Antikoagulált koponyasérült, koponyasérült antikoagulációja, melyik ujjamat harapjam

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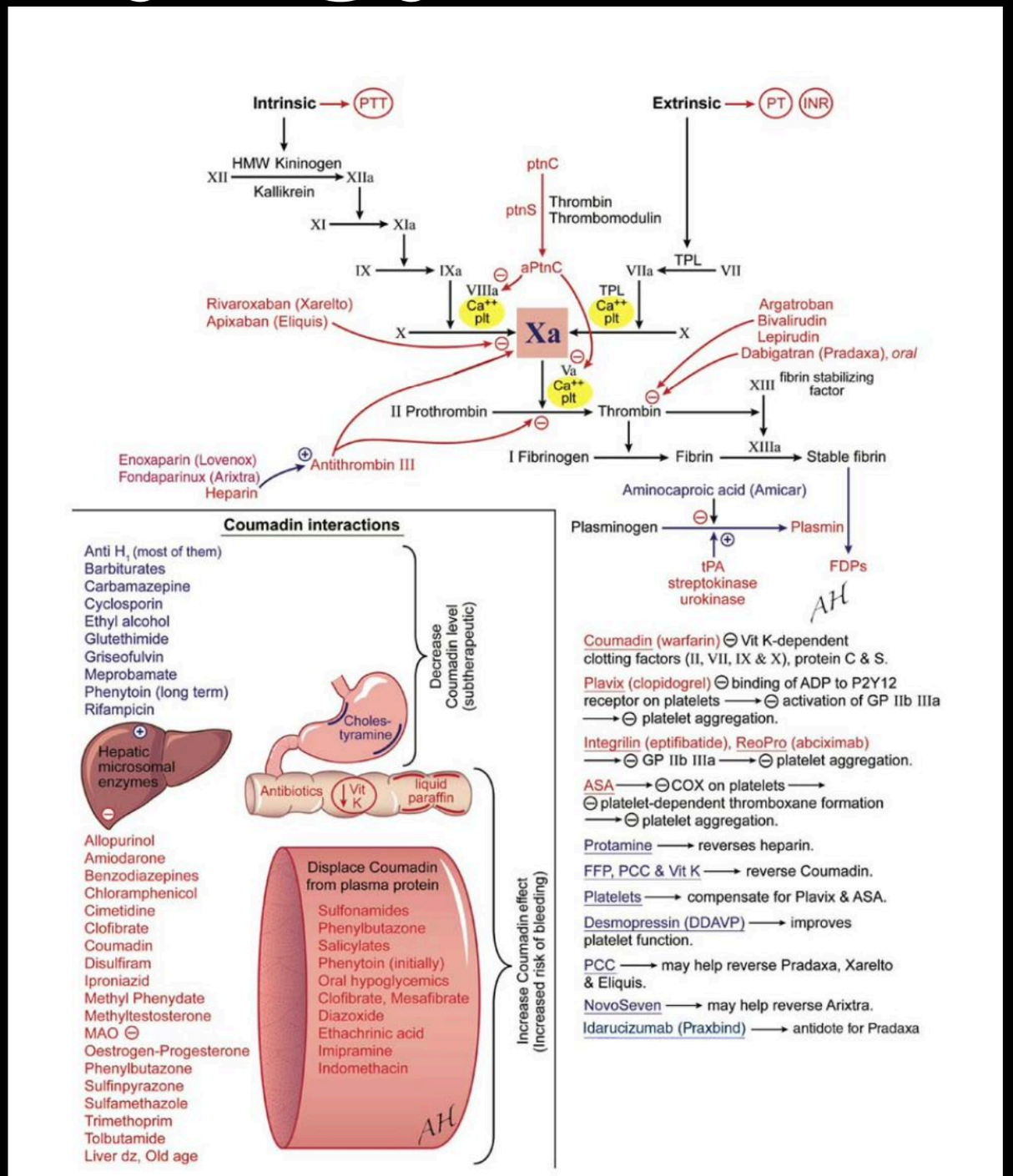
Szeged 2024.03.08

Problémák

- Nagy létszámú, polymorbid beteganyag
- Multidisciplináris szemlélet (intenzív therapia, kardiológia, hematológia, ér-szívsebészet) lenne szükséges
- Hiányzó guideline, anekdotikus, “így szoktuk” elvek
- Rengeteg gyógyszer
- Ismételt vérzés
- Indikáció?

Gyógyszerek



Indikáció-alvadásgátló

- Pitvarfibrillatio
- Mechanikus műbillentyű (VKA)
- Tüdőembólia
- Mély vénás thrombosis (iniciálisan 3 hónapig vagy rizikófaktor eliminációáig)
- Sinus thrombosis (spontán)

NOAC/DOAC

- Rivaroxaban (Xarelto®) máj, felezési idő: 5-9 óra
- Apixaban (Eliquis) epeút, 25%-ban vese!, felezési idő 9-14 óra
- Endoxaban (Lixiana) széklet, vese felezési idő: 10-14 óra
- Dabigatran (Pradaxa®) -direkt thrombin inhibitor, vese felezési idő: 12-17 óra

Warfarin vs NOAC

Warfarin vs NOACs

Feature	Warfarin	NOACs
Onset	Slow	Rapid
Dosing	Variable	Fixed
Food effect	Yes	No
Drug interactions	Many	Few
Routine lab monitoring	Yes	No
Half-life	Long	Short
Reversal agent	Yes	Maybe

