Anaphylaxie; Quand l'épinéphrine ne fonctionne pas

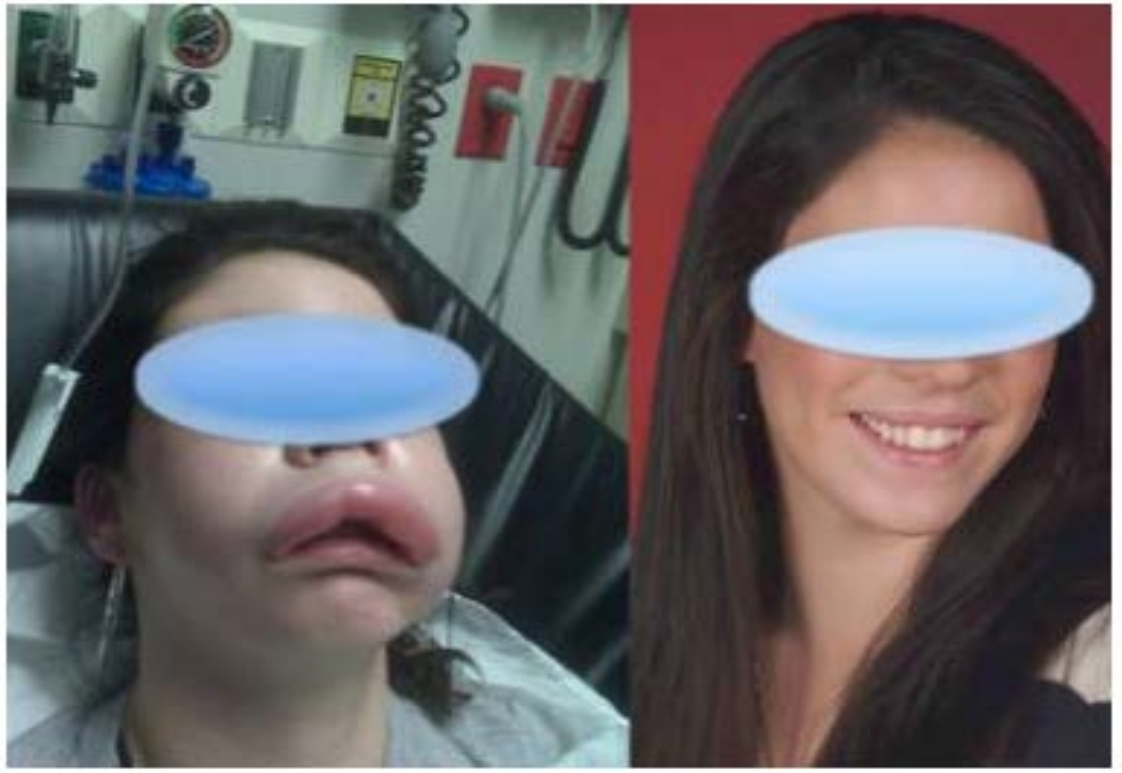
Juliane Guay R2

Cas clinique

- ♀, 19 ans
- Appendicite micro-perforée
- ØATCD med, Ø RX, Habitudes – x 3, Ø allergie
- ATCD chir; Laparotomie exploratrice à 10ans
- SV à l'urgence:
- TA : 85/62 FC : 102 FR : 19 sat : 99% AA T 38.9

Cas clinique

- Tazc
- App
- Pas
  aller
- SV:
- Ø Al



ons

Plan

- Anaphylaxie
  - Critères diagnostic
  - Diagnostic différentiel
  - Traitement
- Angioedème congénital
  - Physiopathologie
  - Facteurs précipitants
  - Diagnostic
  - Traitement
- Autres causes d'angioedème
- Conclusion: Retour sur le cas clinique et les considérations anesthésiques

Anaphylaxie

Anaphylaxis is highly likely when any 1 of the following 3 criteria is fulfilled following exposure to an allergen:	
1	**Acute onset of an illness** (minutes to several hours) **with involvement of the skin, mucosal tissue, or both** (e.g., generalized hives, pruritus or flushing, swollen lips-tongue-uvula) **and at least 1 of the following:** a. **Respiratory compromise** (e.g. dyspnea, wheeze, bronchospasm, stridor, reduced PEF, hypoxemia) b. **Reduced BP** or associated symptoms of end-organ dysfunction (e.g. hypotonia [collapse], syncope, incontinence)
2	**2 or more of the following that occur rapidly after exposure to a *likely* allergen for that patient** (minutes to several hours): a. **Involvement of the skin-mucosal tissue** (e.g., generalized hives, itch-flush, swollen lips-tongue-uvula) b. **Respiratory compromise** (e.g., dyspnea, wheeze, bronchospasm, stridor, reduced PEF, hypoxemia) c. **Reduced BP** or associated symptoms (e.g., hypotonia [collapse], syncope, incontinence) d. **Persistent GI symptoms** (e.g., painful abdominal cramps, vomiting)
3	**Reduced BP after exposure to a *known* allergen for that patient** (minutes to several hours): a. **Infants and children:** low systolic BP (age specific) or > 30% decrease in systolic BP* b. **Adults:** systolic BP < 90 mmHg or > 30% decrease from that person's baseline

