelet measurements. It is often assumed that the conventional coagulation screens [international normalised ratio (INR) and APTT] monitor coagulation, however these tests monitor only the initiation phase of blood coagulation, and represent only the first 4 % of thrombin production [178]. It is there-

tion is abnormal [13, 179–183]. In addition, the delay in detection of traumatic coagulopathy can influence outcome, and the turnaround time of thromboelastometry has been shown to be significantly shorter than conventional laboratory testing, with a time saving of 30–60 min [181, 184, 185]. Viscoelastic testing may

Proč vůbec INR a aPTT ????

Stanovení koagulopatie

J Neurotrauma. 2011 Oct;28(10):2033-41. doi: 10.1089/neu.2010.1744. Epub 2011 Sep 23.

Thromboelastometric (ROTEM) findings in patients suffering from isolated severe traumatic brain injury

Herbert Schöchl [1], Cristina Solomon, Stefan Traintinger, Ulrike Nienaber, Astrid Tacacs-Tolnai, Christian Windhofer, Soheyl Bahrami, Wolfgang Voelckel

results. Thirty-two patients were included in the study. Complete adherence to the algorithm was observed in 20 out of 32 cases. The availability of thromboelastometric results after hospital admission was reported significantly earlier than conventional coagulation tests (median (IQR [range]) 33 (20-40 [14-250]) min vs. 71 (51-101 [32-290]) min; p = 0.037). Although only 5 out of 32 patients had abnormalities of conventional coagulation tests 21 out of 32 patients had a coagulopathic baseline thromboelastometric trace. Implementing a thromboelastometric-guided algorithm for the

Výhody:

- **přesnější popis reálné hemostázy**
- **rychlejší výsledky**

Multicenter Study > Anaesthesia. 2019 Jul;74(7):883-890. doi: 10.1111/anae.14670. Epub 2019 Apr 29.

Protocolised thromboelastometric-guided haemostatic management in patients with traumatic brain injury: a pilot study

J Gratz [1], H Güting [2], S Thorn [3], A Brazinova [4], K Görlinger [5 6], N Schäfer [2], H Schöchl [7 8],

https://www.clot.pro/

https://www.werfen.com/cz/cs/diagnostika-hemostazy/rotem-delta

https://www.medgadget.com/2019/05/haemonetics-teg-6s-hemostasis-analyzer-cleared-for-trauma-testing.html

Patofyziologie

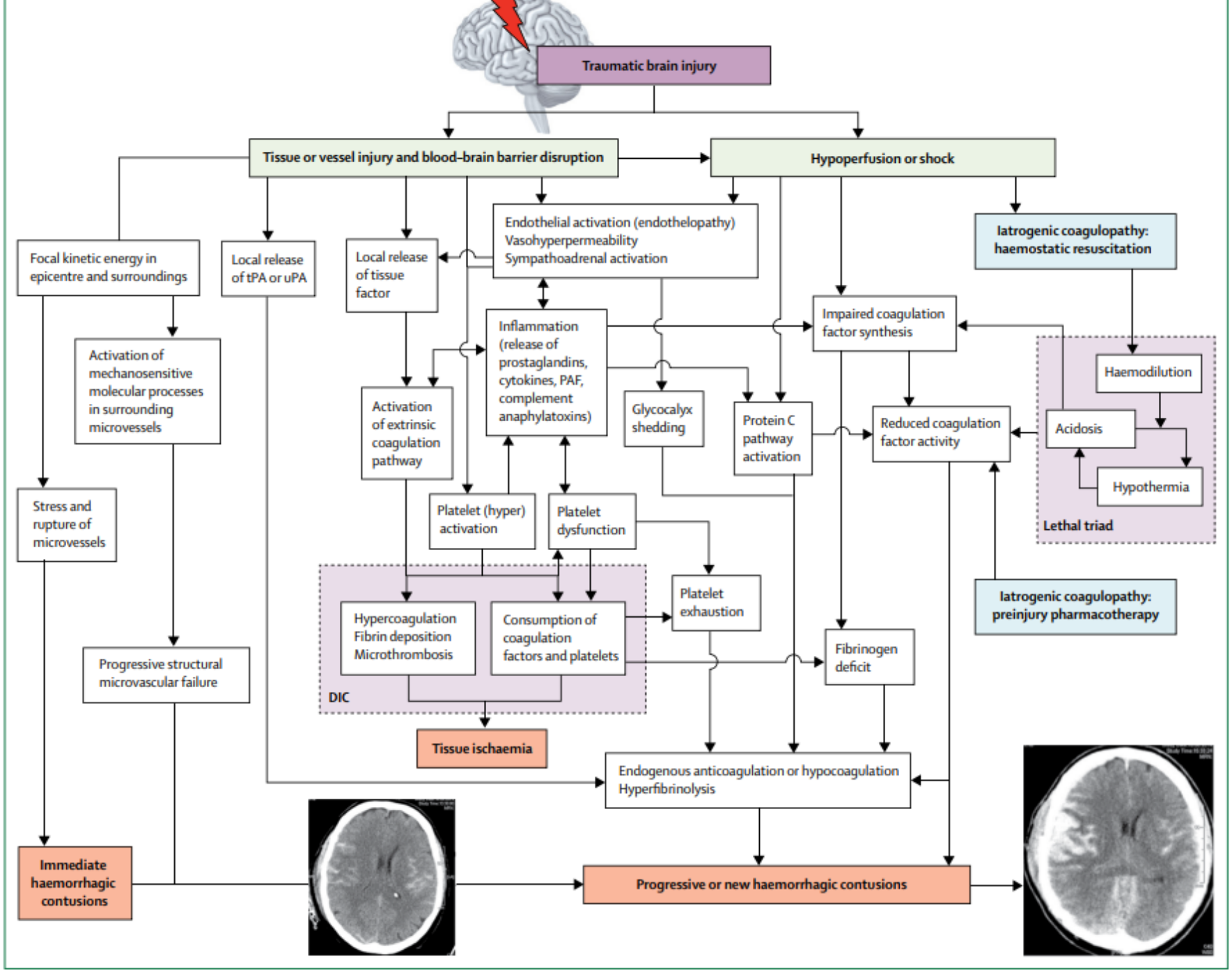


Figure 1: Current understanding of the mechanisms underlying coagulopathy and haemorrhagic contusions after traumatic brain injury

Review > Neurosurgery. 2021 Nov 18;89(6):954-966. doi: 10.1093/neuros/nyab358.