Indikáció-TAG

- ACS
- Stent (bármely érszakaszban), műbillentyű
- Stroke
- Stabil angina, CABG műtét
- Essentialis thrombocytosis
- Egyéb ritka indikációk (Kawasaki)

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

Effects of preinjury pharmacotherapies

The demographic change towards TBI in older age is accompanied by an increased incidence of comorbidities in patients with TBI,[27,48] and modern treatment of chronic cerebrovascular and coronary artery disease means that these patients are often taking anticoagulant or antiplatelet drugs, both of which have been explored as causes of increased bleeding and worse outcome after TBI.[27,49–53] According to a meta-analysis,[49] patients taking warfarin at the time of TBI have double the risk of poor outcome than patients not taking warfarin, but a similar analysis of antiplatelet therapy did not show a clear increase in risk.[50] Although retrospective evidence echoes this observation,[52] other studies[51,54,55] have suggested that preinjury use of antiplatelet therapy could result in a two-times higher occurrence of traumatic ICH even after mild TBI than in patients not taking antiplatelet therapy, particularly in the elderly population. Preinjury clopidogrel or warfarin intake are independent predictors of immediate traumatic

4 PRIMARY PREVENTION

Despite the now well-established role of aspirin in secondary cardiovascular prophylaxis, the benefit/risk ratio in primary prevention is far less clear. In low- and middle-income countries, aspirin-containing polypill strategies have proved effective in preventing major cardiovasacular events, for example in the PolyIran study.[24] However, a large meta-analysis conducted by the Antithrombotic Trialists' collaboration from 2009 questioned the net benefit of aspirin in primary prevention as a result of an observed increased risk of major extracranial and gastrointestinal bleeding complications in spite of only a small protective effect against vascular events.[25] These findings have been confirmed by the most recent trials conducted in primary prevention: for example, ASPREE, which focused on elderly subjects, ASCEND, which studied patients with diabetes, and ARRIVE, which examined patients with a moderate estimated risk of a first cardiovascular event,[26-28] showing that the net benefit of aspirin in this setting is marginal at best whilst posing a major bleeding hazard in subjects with cardiovascular risk factors who are otherwise healthy. At present, therefore, a cautious approach is advised as regards the use of aspirin in primary prevention, weighing the benefit to risk ratio in order to personalise treatment.

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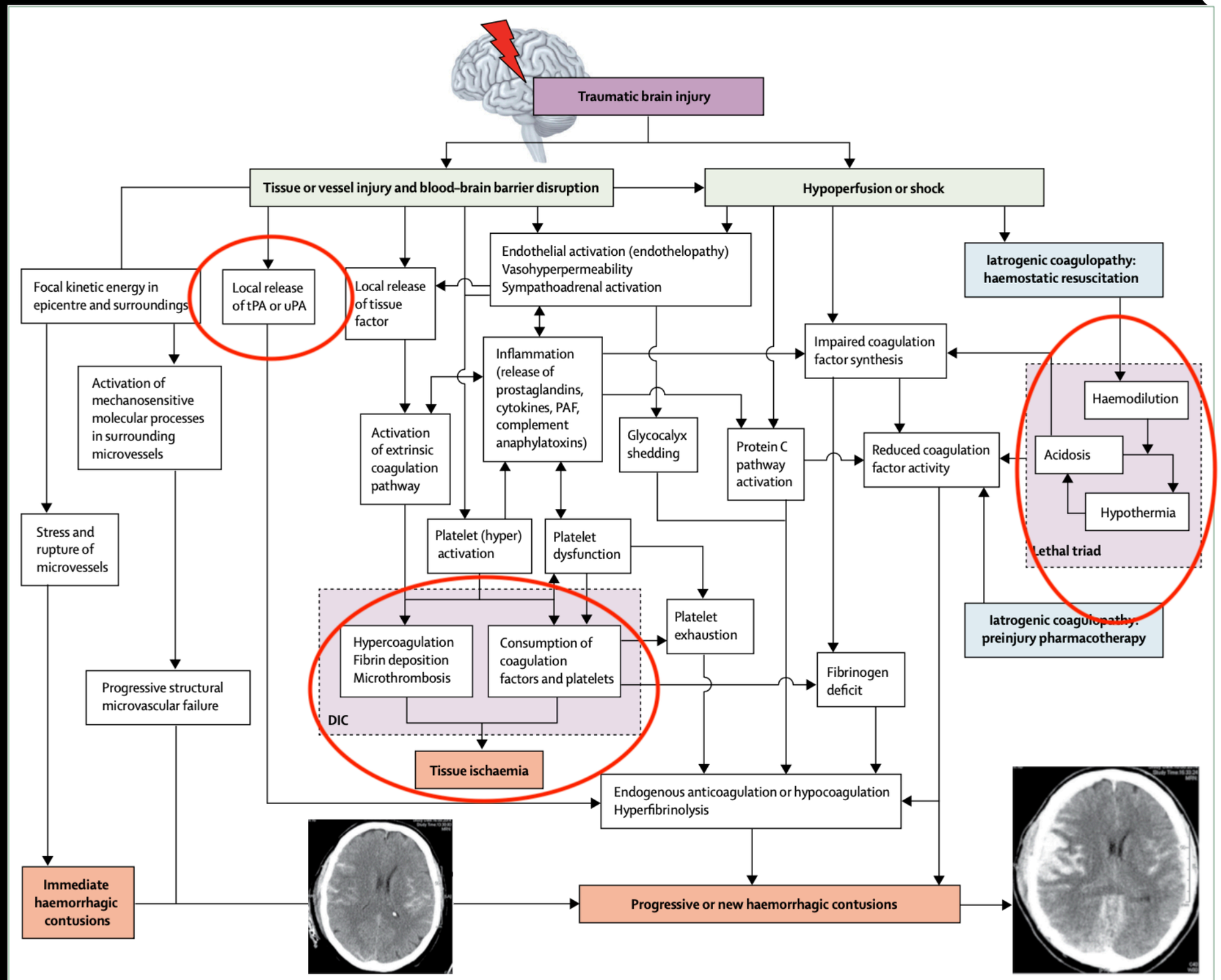
Ha ez nem lenne elég