Anaphylaxie

Therapy	Indication	Dosage	Goals
AIRWAY OR CUTANEOUS REACTIONS			
Epinephrine	Bronchospasm, laryngeal edema, hypotension, urticaria, angioedema	0.3–0.5 mL of 1:1,000 solution IM, every 10 minutes as needed	Maintain airway patency, reduce fluid extravasation
Oxygen	Hypoxemia	Up to 100%	Maintain SaO_2 > 90%
Albuterol	Bronchospasm	0.5 mL of 0.5% solution in 2.5 mL of isotonic saline by nebulizer, or two puffs by metered-dose inhaler every 15 minutes, up to three doses	Maintain airway patency
Diphenhydramine	Urticaria	1 to 2 mg/kg or 25–50 mg parenterally	Reduce pruritis and antagonize histamine effects
Methylprednisolone	Bronchospasm	125 mg IV every 6 hours	Reduce late-phase reactions
CARDIOVASCULAR REACTIONS			
Epinephrine	Hypotension	1:10,000 solution given IV at 1 µg/min initially, then 2–10 µg/min	Maintain systolic blood pressure > 90 mm Hg
Intravenous fluids	Hypotension	1 L of isotonic saline (0.9% normal saline) every 20–30 minutes as needed	Maintain systolic blood pressure > 90 mm Hg
Ranitidine[4, 12]	Hypotension	50 mg in 20 mL D_5W infused over 10–15 minutes	Can be used in addition to epinephrine and IV fluids to maintain systolic blood pressure > 90 mm Hg
Secondary Therapy			
Norepinephrine	Hypotension	4 mg in 1 L D_5W at 2–12 µg/min	Maintain systolic blood pressure > 90 mm Hg
Glucagon	Refractory hypotension	1 mg in 1 L D_5W at 5–15 µg/min	Increase heart rate and cardiac output

Anaphylaxie

VIA MEDICA

ORIGINAL AND CLINICAL ARTICLES

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Anaphylaxis and cardiac surgery for hypertrophic obstructive cardiomyopathy: a case report and review of anaesthetic management

Kevin Fu Hong Yee, Marcin Wąsowicz

University Health Network, Toronto General Hospital, Canada

Anaphylaxie

Vasopressin for treatment of shock following aprotinin administration

[Traitement à la vasopressine pour un choc suivant l'administration d'aprotinine]

Stephan R. Williams MD PhD,* André Y. Denault MD FRCPC,* Michel Pellerin MD FRCSC,†
Raymond Martineau MD FRCPC*

Case Report

The Pivotal Role of Vasopressin in Refractory Anaphylactic Shock

Claudia Schummer, MD*

Melanie Wirsing, MD†

Wolfram Schummer, MD, DEAA, EDIC‡

BACKGROUND: Severe anaphylaxis can be associated with cardiovascular collapse that is difficult to manage and does not respond to treatment with epinephrine. Because anaphylaxis is uncommon, unpredictable and may be fatal, a prospective, randomized, controlled trial in humans on the best management is difficult and guidelines are based on theory and anecdotes only.
METHODS AND RESULTS: We report six cases in which the use of vasopressin was successful in the treatment of anaphylactic shock.
CONCLUSIONS: Standard treatment of anaphylactic shock, including discontinuation of the causative agent, administration of epinephrine, and infusion of IV fluids, did not stabilize cardiocirculatory function, and adding arginine vasopressors resulted in prompt hemodynamic stabilization.
(Anesth Analg 2008;107:620–4)

Cas clinique

- ABC
- Monitoring
- O2
- Accès veineux
- Canule artérielle
- 1L NS flush
- Épinéphrine 50 mcg iv x 2 puis 2mcg/min
- Benadryl 50mg iv
- Solumedrol 125mg iv
- Zantac 50mg iv
- Préparer matériel pour fibre optique
- Préparer matériel pour airway chirurgical
- Faire signaler l'ORL
- Ø stridor , Ø bronchospasme, progression angioedème

Cas clinique

- Topicalisation du airway
- Intubation FOB éveillé
- Stable HD, pressions de ventilation N

