

Embolie plodovou vodou

Amniotic Fluid Embolism (AFE)

Doc. MUDr. Antonín Pařízek, CSc.

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MUDr. Vratislav Krejčí

2014 - 2016

 10 years review of atypical amniotic	28.10.2016 22:04	Adobe Acrobat D...	357 kB
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cca_2001

Proposed diagnostic criteria for the case definition of amniotic fluid embolism in research studies



Steven L. Clark; Roberto Romero; Gary A. Dildy; William M. Callaghan; Richard M. Smiley; Arthur W. Bracey; Gary D. Hankins; Mary E. D'Alton; Mike Foley; Luis D. Pacheco; Rakesh B. Vadhera; J. Patrick Herlihy; Richard L. Berkowitz; Michael A. Belfort



SMFM Clinical Guidelines No. 9:

smfm.org

Amniotic fluid embolism: diagnosis and management



Society for Maternal-Fetal Medicine (SMFM) with the assistance of Luis D. Pacheco, MD; George Saade, MD; Gary D. V. Hankins, MD; Steven L. Clark, MD

OBSTETRICS

Amniotic fluid embolism: an evidence-based review

Agustín Conde-Agudelo, MD, MPH; Roberto Romero, MD

Amniotic fluid embolism (AFE) is a rare and often fatal obstetric condition, characterized by sudden cardiovascular collapse, altered mental status, and disseminated intravascular coagulation (DIC). The presence of fetal debris in the pulmonary blood vessels of a mother who had died suddenly in labor was first described by Meyer¹ in 1926, but it was not until 1941 that death following sudden collapse during labor or in the immediate postpartum period was attributed to AFE.²

Understanding of the pathogenesis of AFE has increased in the last 2 decades since this disorder was recognized as 1 of the main causes of maternal mortality in the United States.³ Evidence on AFE,

We conducted an evidence-based review of information about amniotic fluid embolism (AFE). The estimated incidence of AFE is 1:15,200 and 1:53,800 deliveries in North America and Europe, respectively. The case fatality rate and perinatal mortality associated with AFE are 13-30% and 9-44%, respectively. Risk factors associated with an increased risk of AFE include advanced maternal age, placental abnormalities, operative deliveries, eclampsia, polyhydramnios, cervical lacerations, and uterine rupture. The hemodynamic response in AFE is biphasic, with initial pulmonary hypertension and right ventricular failure, followed by left ventricular failure. Promising therapies include selective pulmonary vasodilators and recombinant activated factor VIIa. Important topics for future research are presented.

Key words: amniotic fluid embolism, cardiovascular collapse, DIC, disseminated intravascular coagulation, maternal death, maternal morbidity, maternal mortality, perinatal mortality, pregnancy

Cite this article as: Conde-Agudelo A, Romero R. Amniotic fluid embolism: an evidence-based review. *Am J Obstet Gynecol* 2009;201:445.e1-13.

From appearance to essence: 10 years review of atypical amniotic fluid embolism

Fangrong Shen^{1,2} · Lu Wang¹ · Weiwen Yang¹ · Youguo Chen¹

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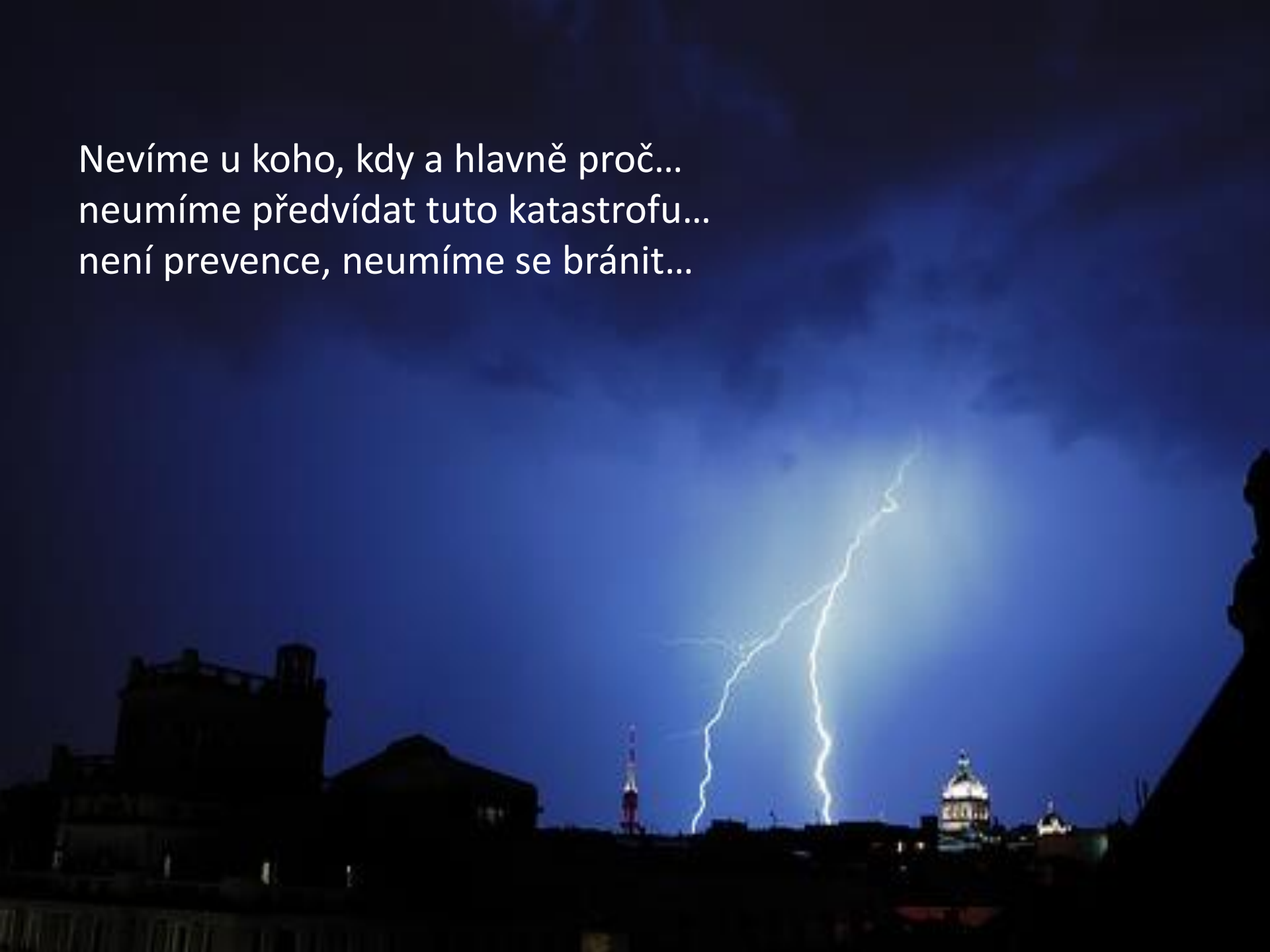
Abstract