Traumás coagulopathia

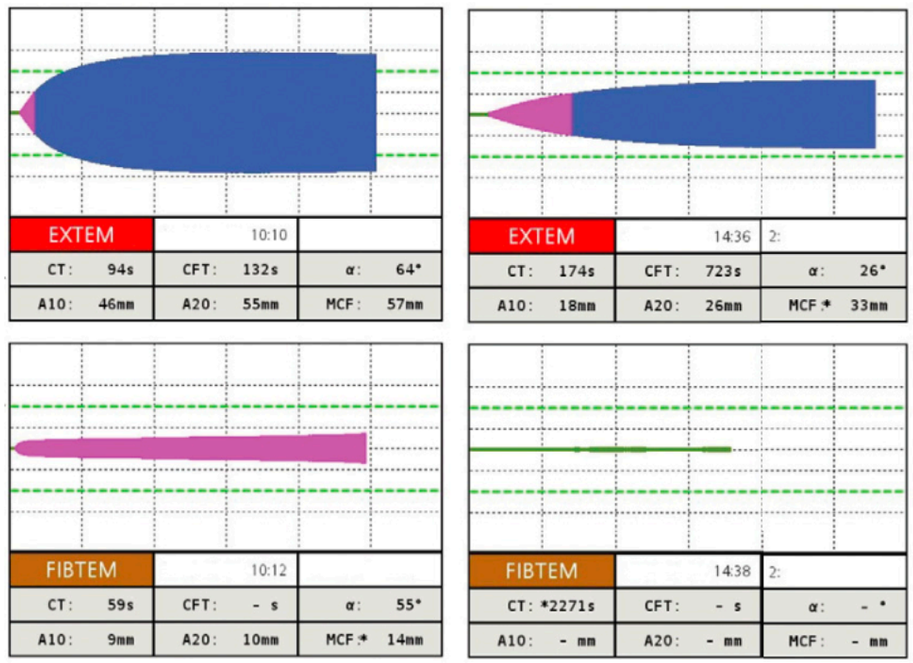
- Definíció szerint: aPTT >40 sec vagy INR >1,2 vagy <120 ezer thrombocyta

- Újabb ajánlások: funkcionális alvadási tesztekkel KIEGÉSZÍTVE! (ROTEM: EXTEM, FIBTEM)

- Aggregációs tesztek (ASPI, ADP)

Viscoelastográphias tesztek

B Point-of-care viscoelastic testing (ROTEM)