Coagulopathy and Progression of Intracranial Hemorrhage in Traumatic Brain Injury: Mechanisms, Impact, and Therapeutic Considerations

Marc Maegele [1 2 3]

Traumatic brain injury 2

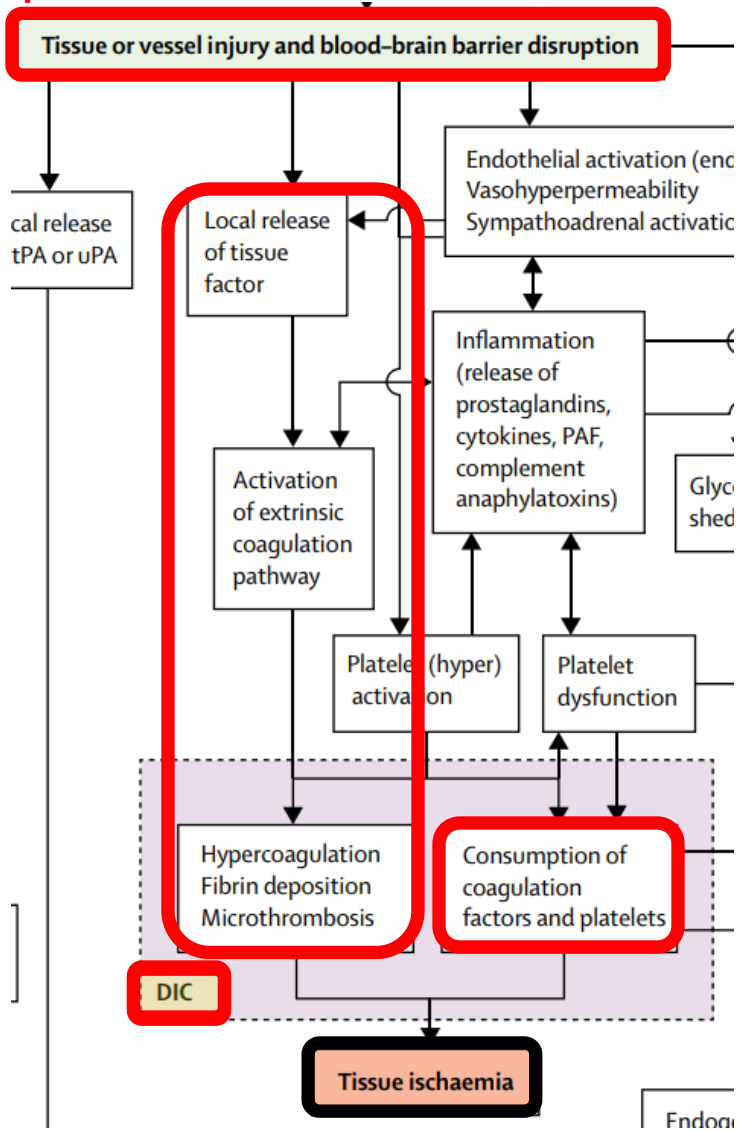
Coagulopathy and haemorrhagic progression in traumatic brain injury: advances in mechanisms, diagnosis, and management

Marc Maegele, Herbert Schöchl, Tomas Menovsky, Hugues Maréchal, Niklas Marklund, Andras Buki, Simon Stanworth

Klinika anesteziologie, resuscitace a intenzivní medicíny
Fakultní nemocnice Brno
Lékařská fakulta Masarykovy univerzity

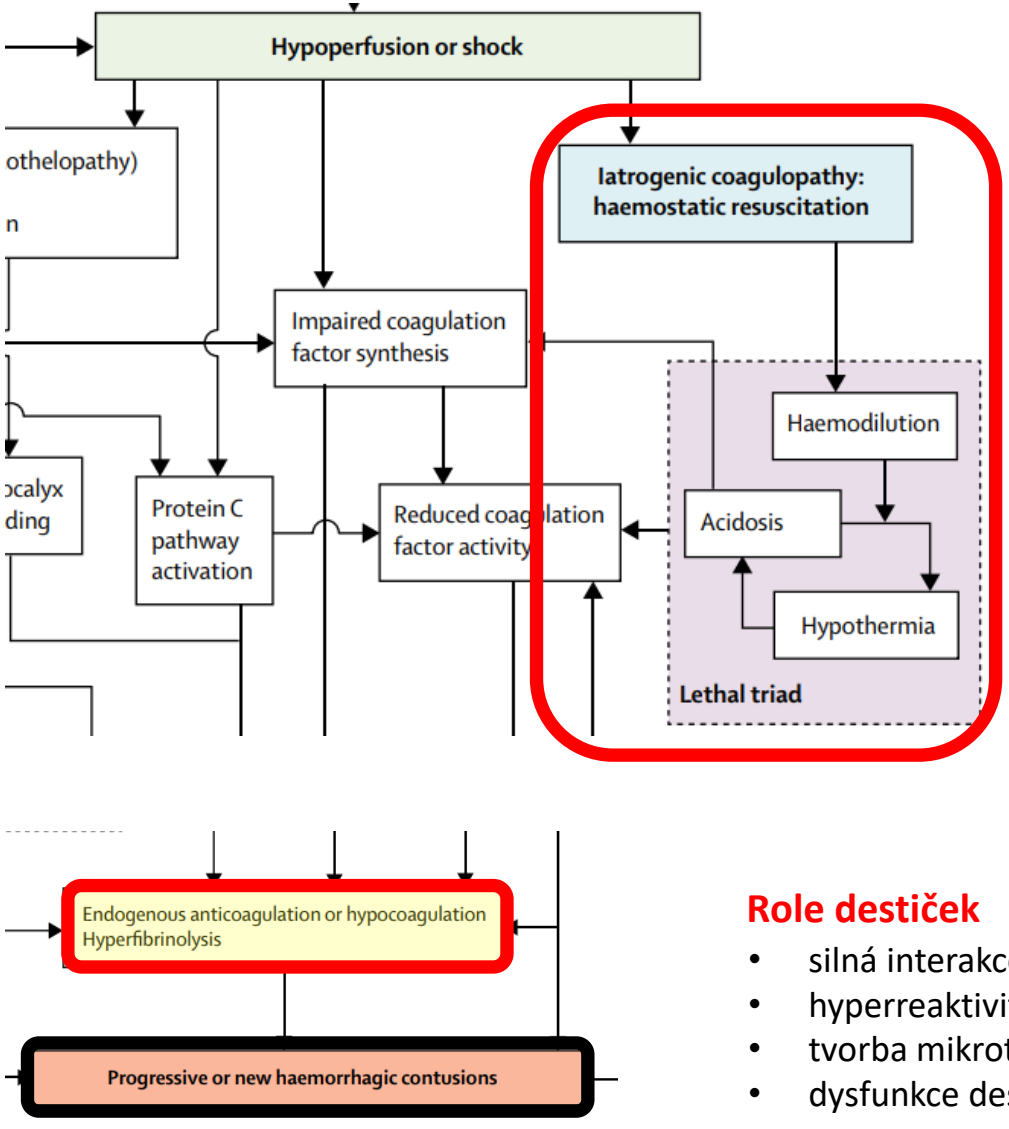
Patofyziologie

Koagulopatie vázaná na TBI



Koagulopatie vázaná na šok

Endotheliopathy, inflammation, and glycocalyx shedding Protein C pathway activation