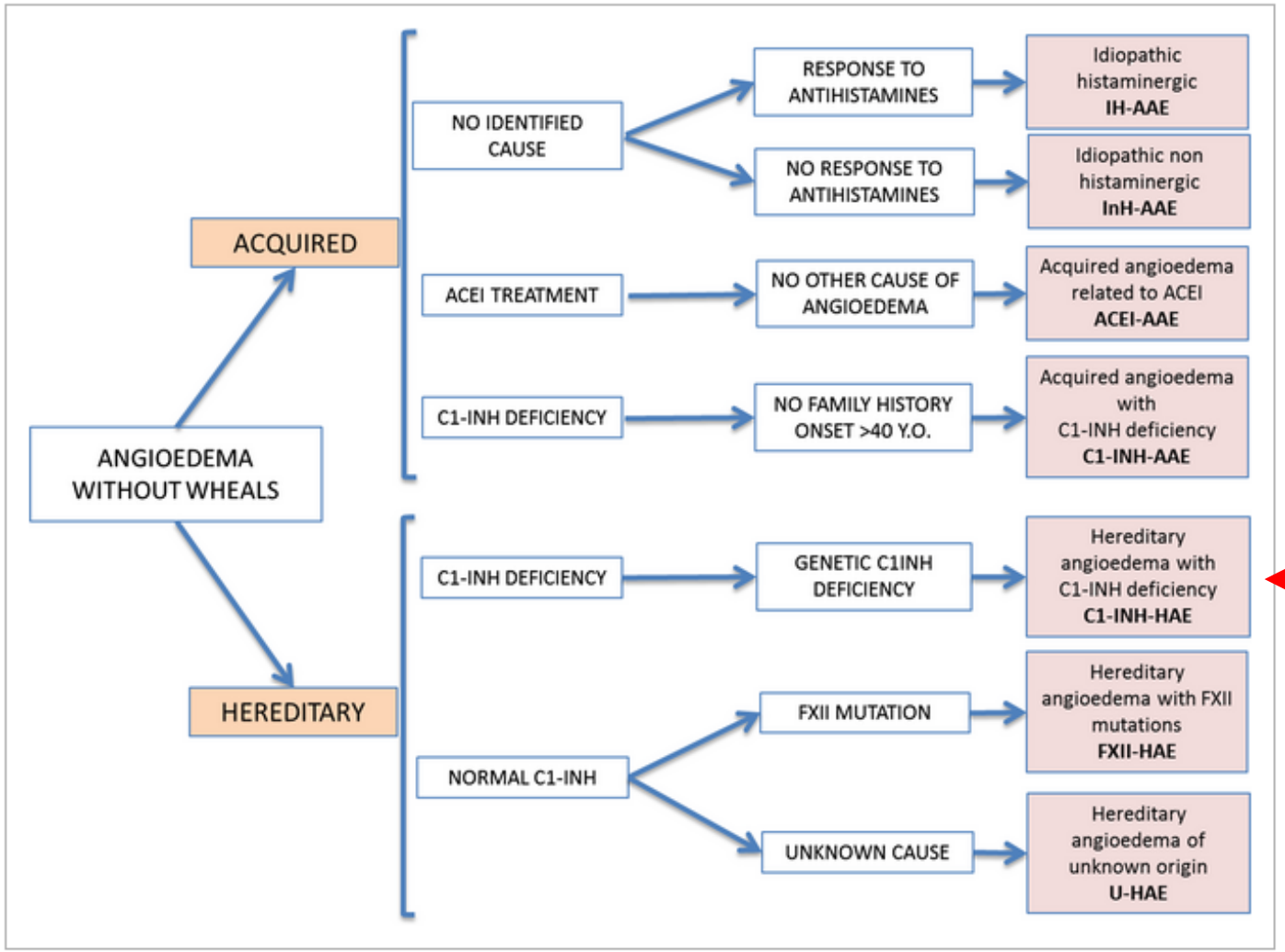
Cas clinique

- Éléments en défaveur de l'anaphylaxie;
  - Progression de l'angioedème malgré Tx adéquat
  - Autre cause pour expliquer l'instabilité HD
  - HypoTA et tachycardie avant Tazo
  - Angioedème disproportionné par rapport à l'instabilité HD

Anaphylaxie

- Diagnostic différentiel
  - Toutes causes de choc
  - Asthme
  - Corps étranger
  - Hypoglycémie
  - Flushing syndrome; Red-man syndrome (vancomycin), tumeur carcinoide, pheochromocytome
  - Cellulite, épiglottite, abcès rétro-pharyngé
  - Autres causes d'angioedème