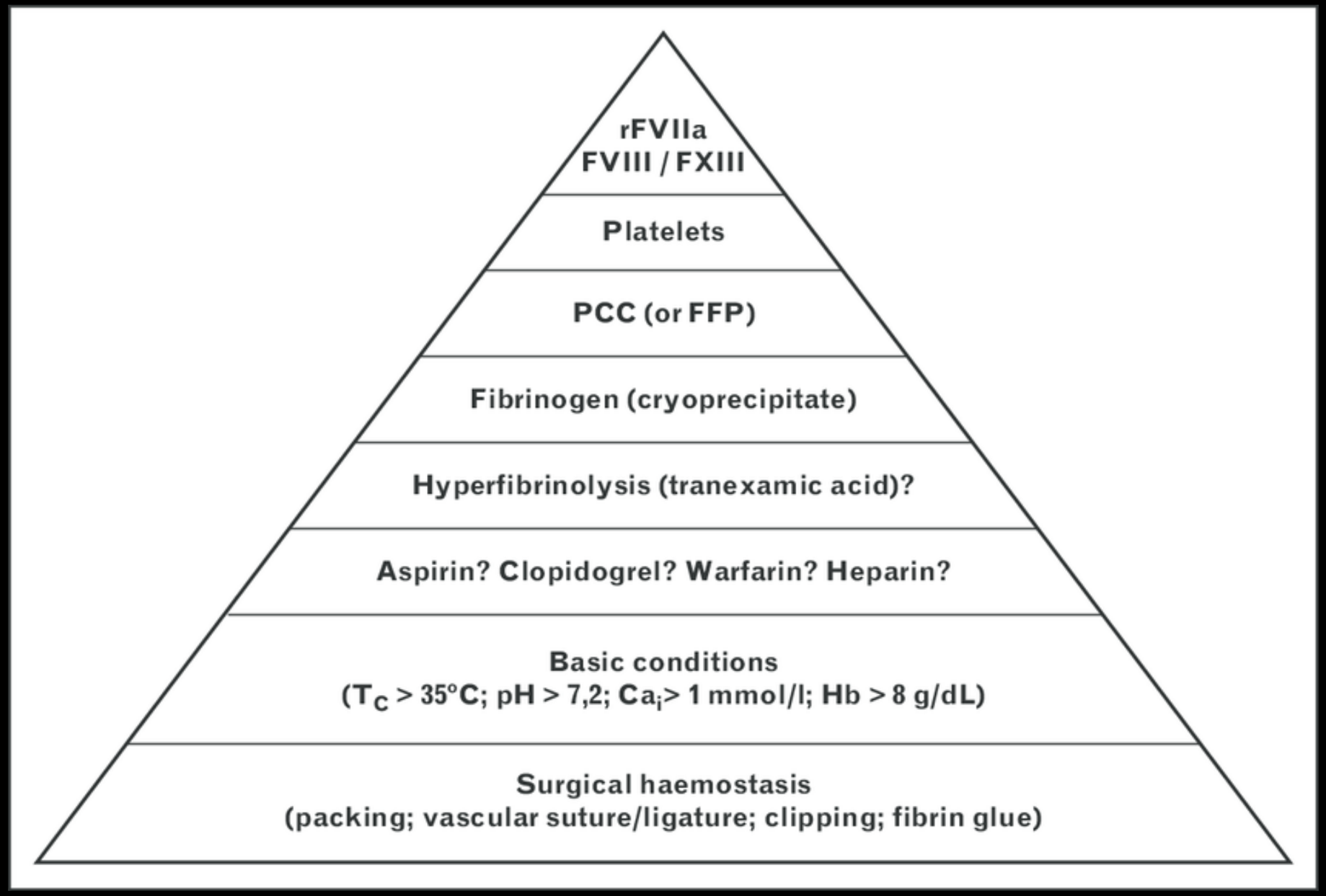
C Standard laboratory test results

Haemoglobin	12·6 g/dL↓	6·6 g/dL↓
Platelets	174×10⁹/L	55×10⁹/L↓
pH	7·33↓	7·22↓
BE	–2·7 mmol/L	–8·7 mmol/L↓
Lactate	0·99 mmol/L	6·99 mmol/L↑
PTr	60%↓	15%↓
PT	16·3 s↑	45·3 s↑
aPTT	40 s↑	161 s↑
Fibrinogen	135 mg/dL↓	81 mg/dL↓

Figure 2: **Clinical assessment of progression of haemorrhagic injury and coagulopathy after traumatic brain injury**

A 45-year-old male patient with severe traumatic brain injury had cranial CT scans and coagulopathy assessments with viscoelastic assays and conventional coagulation assays at emergency department admission and 3 h after admission. (A) Clinical CT scans show deteriorating intracranial haemorrhage. (B) Clinical viscoelastic assessments using ROTEM were based on the EXTEM and FIBTEM subtests: the EXTEM test is used as a screening test for the (extrinsic) haemostasis system and the FIBTEM test is an assay for the fibrin part of the clot. Assay results are assessed along the time axis from left to right. The patient viscoelastic findings at admission indicate early signs of coagulopathy, with abnormal clot formation reflected in prolonged EXTEM clotting times (CT, 94 s) and a reduced FIBTEM clot amplitude after 10 min (A10, 9 mm), indicative of reduced clot stability due to fibrinogen deficiency. At 3 h after admission, findings indicate delayed and insufficient clotting, reflected in prolonged EXTEM CT (174 s) and absent FIBTEM A10, suggesting the development of complicating hypotensive (multifactorial) coagulopathy associated with the deteriorating haemorrhage. The flat line in the FIBTEM channel reflects complete absence of fibrin polymerisation. Abnormal clot formation can also be indicated by a prolonged CFT, a reduced angle reflecting the speed of clot formation (α), or a reduced MCF, which reflects overall clot stability and sustainability (for more on assessment parameters for viscoelastic tests, see appendix). (C) The results from standard laboratory assays and CCAs at 3 h confirm severe shock with coagulation failure along with hypofibrinogenaemia and thrombocytopenia (arrows indicate abnormal findings). The more rapid availability of viscoelastic test results compared with CCA and standard laboratory test results enabled timely, targeted treatment of bleeding in this patient. BE=base excess. PTr=prothrombin ratio. PT=prothrombin time. aPTT=activated partial thromboplastin time. CFT=clot formation time. MCF=maximum clot firmness. CCAs=conventional coagulation assays.

Felfüggesztés



Felfüggesztés

- TAG, érdemes megvárni a hatás elmúltát, akut esetben thrombocyta készítmény+ desmopressin- irodalom alapján hatása kérdéses (PATCH study-non traumás IC vérzés)

Felfüggesztés

Routine platelet transfusion in patients with traumatic intracranial hemorrhage taking antiplatelet medication: Is it warranted?

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Background: After a traumatic intracranial hemorrhage (tICH), patients often receive a platelet transfusion to reverse the effects of antiplatelet medication and to reduce neurologic complications. As platelet transfusions have their own risks, this study evaluated their effects on tICH progression, need for operations and mortality.

Methods: In this retrospective study, we identified patients admitted to a level 1 trauma centre with a tICH from 2011 to 2015 who were taking acetylsalicylic acid (ASA) or clopidogrel, or both. We categorized patients into 2 groups: platelet transfusion recipients and nonrecipients. We collected data on demographic characteristics, changes in brain computed tomography findings, neurosurgical interventions, in-hospital death and intensive care unit (ICU) length of stay (LOS). We used multivariable logistic regression to compare outcomes between the 2 groups.