Shrnutí

- **progrese z hyperkoagulačního do hypokoagulačního stavu**
- **podílí se**
  - aktivace endotelu
  - tkáňový faktor
  - endogenní antikoagulace
  - zánět
  - dysfunkce destiček
  - přeměna fibrinogenu
  - hyperfibrinolýza

Role destiček

- silná interakce poškozeného endotelu a destiček
- hyperreaktivita destiček (PAF)
- tvorba mikrotrombů a konzumpce destiček
- dysfunkce destiček

Traumatic brain injury 2
Coagulopathy and haemorrhagic progression in traumatic brain injury: advances in mechanisms, diagnosis, and management
Marc Maegele, Herbert Schöchl, Tomas Menovsky, Hugues Maréchal, Niklas Marklund, Andras Buki, Simon Stanworth

Kazuistika

Datum a čas odběru:
Vyšetření
Leukocyty
Erytrocyty
Hemoglobin
Hematokrit
Střední objem ERY
Trombocyty
Množství HGB v ERY
Koncentr. HGB v ERY
Střední objem trombo
Šíře distribuce ERY
Normoblasty
Normoblasty/100WBC
Čas telefon.hlášení

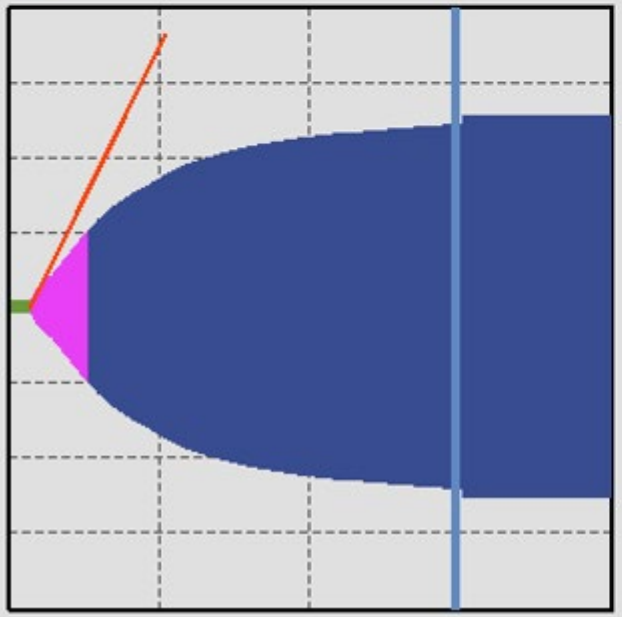
11.07.2024 12:16

FI

11.07.2024 12:47:02 / 60:00

EX-test

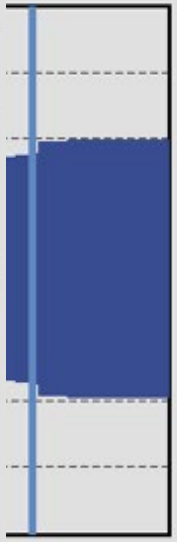
CT	99s	▲ 38-65
A5	26mm	▼ 39-58
A10	38mm	▼ 47-64
A20	46mm	▼ 52-67
MCF	51mm	▼ 53-68
CFT	213s	▲ 42-93