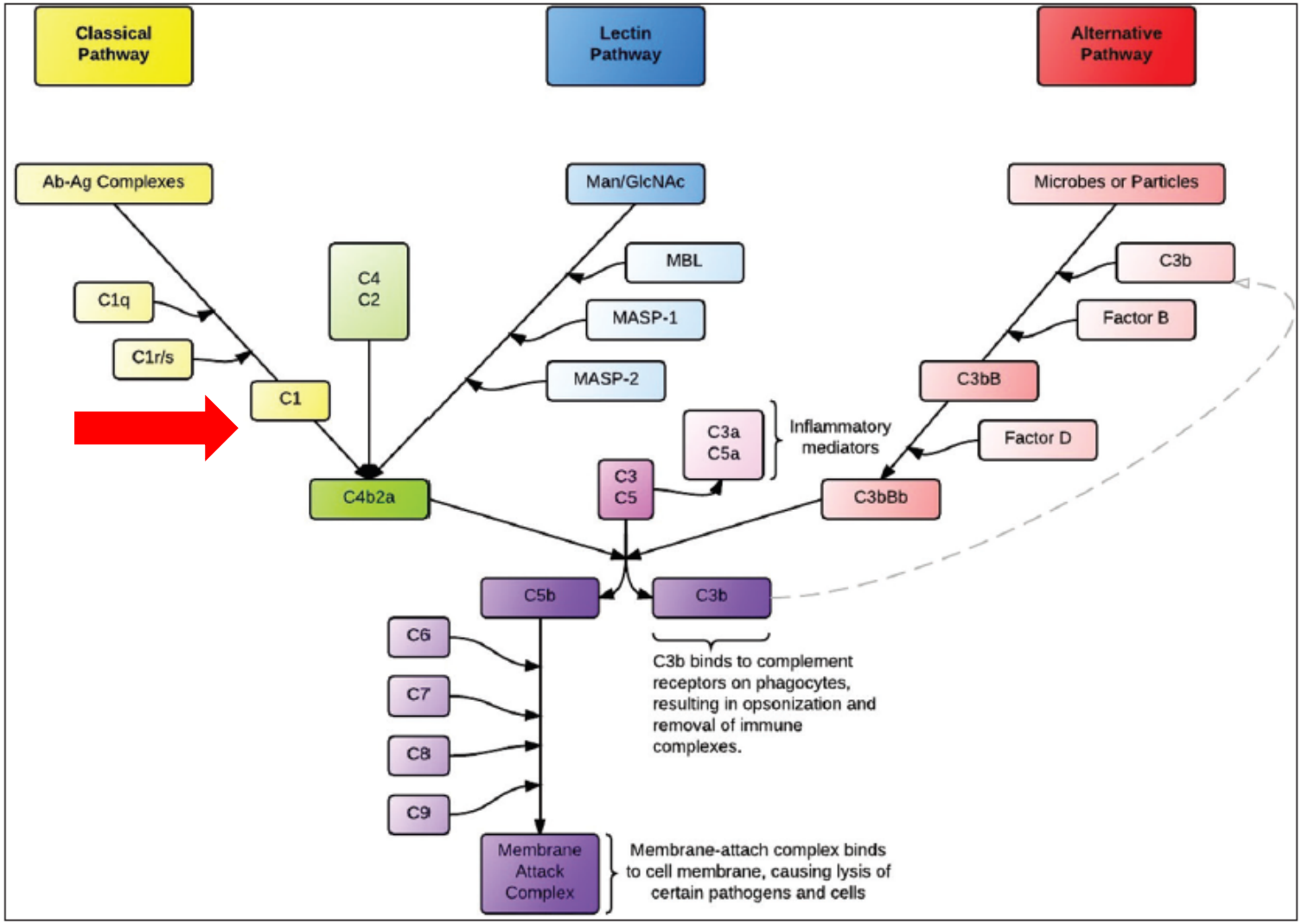
Angioedème


ANGIOEDEMA WITHOUT WHEALS
ACQUIRED
HEREDITARY
NO IDENTIFIED CAUSE
RESPONSE TO ANTIHISTAMINES
Idiopathic histaminergic IH-AAE
NO RESPONSE TO ANTIHISTAMINES
Idiopathic non histaminergic InH-AAE
ACEI TREATMENT
NO OTHER CAUSE OF ANGIOEDEMA
Acquired angioedema related to ACEI ACEI-AAE
C1-INH DEFICIENCY
NO FAMILY HISTORY ONSET >40 Y.O.
Acquired angioedema with C1-INH deficiency C1-INH-AAE
C1-INH DEFICIENCY
GENETIC C1INH DEFICIENCY
Hereditary angioedema with C1-INH deficiency C1-INH-HAE
NORMAL C1-INH
FXII MUTATION
Hereditary angioedema with FXII mutations FXII-HAE
UNKNOWN CAUSE
Hereditary angioedema of unknown origin U-HAE

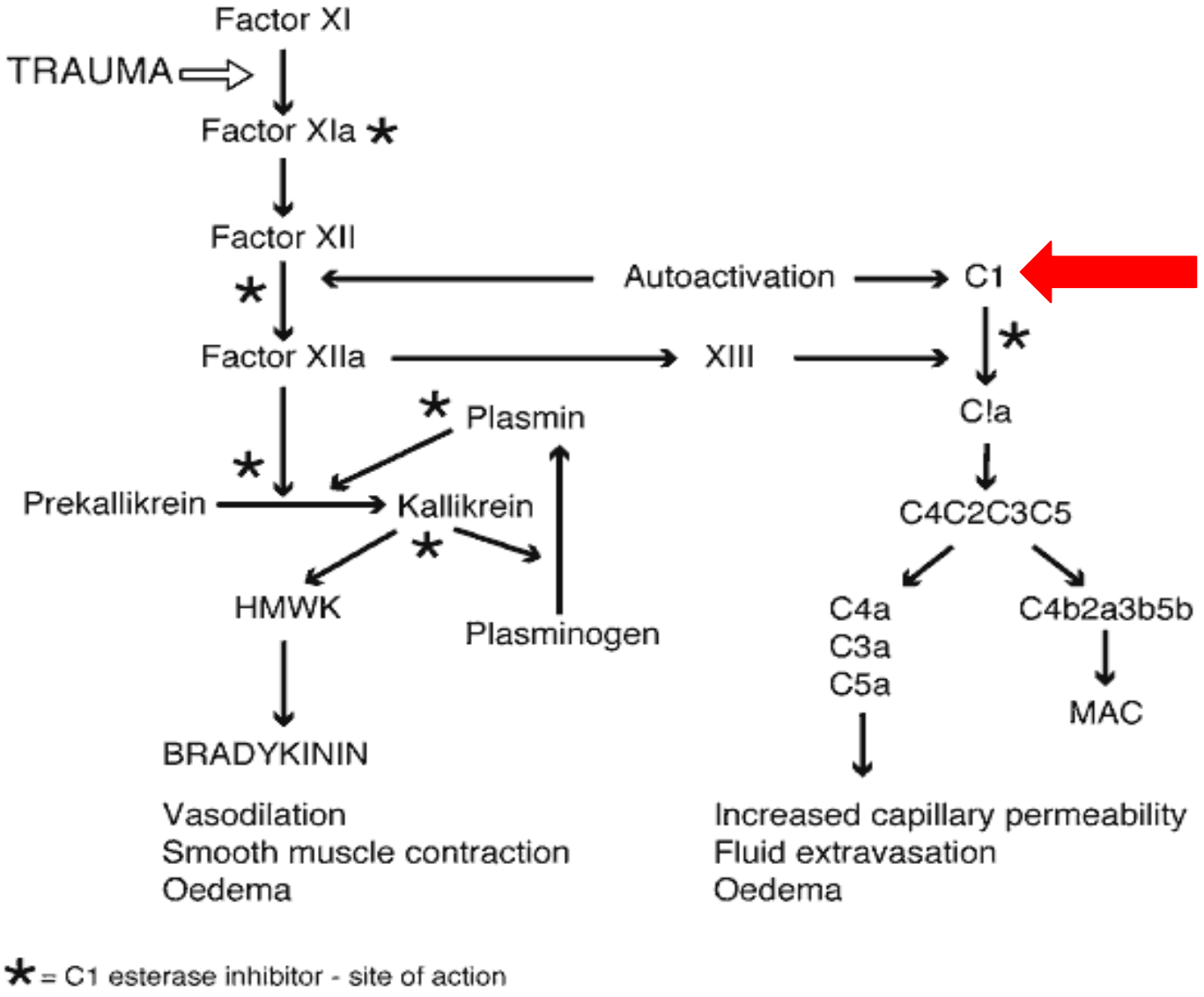
Angioedème congénital

- Prévalence; 1:10 000 a 1:50 000
- Maladie autosomal dominante
  - Mutation sur gène inhibiteur de C1 sur chromosome 11
- Mutation spontannée : 20-25%
- 3 types:
  - 1. Déficit quantitatif en inhibiteur de C1
  - 2. Déficit qualitatif en inhibiteur de C1 (fonction anormale)
  - 3. Mutation facteur XII