Results: We identified 224 patients with tICH, 156 (69.6%) in the platelet transfusion group and 68 (30.4%) in the no transfusion group. There were no between-group differences in progression of bleeds or rates of neurosurgical interventions. In the transfusion recipients, there was a trend toward increased ICU LOS (adjusted odds ratio [OR] 1.59, 95% confidence interval [CI] 0.74–3.40) and in-hospital death (adjusted OR 3.23, 95% CI 0.48–21.74).

Conclusion: There were no differences in outcomes between patients who received platelet transfusions and those who did not; however, the results suggest a worse clinical course, as indicated by greater ICU LOS and mortality, in the transfusion recipients. Routine platelet transfusion may not be warranted in patients taking ASA or clopidogrel who experience a tICH, as it may increase ICU LOS and mortality risk.

Felfüggesztés

- TAG, érdemes megvárni a hatás elmúltát, akut esetben thrombocyta készítmény+ desmopressin- irodalom alapján hatása kérdéses (hatás mortalitásra kérdéses-PATCH study-non traumás IC vérzés)
- VKA- PCC+ K1 vitamin (Konakion) INR 1,4-5-> 500 NE Beriplex, >5 1000 NE Beriplex
- NOAC- antidótumok, annak hiányában PCC
- Enoxaparin, Heparin- protamin (enoxaparin mg-ként 1 mg, heparin 100 NE-ként 1 mg)

Intracranial hemorrhage: Reversal of anticoagulant, antiplatelet and thrombolytic agents

Non traumatic / spontaneous intracranial hemorrhages and intraventricular hemorrhages
Subdural hemorrhages without underlying significant parenchymal traumatic brain injury

Warfarin **INR known 1.4- 5** Vit K 10 mg IV plus FEIBA 500 units IV once **INR >5** Vit K 10 mg IV plus FEIBA 1000 units IV once **INR unknown Draw PT/INR** Vit K 10 mg IV plus FEIBA 500 units IV once Pre FEIBA INR >1.4 -5no additional FEIBA necessary Pre FEIBA INR >5 additional FEIBA 500 units IV once **Repeat INR 15-60 min after infusion complete - if INR >1.4- repeat Vit K 10 mg IV once . Consider additional** FEIBA 500 units IV once **Repeat INR every 6 hours until 2 consecutive values show <1.4**	**Rivaroxaban / Apixaban / Edoxaban** consider activated charcoal if dose within 2 hours Kcentra 50 units /kg once If time since last dose is > 5 elimination half-lives may not reverse, 3-5 half lives reverse with 25 units/kg Dabigatran Praxbind 5 mg IV once. Check Thrombin time
Heparin IV IV protamine 1 mg for 100 units of heparin the patient received during previous 3 hours Reduce dose by half if 30-60 minutes have elapsed since heparin given. Max protamine dose 50 mg Fondaparinaux FEIBA 20 units /kg Argatroban Stop infusion. Supportive care. Dialyze of liver failure Bivalirudin Renal consult for dialysis for critical bleeding.	**Enoxaparin** Anti-Factor Xa levels Administer Protamine slowly 50 mg over 10 min <8 hours since last dose with normal renal function- IV protamine 1 mg for every 1 mg of enoxaparin administered. 8-24 hours since last dose with normal renal function IV protamine 0.5 mg for every 1 mg of enoxaparin administered 8-24 hours since last dose with IMPAIRED renal function IV protamine 1 mg for every 1 mg of enoxaparin administered. >24 hours since last dose with IMPAIRED renal function IV protamine 0.5 mg for every 1 mg of enoxaparin administered
Platelet units 1 unit if Aspirin /dipyridamole/cilostazol. 2 units if clopdogrel/Prasugrel/ticlopidine /ticagreclor or dual agents More than 2 units may be needed if ticagreclor	Non surgical bleeds on single antiplatelet agents No action OR ddAVP 0.4 mcg/kg once only. Non surgical bleeds on dual antiplatelet agents No action or ddAVP 0.4 mcg/kg ± plus platelet transfusion 1 unit Surgical bleeds /EVD planned ddAVP 0.4 mcg/kg once only PLUS platelet transfusion.
tPA REVERSAL If CT scan shows significant, hemorrhage causing symptomatic decline Cryoprecipitate 0.15 units/kg (or 10 units) , IF fibrinogen < 150 mg/dL. Repeat fibrinogen level 30 minutes after infusion. Repeat cryo Amicar 4-5 gram over 20 minutes	

Progressio

- Leggyakrabban 12 órán belüli, ritkábban 48 óra alatt
- Leggyakoribb MEGELŐZHETŐ halálok TBI esetén
- Tramexánsav 3 órán belül
- A coagulopathia a morbiditást, és mortalitást is rontja
- Gyanú esetén konvencionális alvadás határértéken belüli volta esetén is funkcionális alvadási tesztek végzendőek!
- Alapvető dolgok is lehetnek végzetesek!