11.07.2024 12:47:32 / 60:00

FIB-test

CT	104s	▲ 55-87
A5	8mm	▶ 6-21
A10	9mm	▶ 7-23
A20	10mm	▶ 8-25
MCF	11mm	▶ 9-27



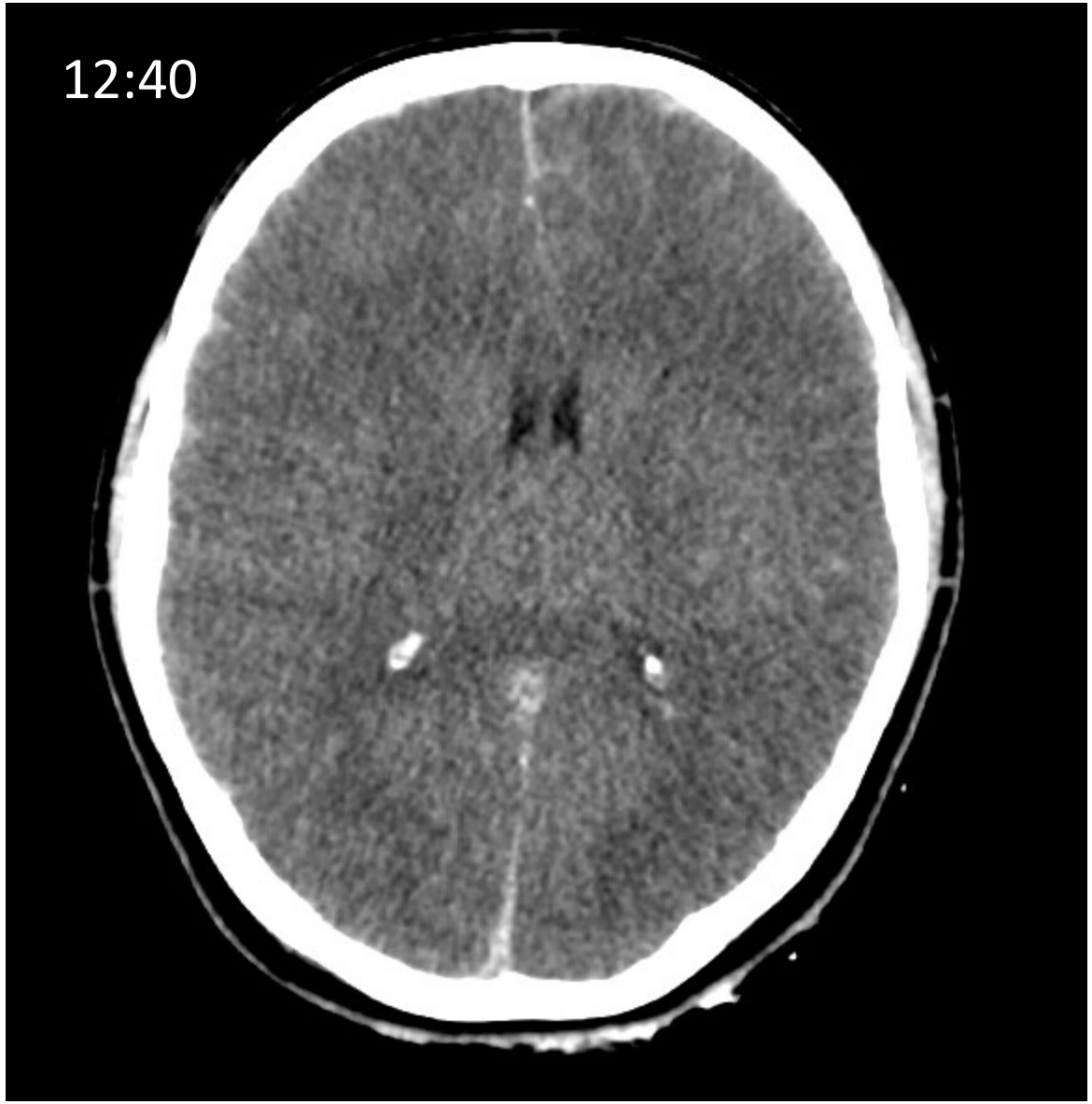
0

▲ 44-76
▼ 36-59
▼ 46-65
▼ 51-67
▼ 51-67
▲ 46-118

Termín: S

ent.

- 1.2)
17)
- 1.2)
- 4.2)
- 1.2)
40)
- 1.1)
19)
120)
0.5) větší než 20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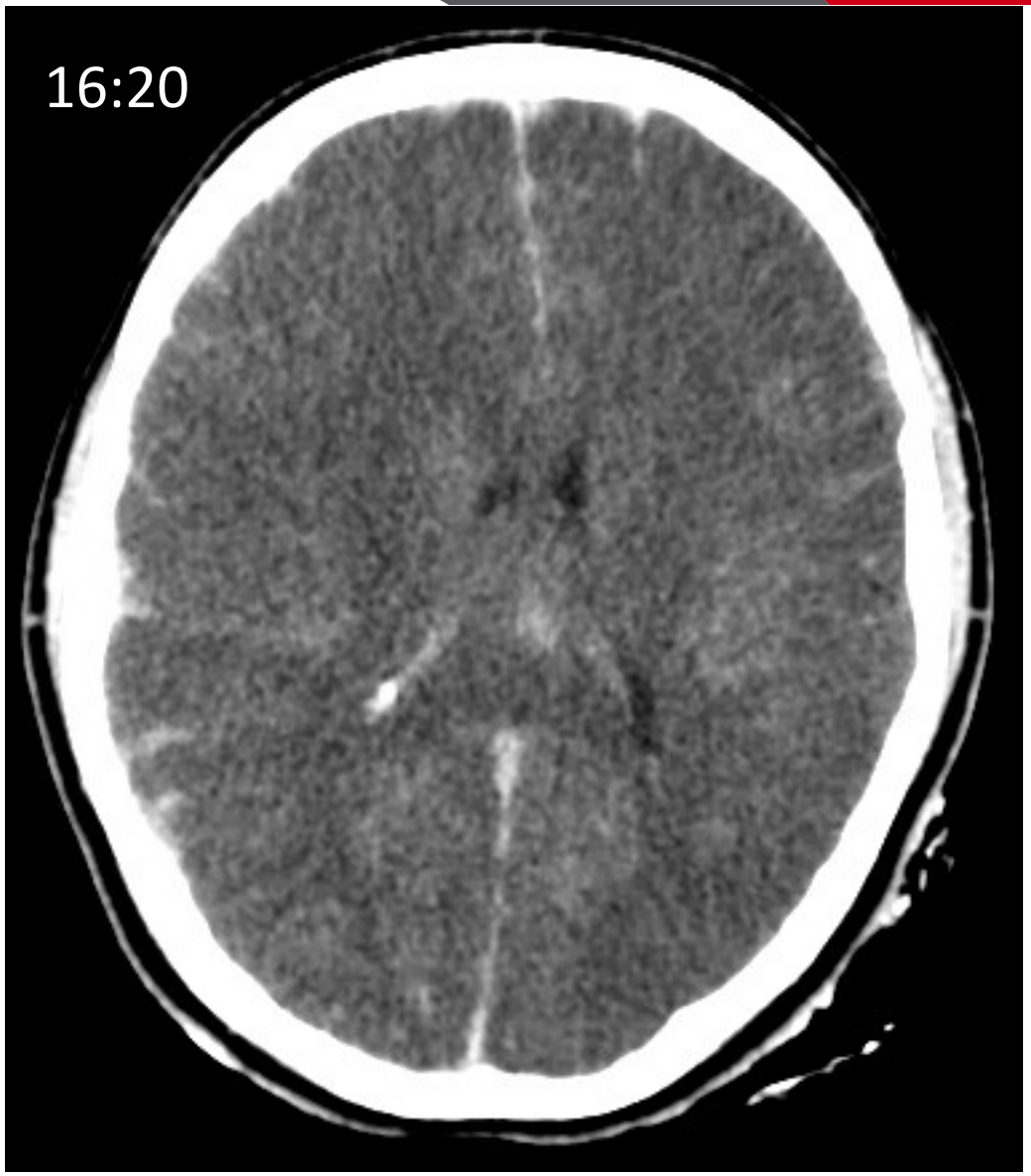
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Stanovení koagulopatie

2.3.5

Doporučujeme rutinně a včasně provádět opakovaná stanovení PT, aPTT, fibrinogenu a trombocytů. (1C)

2.3.6

K identifikaci koagulační poruchy doporučujeme používat viskoelastometrické metody, jsou-li dostupné. (1)§

Diagnostika a léčba život ohrožujícího krvácení u dospělých pacientů v intenzivní a perioperační péči

Česko-slovenský mezioborový doporučený postup

Blatný J., Bláha J., Cvachovec K., Černý V.*, Firment J., Kubisz P., Kvasnička J., Masopust J., Penka M., Salaj P., Staško J., Záhorec R., Zýková I.

We recommend the application of intervention algorithms incorporating pre-defined triggers and targets based on viscoelastic haemostatic assay (VHA) coagulation monitoring to guide individualised haemostatic intervention in the case of perioperative bleeding. **1C**

If VHA is not available we recommend the application of intervention algorithms incorporating pre-defined triggers based on conventional coagulation tests. **1C**

R11
Coagulation monitoring

***

Early, repeated haemostasis monitoring, including laboratory measurements (PT/INR, fibrinogen, platelets, fibrinogen) and/or point-of-care PT/INR and/or viscoelastic methods, should be employed.