Physiopathologie



Physiopathologie



Facteurs précipitants

- Intubation endotrachéale
- Procédures dentaires
- CEC
- Stre
- AIN
- Hor
- Aut
  can

Case Report

Lingual angioedema after alteplase treatment in a patient with acute ischemic stroke

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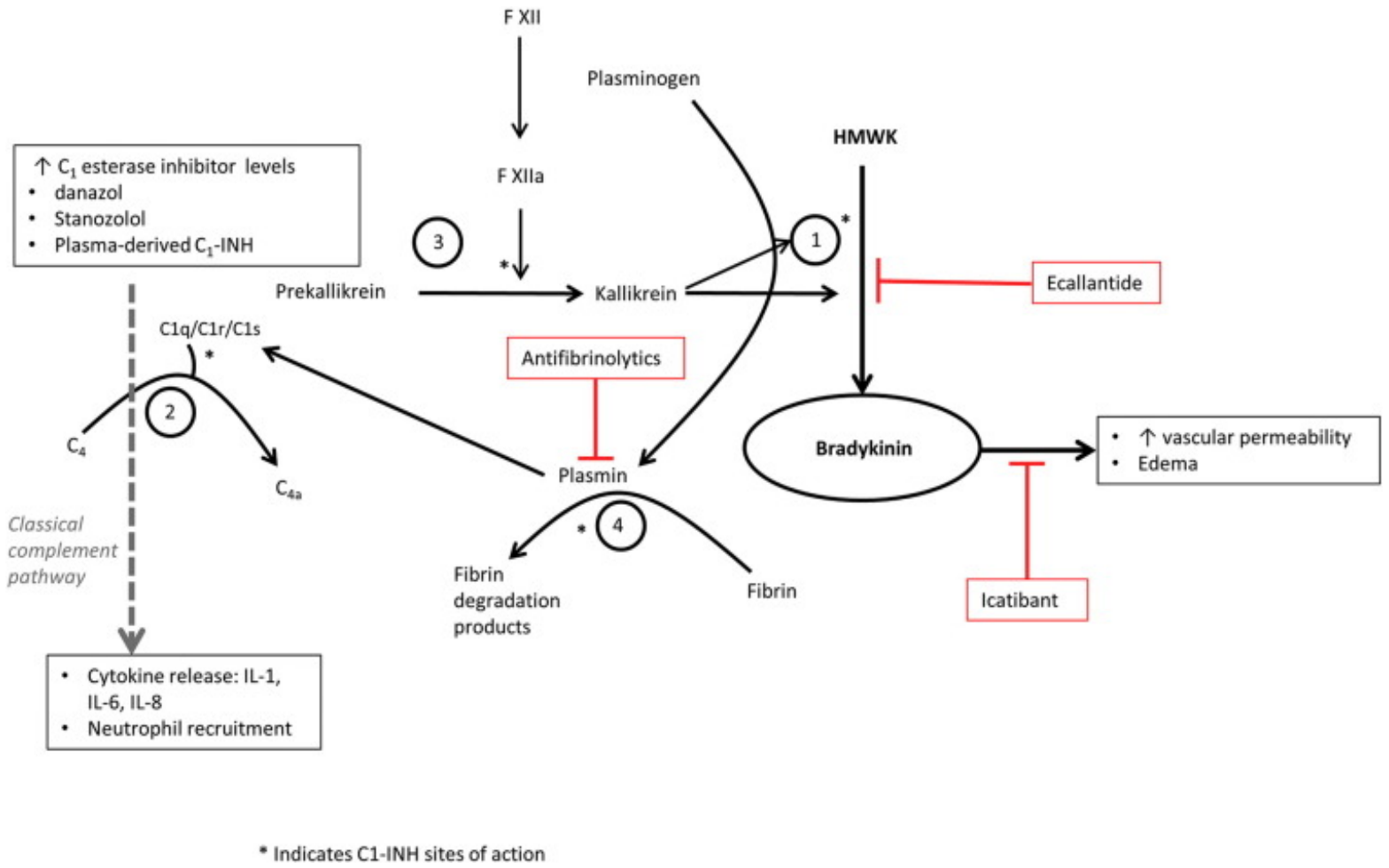
Diagnostic

Clinical criteria
Major
(1) Self-limiting, noninflammatory subcutaneous angioedema without major urticarial rash, often recurrent and often lasting more than 12 hours
(2) Self-remitting abdominal pain without clear organic etiology, often recurrent and often lasting more than 6 hours
(3) Recurrent laryngeal edema
Minor
(4) Family history of recurrent angioedema and/or abdominal pain and/or laryngeal edema
Laboratory criteria
(1) C1 inhibitor antigenic levels <50% of normal at 2 separate determinations with patient in basal condition and after the first year of age
(2) C1 inhibitor functional levels <50% of normal at 2 separate determinations with patient in basal condition and after the first year of age
(3) Mutation in C1 inhibitor gene altering protein synthesis and/or function
Diagnosis can be established in presence of 1 major (1-3) clinical criterion and 1 laboratory criterion