Progressio

Effects of tranexamic acid on death, disability, vascular occlusive events and other morbidities in patients with acute traumatic brain injury (CRASH-3): a randomised, placebo-controlled trial

Results Between July 20, 2012, and Jan 31, 2019, we randomly allocated 12737 patients with TBI to receive tranexamic acid (6406 [50·3%] or placebo [6331 [49·7%], of whom 9202 (72·2%) patients were treated within 3 h of injury. Among patients treated within 3 h of injury, the risk of head injury-related death was 18·5% in the tranexamic acid group versus 19·8% in the placebo group (855 *vs* 892 events; risk ratio [RR] 0·94 [95% CI 0·86–1·02]). In the prespecified sensitivity analysis that excluded patients with a GCS score of 3 or bilateral unreactive pupils at baseline, the risk of head injury-related death was 12·5% in the tranexamic acid group versus 14·0% in the placebo group (485 *vs* 525 events; RR 0·89 [95% CI 0·80–1·00]). The risk of head injury-related death reduced with tranexamic acid in patients with mild-to-moderate head injury (RR 0·78 [95% CI 0·64–0·95]) but not in patients with severe head injury (0·99 [95% CI 0·91–1·07]; p value for heterogeneity 0·030). Early treatment was more effective than was later treatment in patients with mild and moderate head injury (p=0·005) but time to treatment had no obvious effect in patients with severe head injury (p=0·73). The risk of vascular occlusive events was similar in the tranexamic acid and placebo groups (RR 0·98 (0·74–1·28). The risk of seizures was also similar between groups (1·09 [95% CI 0·90–1·33]).

Interpretation Our results show that tranexamic acid is safe in patients with TBI and that treatment within 3 h of injury reduces head injury-related death. Patients should be treated as soon as possible after injury.

Progressio

Antithrombotic agents and traumatic brain injury in the elderly population: hemorrhage patterns and outcomes

Pasquale Scotti, MSc,[1] Chantal Séguin, MD, FRCPC,[2] Benjamin W. Y. Lo, MD, PhD, FRCSC,[1] Elaine de Guise, PhD,[3] Jean-Marc Troquet, MD,[4] and Judith Marcoux, MD, MSc, FRCSC[1]

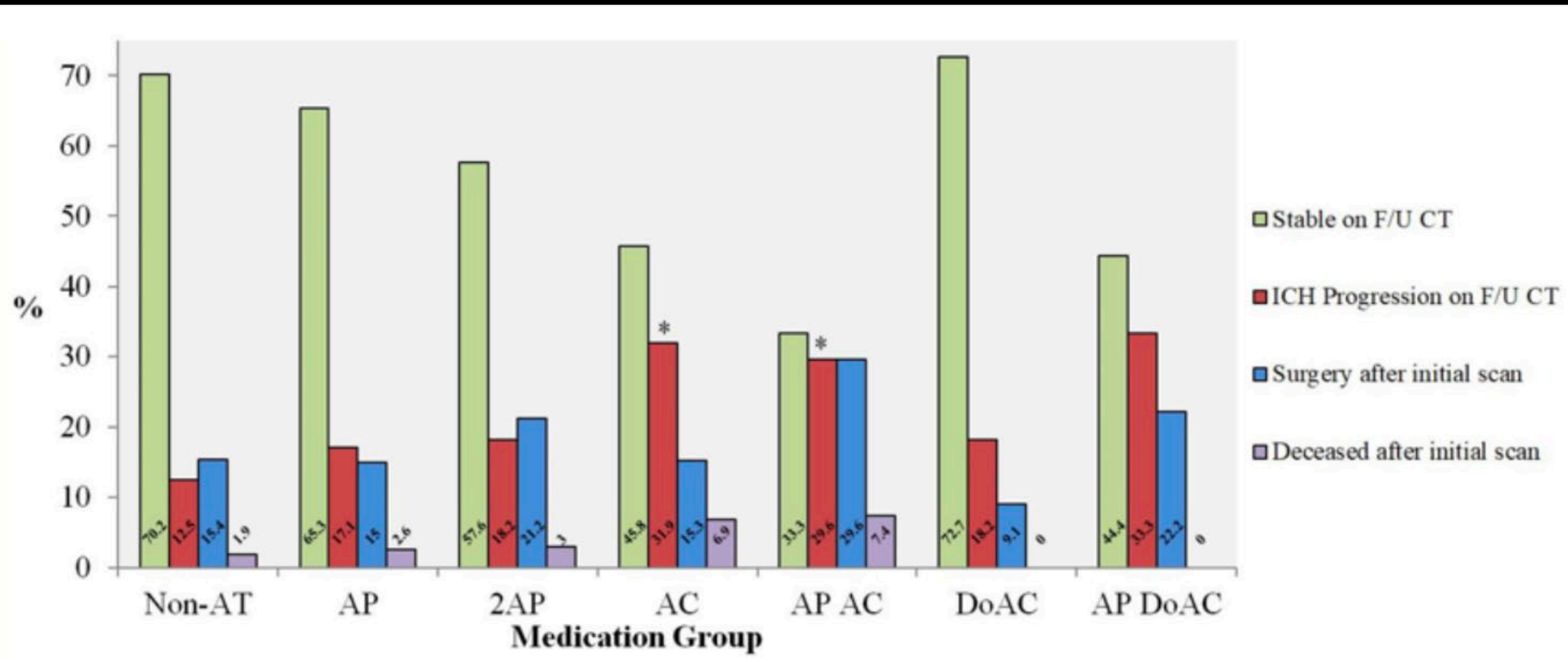
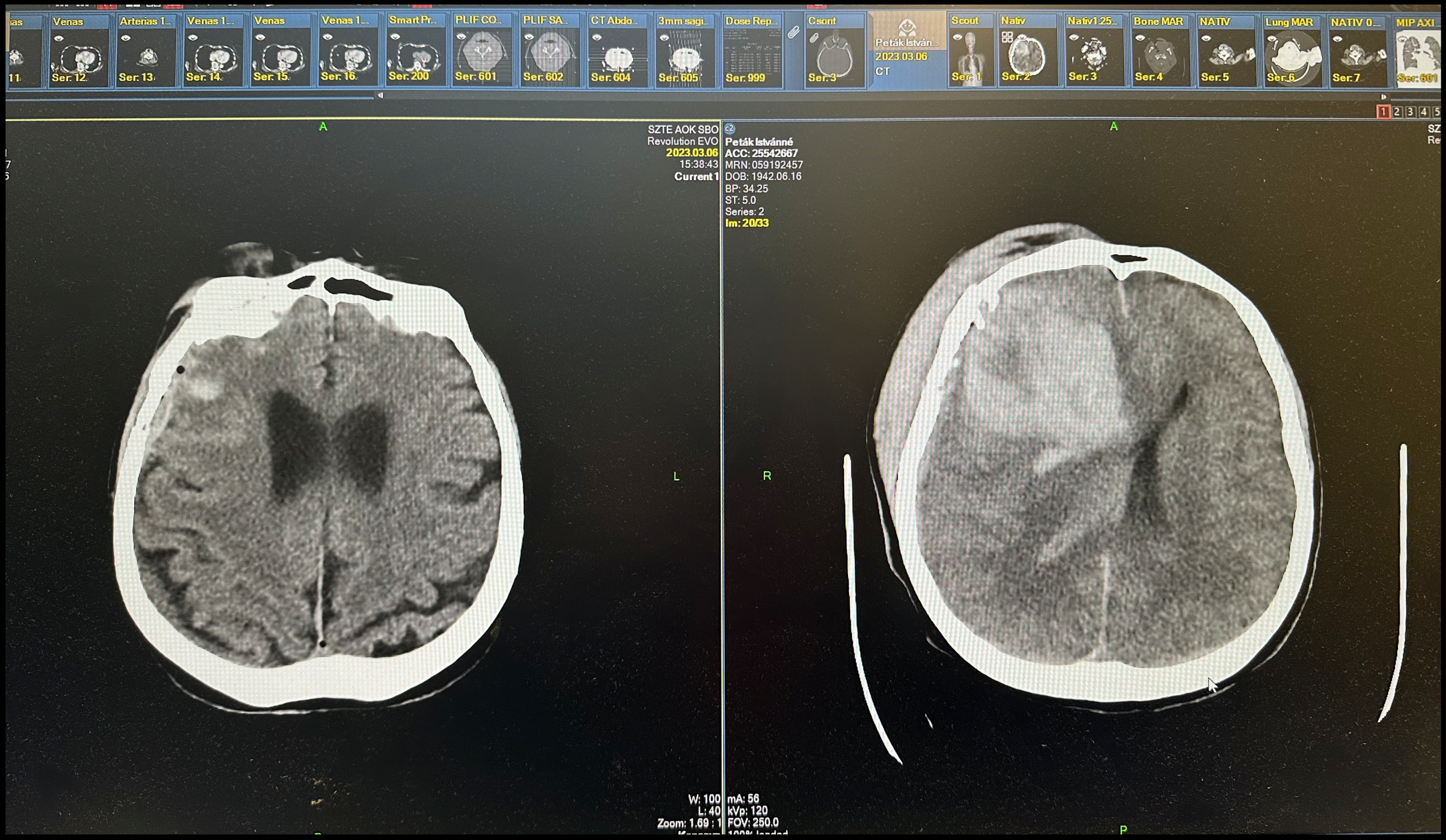