Rossaint *et al. Critical Care* (2023) 27:80
https://doi.org/10.1186/s13054-023-04327-7

Critical Care

GUIDELINES Open Access

The European guideline on management of major bleeding and coagulopathy following trauma: sixth edition

EJA *Eur J Anaesthesiol* 2017; **34**:332–395

GUIDELINES

Management of severe perioperative bleeding: guidelines from the European Society of Anaesthesiology

First update 2016

EJA *Eur J Anaesthesiol* 2023; **40**:226–304

GUIDELINES

Management of severe peri-operative bleeding: Guidelines from the European Society of Anaesthesiology and Intensive Care

Second update 2022

Komplikace…

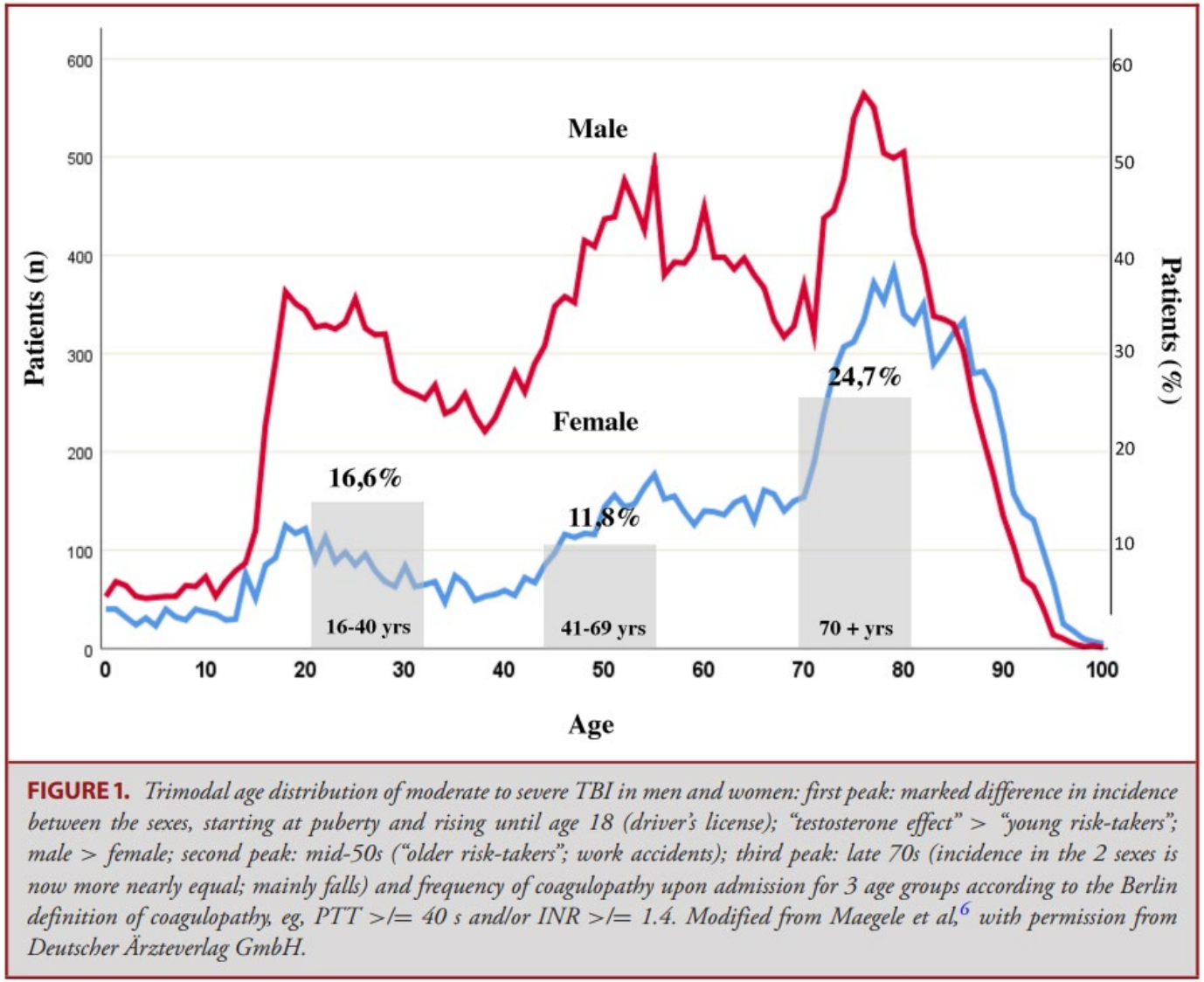

FIGURE 1. *Trimodal age distribution of moderate to severe TBI in men and women: first peak: marked difference in incidence between the sexes, starting at puberty and rising until age 18 (driver's license); "testosterone effect" > "young risk-takers"; male > female; second peak: mid-50s ("older risk-takers"; work accidents); third peak: late 70s (incidence in the 2 sexes is now more nearly equal; mainly falls) and frequency of coagulopathy upon admission for 3 age groups according to the Berlin definition of coagulopathy, eg, PTT >/= 40 s and/or INR >/= 1.4. Modified from Maegele et al,[6] with permission from Deutscher Ärzteverlag GmbH.*

- antikoagulační/antiagregační terapie – 20% pacientů

ORIGINAL WORK

Global Characterisation of Coagulopathy in Isolated Traumatic Brain Injury (iTBI): A CENTER-TBI Analysis

Julia K. Böhm[1], Helge Güting[1], Sophie Thorn[2], Nadine Schäfer[1], Victoria Rambach[1], Herbert Schöchl[4,5], Oliver Grottke[6], Rolf Rossaint[6], Simon Stanworth[7], Nicola Curry[7], Rolf Lefering[1], Marc Maegele[1,3] and CENTER-TBI Participants and Investigators

- zhoršení krvácení a outcome

Acta Neurochir (Wien). 2016 Jan;158(1):117-23. doi: 10.1007/s00701-015-2645-8. Epub 2015 Nov 27.

High prevalence of pharmacologically induced platelet dysfunction in the acute setting of brain injury

Vincent Prinz[1], Tobias Finger[1], Simon Bayerl[1], Christoph Rosenthal[2], Stefan Wolf[1],

- warfarin zdvojnásobuje riziko špatného outcomu

Review. Br J Neurosurg. 2012 Aug;26(4):525-30. doi: 10.3109/02688697.2011.650736. Epub 2012 Feb 10.