Traitements



Traitement

Angioedema pattern	Clinical description	Management	Drugs	Monitoring
Type 1	Limited to face and/or anterior tongue	Observe on the ward after fiberoptic laryngeal examination via nose or mouth. If oedema limited just to face/lips-after medications consider discharge home.	Discontinue exposure to any possible factor/drug; oral antihistamines	Continuous pulse oximetry. Repeated evaluation of the patient looking for voice change, hoarseness, stridor, and dyspnoea.
Type 2	Extends to base of tongue or floor of the mouth, tongue, soft palate, and/or uvula.	Close observation in the ICU with serial fiberoptic laryngeal examinations via nose or mouth; tracheostomy tray by the bedside	As above; IV steroids and H_1 and H_2 antagonist or subcutaneous epinephrine injections	Continuous pulse oximetry. Repeated evaluation of the patient looking for voice change, hoarseness, stridor, and dyspnoea. If tongue size diminished during/shortly after IV steroids-oedema unlikely to progress.
Type 3	Extends to supraglottic and glottic structures	Proceed immediately to videolarngoscopic or alternative methods of intubation. Use difficult airway algorithms. Avoid sedation before intubation.	subcutaneous epinephrine injections and IV steroids and H_1 and H_2 antagonist	Continuous pulse oximetry. ABGs to asses oxygenation and ventilation

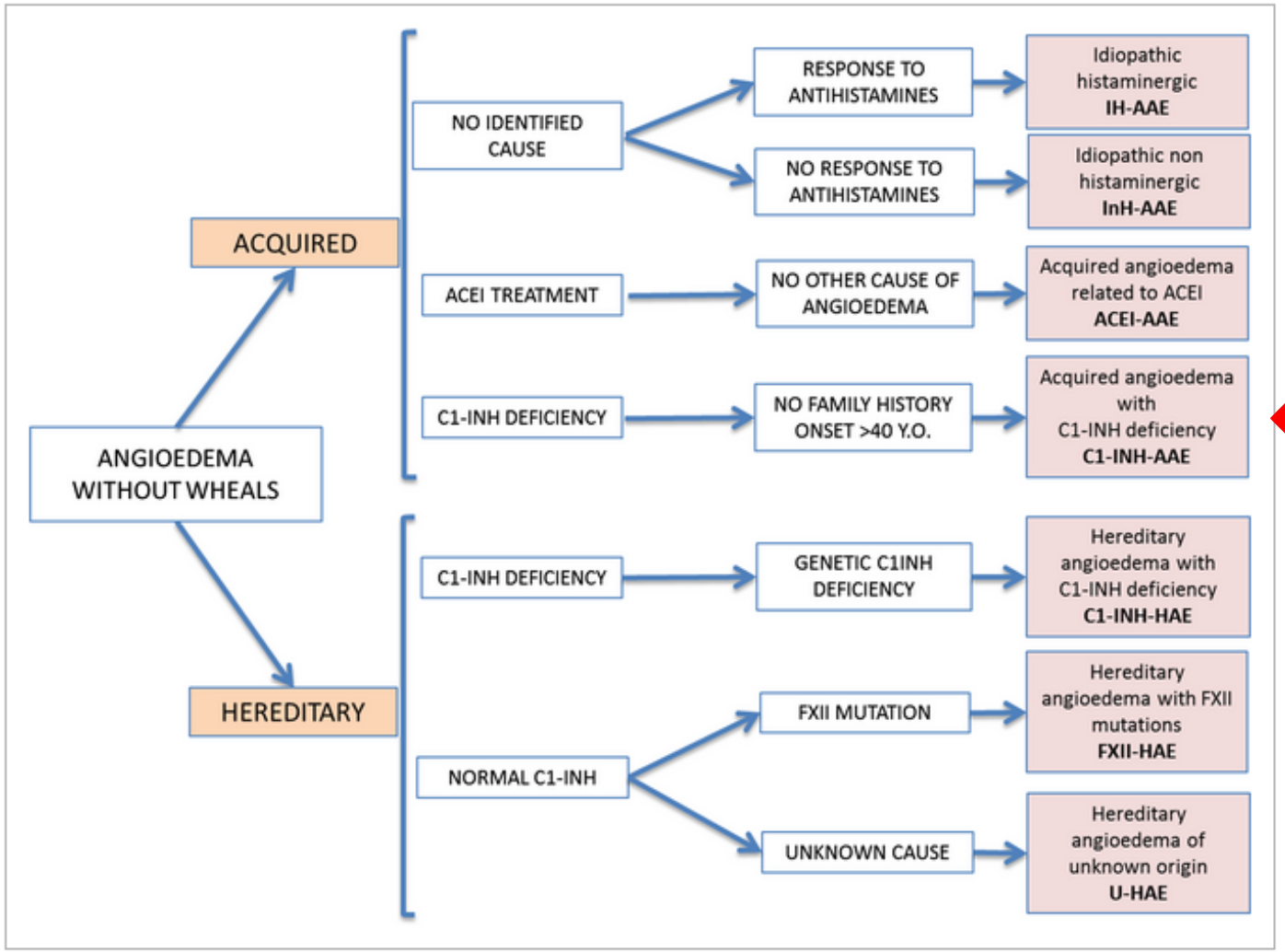
- Risque de décès 25-40% pour crise laryngée
- Série 342 cas angioedème connu; 4 intubations, 2 cricothyrotomies