FIG. 3. ICH progression categories for patients with ICH (n = 564). *Statistically significant difference ($p \leq 0.05$) on multivariate logistic regression compared with nonusers. F/U = follow-up.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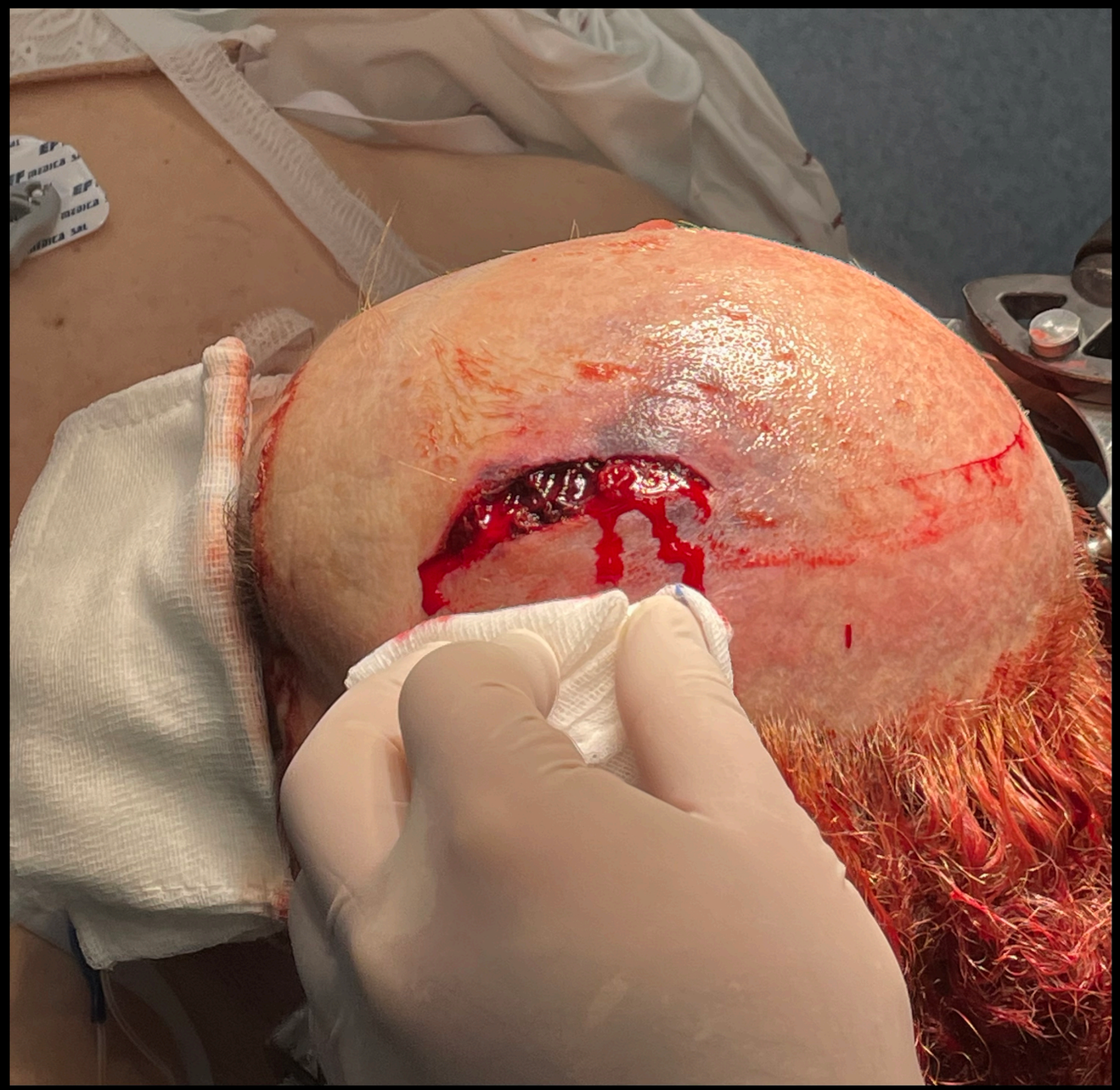
Sebészi hemostasis!