A meta-analysis to determine the effect of anticoagulation on mortality in patients with blunt head trauma

John Stephen Batchelor[1], Alan Grayson

- antiagregační léčba zhoršuje riziko tvorby více druhů krvácení u lehkého TBI

World Neurosurg. 2018 Feb;110:e339-e345. doi: 10.1016/j.wneu.2017.10.173. Epub 2017 Nov 10.

Are Antiplatelet and Anticoagulants Drugs A Risk Factor for Bleeding in Mild Traumatic Brain Injury?

Laura Uccella[1], Cesare Zoia[2], Daniele Bongetta[3], Paolo Gaetani[2], Franz Martig[4], Christian Candrian[4], Raffaele Rosso[4]

- málo dat na DOAC, podobné jako warfarin

Journal of Clinical Medicine

Article

Management of Traumatic Brain Injury in Patients with DOAC Therapy–Are the "New" Oral Anticoagulants Really Safer?

Anna Antoni[1,*], Lukas Wedrich[1,2], Martin Schauperl[1,3], Leonard Höchtl-Lee[4], Irene K. Sigmund[1], Markus Gregori[1], Johannes Leitgeb[1], Elisabeth Schwendenwein[1] and Stefan Hajdu[1]

Review. Neurosurgery. 2021 Nov 18;89(6):954-966. doi: 10.1093/neuros/nyab358.

Coagulopathy and Progression of Intracranial Hemorrhage in Traumatic Brain Injury: Mechanisms, Impact, and Therapeutic Considerations

Marc Maegele[1,2,3]

Dnešní hot to

Stroke
Volume 53, Issue 7, July 2022; Pages e282-e361
https://doi.org/10.1161/STR.0000000000000407

AHA/ASA GUIDELINE

2022 Guideline for the Manage
Spontaneous Intracerebral Hem
American Heart Association/Am

Rossaint et al. Critical Care (2023) 27:80
https://doi.org/10.1186/s13054-023-04327-7

GUIDELINES

The European guideline on r
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Rolf Rossaint[1*], Arash Afshari[2], Bertil Bouillon[3], Vladimir Cerny[4,5], D
Jacques Duranteau[9], Daniela Filipescu[10], Oliver Grottke[1], Lars Grø
Alexander Kaserer[13], Radko Komadina[14], Mikkel Herold Madsen[2],
Carolina S. Romero[18], Charles-Marc Samama[19], Jean-Louis Vincent

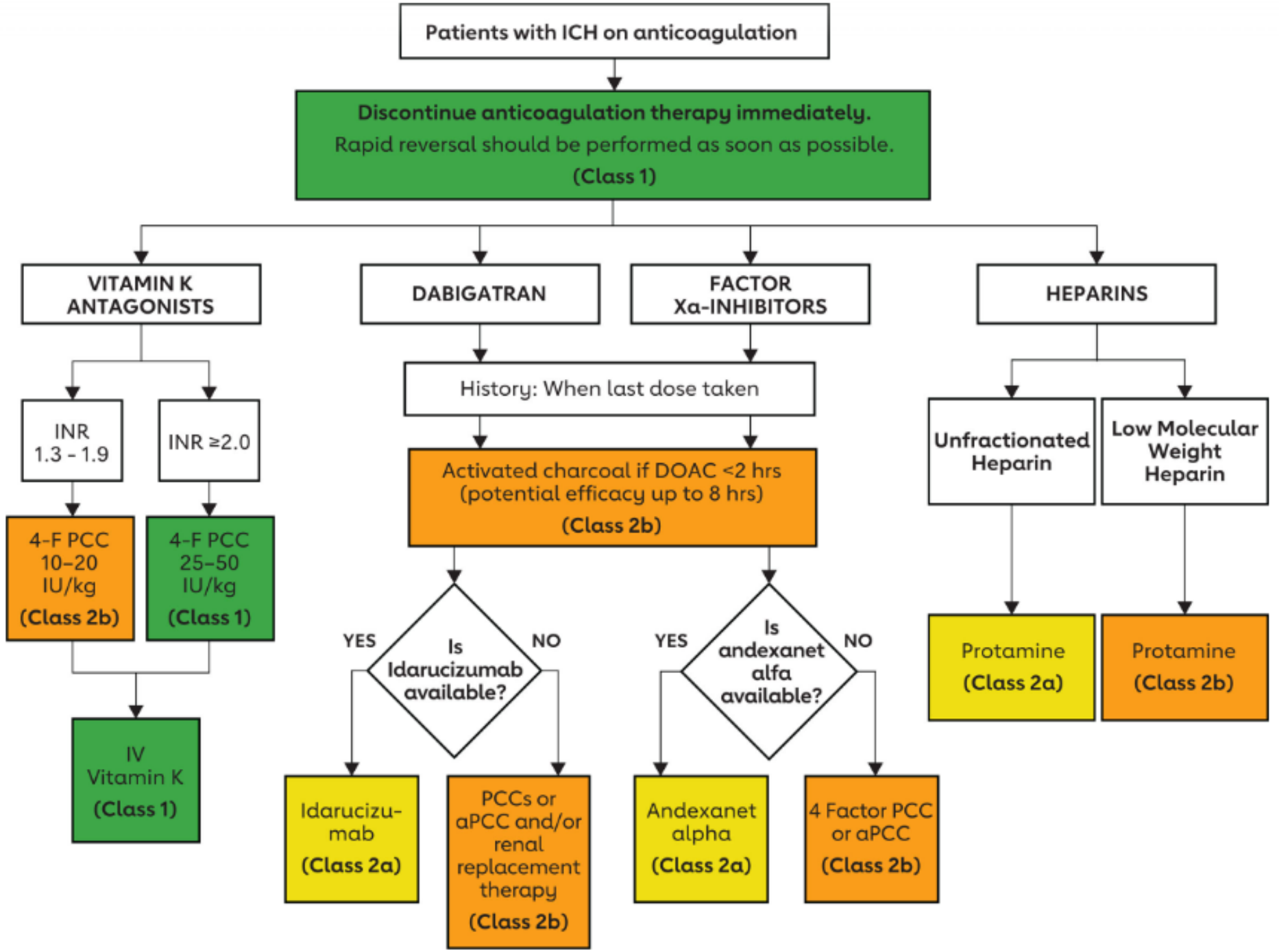
EJA

GUIDELINES

**Management of severe peri-operat
from the European Society of Ana
Intensive Care**

Second update 2022

Sibylle Kietaibl, Aamer Ahmed, Arash Afshari, Pierre Alba
Giedrius Barauskas, Edoardo De Robertis, David Farao
Anne Godier, Thorsten Haas, Matthias Jacob, Marcus D.
Zsolt Molnar, Lidia Mora, Niels Rahe-Meyer, Charles M. S
Christoph Schlimp, Anne J. Wikkelsø and Kai Zacharows



Jak zjistím, že pacient s TBI nebo hCMP užívá DOAC?