- Nouveaux traitements:
  - Ecallantide (Kalbitor): Inhibiteur de la kallikrein. Diminution de la sévérité des symptômes. Dose: 30mg s/c.
  - Icatibant (Firazyr) : Inhibiteur du récepteur B2 de la bradykinine. Approuvé pour traitement aigu. Dose: 30mg s/c

Prophylaxie

- Concentrés d'inhibiteurs de C1: 1h pré procédure
- Danazol: 2,5 a 10mg/kg (max 600mg) donné 5 jours pré procédure
- PFC : 2 unités 1h pré procédure
- Anti-fibrinolytique
- Anesthésie régionale à favoriser
- Risque de développer un angioedème post chirurgie :
  - 30% sans prophylaxie
  - 15% avec 500 unités de concentré d'inhibiteur de C1
  - 5% avec 1000 unités de concentré d'inhibiteur de C1
- Série de 24 patients, 38 AG avec prophylaxie; un seul a développé oedème laryngé nécessitant réintubation

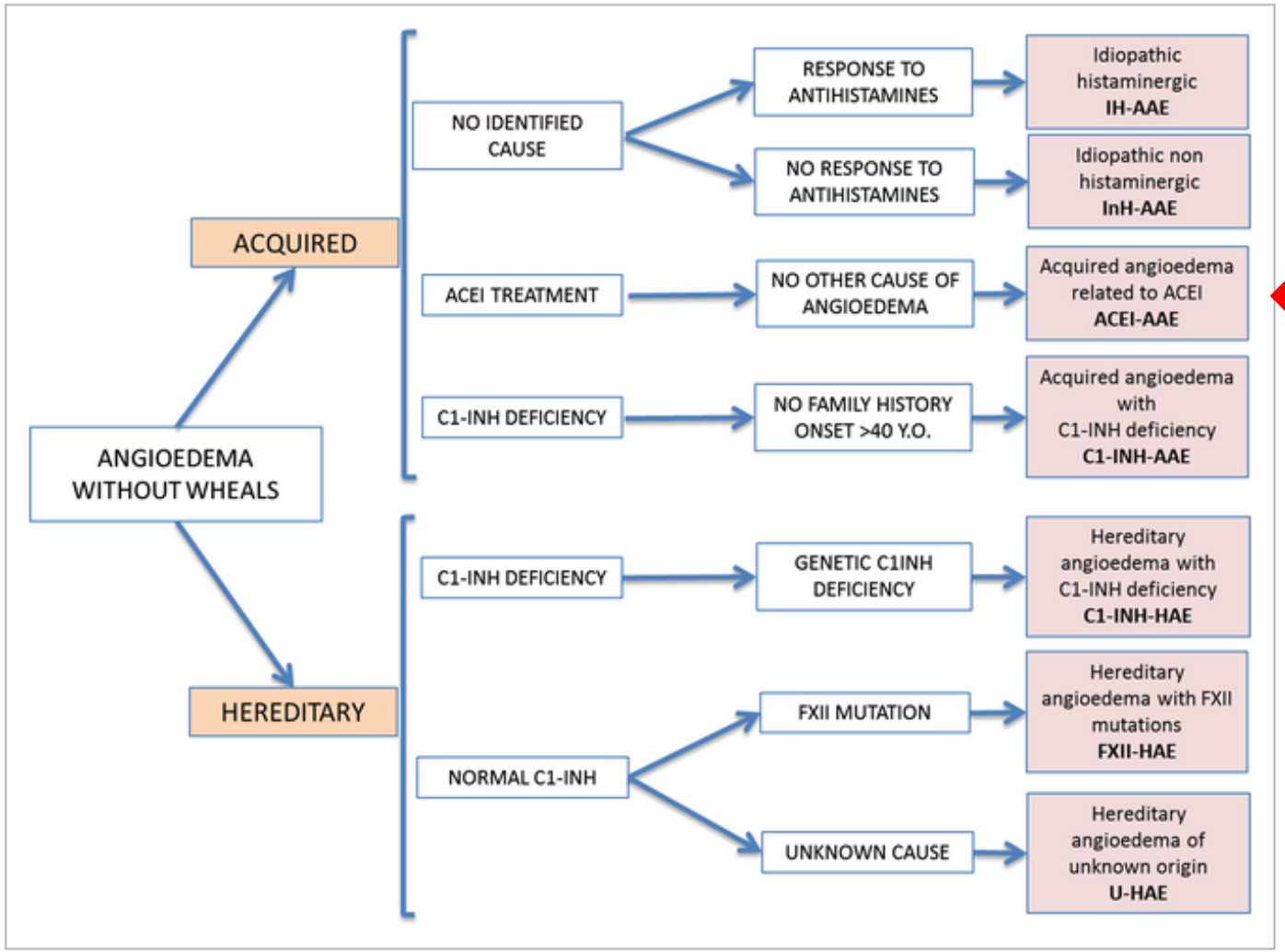
Autres angioedèmes



Autres angioedèmes

- Angioedème acquis :
  - Pas d'histoire familiale
  - Début tardif
  - Secondaire à un déficit en inhibiteur de C1.
- Causes :
  - Maladies auto-immune ou lymphoprolifératives; lymphome, leucémie lymphoïde chronique, myélome multiple, myélofibrose, macroglobulinémie de Waldenström, gammapathie monoclonale, carcinome sein et GI, lupus, Churg-Strauss, hépatite B, VIH, echinococcose.