Visszaadás

- Talán a legbonyolultabb kérdés
- Mikor? (Anekdotikus válaszok)
- VKA mellett létrejövő major vérzés esetén NOAC-ra váltás
- HAS-BLED score

Visszaadás

- Európai Stroke ajánlás és AHA ajánlás alapján 7-10 nap után (anticoagulatio), korai 15 napon belüli visszaállítás jobb
- Ismételt vérzés általában korai, thromboembolias szövődmény KÉSEI
- Magas rizikójú esetben (műbillentyű) korai visszaadás
- Ismételt 48 óra alatt készült scan-eken progresszió hiánya, stabil IC status!

Both the EARLY and LATE groups had a similar risk of 1-year recurrent ICH (EARLY versus LATE: 3.12% versus 3.27%; adjusted hazard ratio [AHR], 0.967 [95% CI, 0.522–1.791]) after matching. Both groups also had a similar risk of each secondary outcome at 1-year follow-up. Subgroup analyses disclosed early antiplatelet resumption in the patients without prior cerebrovascular disease were associated with lower risks of all-cause mortality (AHR, 0.199 [95% CI, 0.054–0.739]) and major hemorrhagic events (AHR, 0.090 [95% CI, 0.010–0.797]), while early antiplatelet resumption in the patients with chronic kidney disease were associated with a lower risk of ischemic stroke (AHR, 0.065 [95% CI, 0.012–0.364]).

Conclusions:

Early resumption of antiplatelet was as safe as delayed antiplatelet resumption in ICH patients. Besides, those without

Visszaadás

Effects of antiplatelet therapy after stroke due to intracerebral haemorrhage (RESTART): a randomised, open-label trial

RESTART Collaboration † • Show footnotes

Between May 22, 2013, and May 31, 2018, 537 participants were recruited a median of 76 days (IQR 29–146) after intracerebral haemorrhage onset: 268 were assigned to start and 269 (one withdrew) to avoid antiplatelet therapy. Participants were followed for a median of 2·0 years (IQR [1·0– 3·0]; completeness 99·3%). 12 (4%) of 268 participants allocated to antiplatelet therapy had recurrence of intracerebral haemorrhage compared with 23 (9%) of 268 participants allocated to avoid antiplatelet therapy (adjusted hazard ratio 0·51 [95% CI 0·25–1·03]; p=0·060). 18 (7%) participants allocated to antiplatelet therapy experienced major haemorrhagic events compared with 25 (9%) participants allocated to avoid antiplatelet therapy (0·71 [0·39–1·30]; p=0·27), and 39 [15%] participants allocated to antiplatelet therapy had major occlusive vascular events compared with 38 [14%] allocated to avoid antiplatelet therapy (1·02 [0·65–1·60]; p=0·92).

Take home message

- Komplex management!
- Egyes hatóanyagok ismerete
- TAG 5-7 nap, VKA 20-60 óra, NOAC 36-72 óra- vesefunkció!
- Gyors felfüggesztés Beriplex, fibrinogén, thrombocyta+desmopresszin
- Visszaadás stabil kontroll képalkotók esetén

Köszönöm a figyelmet!

„There are lies, damn lies – and statistics”