- ANAMNÉZA !!!
  - Zda pacient užívá DOAC a kdy ho naposledy užil
- Laboratorní testy
  - PT, PT-R, aPTT, TT, Fbg, Plt, DD, kreat
  - Hladina DOAC
    - Dabigatran – dTT, anti-IIa, ECT
    - Xabany – specifické anti-Xa
- Viskoelastické metody
  - Test na vnější cestu, vnitřní cestu a fibrinogen
  - RVV-test, ECA-test (ClotPro®)
- Screeningové testy z moči
  - DOASENSE® - DOAC Dipstick

R3.5: In urgent surgery with a high risk of bleeding, the plasma concentrations of DOACs above 50 ng ml^{-1} may be considered for haemostatic or antidote intervention. **(3)**

EJA

Eur J Anaesthesiol 2024; **41**:327–350

GUIDELINES

Clinical guideline on reversal of direct oral anticoagulants in patients with life threatening bleeding

Oliver Grottke, Arash Afshari, Aamer Ahmed, Eleni Arnaoutoglou, Daniel Bolliger, Christian Fenger-Eriksen and Christian von Heymann

Terapie – TBI, hCMP + Dabigatran

PICO 8

Clinical scenario: Adult patients on dabigatran therapy, who present with severe bleeding in urgent surgical or nonsurgical settings.

Should idarucizumab or PCC, aPCC or rFVIIa be used to manage dabigatran associated bleeding in urgent surgical or nonsurgical settings?

Recommendation

R8.1: We recommend that idarucizumab should be considered in patients under dabigatran therapy presenting with severe bleeding or in urgent surgical or nonsurgical settings. **(1C)**

R 8.2: In the absence of the availability of idarucizumab, we suggest the use of PCC or aPCC. However, the superiority of one agent over another has not been demonstrated. **(2C)**

R 8.3: Due to the paucity of clinical data, we are unable to provide any recommendation for the use of rFVIIa. **(3)**

EJA

Eur J Anaesthesiol 2024; **41**:327–350

GUIDELINES

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Terapie – TBI, hCMP + Xabany

PICO 7

Clinical scenario: Adult patients on FXa inhibitor therapy, who present with severe bleeding in urgent surgical or nonsurgical settings.

Should andexanet alfa or PCC, aPCC or rFVIIa be used to manage FXa inhibitor-associated bleeding in urgent surgical or nonsurgical settings?

R7.1: We recommend that PCC or andexanet alfa should be considered in patients under FXa inhibitor therapy presenting with severe bleeding. However, the superiority of one agent over another has not been demonstrated. (**1C**)

R7.2: In the absence of the availability of andexanet alfa and PCC, aPCC may be considered in patients on FXa inhibitor therapy presenting with severe bleeding. (**2C**)

R7.3: Due to the paucity of clinical data, we are unable to provide any recommendation for the use of rFVIIa in patients on FXa inhibitor therapy presenting with severe bleeding. (**3**)

EJA

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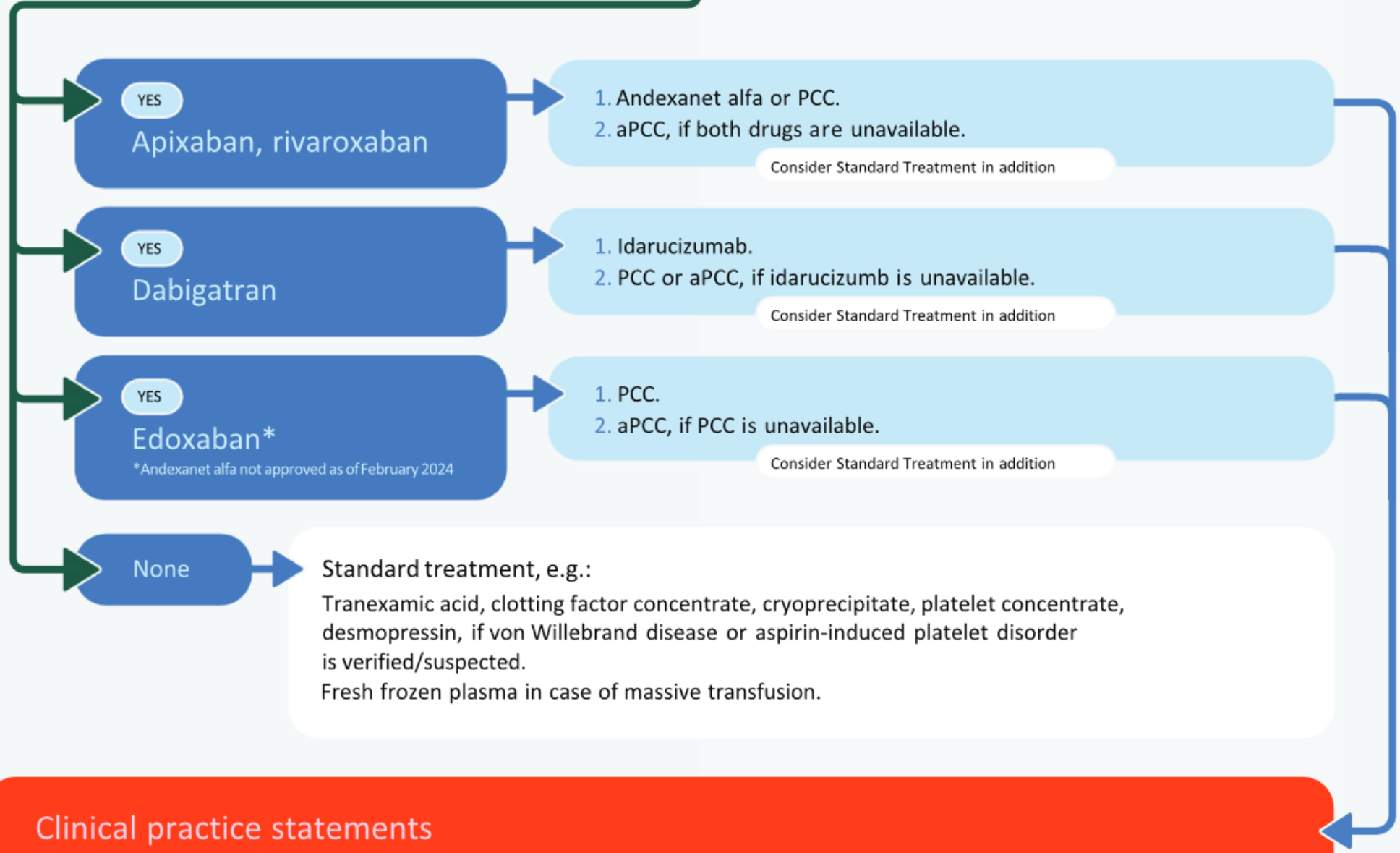