Autres angioedèmes



Autres angioedèmes

- Angioedème secondaire aux IECA :
  - 1er mois suivant le début du traitement
  - Race noire
  - Mécanisme : Bradykinine
  - Tx: Concentrés d'inhibiteur de C1

Patient Case Report

C1 Esterase Inhibitor (Berinert) for ACE Inhibitor-Induced Angioedema: Two Case Reports

Maria Leibfried, PharmD[1], and Alexandra Kovary, PharmD[2]

Journal of Pharmacy Practice
1-4

DOI: 10.1177/0897190016677427
jpp.sagepub.com
SAGE

Cas clinique

- Appel à la famille; Confirme l'existence d'une histoire familiale positive
- Berinert 20 u/kg
- Appendicectomie s/p
- Extubée lorsque résolution oedème USI
- Positif pour déficit en inhibiteur de C1

Considérations

- Appendicite aigue compliquée
  - Estomac plein
  - Sepsis
- Chirurgie: Reporter ou non
- Angioedème

Conclusion

- Anaphylaxie; Cause la plus fréquente d'angioedème
- Élargir Dx différentiel de l'angioedème
- Prophylaxie et traitement: Berinert
- Vasopressine

Questions ?

- Merci pour votre attention

Bibliographie

- Franzen, D., Ursprung, T., Wuthrich, B. and Reber, A., *Idiopathic non-histaminergic angio-oedema after routine extubation successfully treated with fresh frozen plasma*. Anaesthesia, 61, 2006, 698–701
- Yu S., Callum J., Alam A., *C1-esterase inhibitor for short-term prophylaxis in a patient with hereditary angioedema with normal C1 inhibitor function, Journal of clinical anesthesia*, 35, 2016, 488-491
- Spyridonidou, A., Iatrou, C., Alexoudis, A., Vogiatzaki, T., Polychronidis, A. and Simopoulos, C., *Peri-operative management of a patient with hereditary angioedema undergoing laparoscopic cholecystectomy*. Anaesthesia, 65, 2010, 74–77
- Narayanan A, Date RR, Birur S, Bhakta P, Srinivasan S. Anaesthesia *Management of a Patient with Hereditary Angioedema with Prophylactic Administration of C1 Esterase Inhibitor: Case report and literature review*. Sultan Qaboos University Medical Journal. 13(3), 2013, 467-471.
- Soltanifar D., Afzal S., Harrison S., Sultan P., *Caesarean delivery in a parturient with type III hereditary angioedema*, International journal of obstetric anesthesia, 23(4), 2014, 398-399
- Williams AH, Craig TJ. *Perioperative management for patients with hereditary angioedema*. Allergy & Rhinology. 6(1), 2015, 50-55.
- Macbeth L, Volcheck G, Sprung J, Weingarten T, *Perioperative course in patients with hereditary or acquired angioedema*, Journal of clinical anesthesia, 34, 2016, 385-391
- Aygören-Pürsün E, Martinez Saguer I, Kreuz W, Klingebiel T, Schwabe D. *Risk of angioedema following invasive or surgical procedures in HAE type I and II – the natural history*. Allergy. 2013, 68(8), 1034-1039.
- Bork K, Barnstedt S, *Laryngeal edema and death from asphyxiation after tooth extraction in four patients with hereditary angioedema*, The Journal of the American dental association, 2003, 134(8), 1088-1094
- Bowen T, Cicardi M, Farkas H, et al. *2010 International consensus algorithm for the diagnosis, therapy and management of hereditary angioedema*. Allergy, Asthma, and Clinical Immunology : Official Journal of the Canadian Society of Allergy and Clinical Immunology, 6(1), 2010, 24
- Betschel S, Badiou J, Binkley K, et al. *Canadian hereditary angioedema guideline*. Allergy, Asthma, and Clinical Immunology : Official Journal of the Canadian Society of Allergy and Clinical Immunology, 10(1), 2014, 50
- Agostini A, Aygören-Pürsün E, Binkley K, Blanch A, Bork K, Bouillet L, Bucher C, Castaldo A, *Hereditary and acquired angioedema: Problems and progress: Proceedings of the third C1 esterase inhibitor deficiency workshop and beyond*, Journal of allergy and clinical immunology, 114(3), 2004, 51-131
- Eidelman FJ. *Hereditary angioedema: New therapeutic options for a potentially deadly disorder*. BMC Blood Disorders, 10, 2010, 3
- Cicardi M, Aberer W, Banerji A, Bas M, Bernstein JA, Bork K, Caballero T, Farkas H, Grumach A, Kaplan AP, Riedl MA, Triggiani M, Zanichelli A, Zuraw B, *Classification, diagnosis, and approach to treatment for angioedema: consensus report from the Hereditary Angioedema International Working Group*. Allergy 2014; 69: 602–616.
- Li H, Kaliner M, *Angioedema,* Medscape, 2016
- Bork K, Hardt J, Staubach-Renz P, Witzke G, *Risk of laryngeal edema and facial swellings after tooth extraction in patients with hereditary angioedema with and without prophylaxis with C1 inhibitor concentrate: a retrospective study*, Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology, 112(1), 2011, 58-64
- Craig T, Levy R, Wasserman R, Bewtra A, Hurewitz D, Obtulowicz K, Reshef A, Ritchie B, *Efficacy of human C1 esterase inhibitor concentrate compared with placebo in acute hereditary angioedema attacks*, Journal of Allergy and Clinical Immunology, 124(4), 2009, 801-808
- Yee KF, Wasowicz M, *Anaphylaxis and cardiac surgery for hypertrophic obstructive cardiomyopathy: a case report and review of anaesthetic management,* Anesthesiology intensive therapy, 48(4), 2016, 252-256