Clinical practice statements

In case of progression to severe or life-threatening bleeding: Rule out surgical source of bleeding; continue standard treatment; consider a second antidote dose or PCC/aPCC dose if there are persistently elevated DOAC levels.
Recurrent bleeding: Consider that elevated plasma levels of apixaban, rivaroxaban and dabigatran may occur after specific antidote application.
Terminated bleeding (e.g.>24-48 hours): Consider resumption of anticoagulation e.g. LMWH at prophylactic dosage or local standard.

Anticoagulant	Antidote	Non-specific haemostatic agent
Dabigatran	Idarucizumab 2x2,5 g over 5-10 minutes, infusions no more than 10 minutes apart.	Not approved: PCC or aPCC at a dose of 25-50 IU/kg; rFVIIa: No recommendation
Apixaban	Low dose: Andexanet alfa 400 mg bolus over 15 minutes followed by an 480 mg infusion over 2 hours High dose: Andexanet alfa 800 mg bolus over 30 minutes followed by an 960 mg infusion over 2 hours	Not approved: PCC or aPCC at a dose of 25-50 IU/kg; rFVIIa: No recommendation
Edoxaban	Andexanet alfa not approved as of February 2024.	Not approved: PCC or aPCC at a dose of 25-50 IU/kg; rFVIIa: No recommendation
Rivaroxaban	Low dose: Andexanet alfa 400 mg bolus over 15 minutes followed by an 480 mg infusion over 2 hours High dose: Andexanet alfa 800 mg bolus over 30 minutes followed by an 960 mg infusion over 2 hours	Not approved: PCC or aPCC at a dose of 25-50 IU/kg; rFVIIa: No recommendation

Drug *FXa Inhibitor*	**Dose** *Strength of Last Dose*	**Time** *Since Last Dose Taken*	
		<8 Hours or Unknown	*≥8 Hours*
Xarelto® (rivaroxaban)	≤10 mg	Low dose	Low dose
	>10 mg or unknown	High dose	
Eliquis® (apixaban)	≤5 mg	Low dose	Low dose
	>5 mg or unknown	High dose	

Terapie – TBI, hCMP + Xabany

RESULTS

A total of 263 patients were assigned to receive andexanet, and 267 to receive usual care. Efficacy was assessed in an interim analysis that included 452 patients, and safety was analyzed in all 530 enrolled patients. Atrial fibrillation was the most common indication for factor Xa inhibitors. Of the patients receiving usual care, 85.5% received prothrombin complex concentrate. Hemostatic efficacy was achieved in 150 of 224 patients (67.0%) receiving andexanet and in 121 of 228 (53.1%) receiving usual care (adjusted difference, 13.4 percentage points; 95% confidence interval [CI], 4.6 to 22.2; P=0.003). The median reduction from baseline to the 1-to-2-hour nadir in anti–factor Xa activity was 94.5% with andexanet and 26.9% with usual care (P<0.001). Thrombotic events occurred in 27 of 263 patients (10.3%) receiving andexanet and in 15 of 267 (5.6%) receiving usual care (difference, 4.6 percentage points; 95% CI, 0.1 to 9.2; P=0.048); ischemic stroke occurred in 17 patients (6.5%) and 4 patients (1.5%), respectively. There were no appreciable differences between the groups in the score on the modified Rankin scale or in death within 30 days.

Terapie – TBI, hCMP + Xabany

CONCLUSIONS

Among patients with intracerebral hemorrhage who were receiving factor Xa inhibitors, andexanet resulted in better control of hematoma expansion than usual care but was associated with thrombotic events, including ischemic stroke. (Funded by Alexion Astra-Zeneca Rare Disease and others; ANNEXA-I ClinicalTrials.gov number, NCT03661528.)

- **PCC nebo andexanet alfa?**
  - Závažnost krvácení (čas)
  - Zdroj krvácení
  - Trombembolické riziko
  - Typ xabanu
  - Zdroje

A co dál po kontrole krvácení?

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Critical Care

GUIDELINES Open Access

The European guideline on management of major bleeding and coagulopathy following trauma: sixth edition

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Recommendation 37

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The optimal timing for the initiation of pharmacological thromboprophylaxis remains inadequately investigated, especially after TBI. Retrospective studies looking at TBI show that there are fewer VTE if thromboprophylaxis is started sooner (within 24–72 h of injury) rather than later, without increased bleeding risk [380], but how much earlier thromboprophylaxis can be used with efficacy and safety is the subject of future clinical trials. We suggest that pharmacological VTE prophylaxis be initiated with either LMWH, or low-dose UFH in patients with renal failure, as early as possible, only after a head CT confirms that ICH is stable and in the absence of persistent bleeding.

A co dál po kontrole krvácení?

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AHA/ASA GUIDELINE

2022 Guideline for the Management of Patients With Spontaneous Intracerebral Hemorrhage: A Guideline From the American Heart Association/American Stroke Association

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COR	LOE	Recommendations
Prophylaxis		
1	B-R	1. In nonambulatory patients with spontaneous ICH, intermittent pneumatic compression (IPC) starting on the day of diagnosis is recommended for VTE (DVT and pulmonary embolism [PE]) prophylaxis.[275,276]
2a	C-LD	2. In nonambulatory patients with spontaneous ICH, low-dose UFH or LMWH can be useful to reduce the risk for PE.[277–280]
2b	C-LD	3. In nonambulatory patients with spontaneous ICH, initiating low-dose UFH or LMWH prophylaxis at 24 to 48 hours from ICH onset may be reasonable to optimize the benefits of preventing thrombosis relative to the risk of HE.[277,281,282]

Take home message

- V neurointenzivní péči musíme umět urgentně vyřešit krvácivý či trombotický stav a poté preventovat trombembolické komplikace a zároveň minimalizovat krvácivé komplikace

- V pátrání po koagulopatii používejte všechny dostupné metody, včetně specifických testů pro DOAC

- V případě koagulopatie vyvolané DOAC u TBI či hCMP, použijte specifická antidota anebo PCC, tak jak je pro jednotlivé DOAC doporučeno