Purpose Amniotic fluid embolism (AFE) is an unpredictable and unpreventable complication of maternity. The presentation may range from relatively subtle clinical events to sudden maternal cardiac arrest. However, the neglected diagnosis of non-classical form of AFE (atypical AFE) is very common. The aim of this study was to

obstetric hemorrhage and/or pulmonary and renal dysfunction postpartum. Hyperfibrinolysis and coagulopathy may be the early laboratory findings of atypical AFE. Atypical and classical AFE shared the same risks, such as advanced maternal age, placental abnormalities, operative deliveries, eclampsia, cervical lacerations, and induction of labor.

Conclusion Staying alert to premonitory symptoms of

Nevíme u koho, kdy a hlavně proč...
neumíme předvídat tuto katastrofu...
není prevence, neumíme se bránit...



Embolie plodovou vodou

USA, Francie

3. nejčastější příčina úmrtí těhotných

UK

2. nejčastější příčina úmrtí těhotných

Singapore, Austrálie

nejčastější příčina úmrtí těhotných

*Amniotic fluid embolism: an evidence-based review.
Conde-Agudelo A, Romero R. Am J Obstet Gynecol. 2009
Nov;201(5):445.e1-13.*

Embolie plodovou vodou

Amniotic Fluid Embolism Register **(USA)**

The United Kingdom Amniotic Fluid Embolism Register **(UK)**

Později

Amniotic Fluid Embolism Register **(Kanada)**

Amniotic Fluid Embolism Register **(Austrálie)**

Amniotic Fluid Embolism Register **(Japonsko)**

TABLE 1

International criteria for diagnosis of amniotic fluid embolism

United Kingdom:

Clinical diagnosis of AFE (acute hypotension or cardiac arrest, acute hypoxia, or coagulopathy in absence of any other potential explanation for signs and symptoms observed) OR pathologic diagnosis of fetal squames or hair in lungs.¹⁴

Australia:

Clinical diagnosis of AFE (acute hypotension or cardiac arrest, acute hypoxia, or coagulopathy in absence of any other potential explanation for signs and symptoms observed) OR pathologic/postmortem diagnosis (presence of fetal squames/debris in pulmonary circulation).¹⁵

Japan:

1. Symptoms appeared during pregnancy or within 12 h of delivery;
2. Intensive medical intervention was conducted to treat ≥ 1 of following symptoms/diseases: (a) cardiac arrest, (b) severe bleeding of unknown origin within 2 h of delivery (≥ 1500 mL), (c) DIC, or (d) respiratory failure; and
3. If findings or symptoms obtained could not be explained by other diseases. Consumptive coagulopathy/DIC due to evident etiologies such as abnormal placentation, trauma during labor, and severe preeclampsia/eclampsia should be excluded.

Uterine AFE was considered to have occurred when fetal debris and amniotic fluid components were found in uterus in pathological examination of cases of severe uterine hemorrhage after placental removal (eg, atonic bleeding) in absence of other obstetric hemorrhagic complications such as abnormal placentation, trauma during labor and delivery, and severe preeclampsia/eclampsia.¹⁶

AFE, amniotic fluid embolism; *DIC*, disseminated intravascular coagulation.

Clark. Case definition of amniotic fluid embolism to improve quality of clinical and translational research. Am J Obstet Gynecol 2016.

Incidence

UK	1,9/100 000 > 24. týden
Holandsko	2,5/100 000 > 24. týden
USA	5,5/100 000
Kanada	6,0/100 000 > 20. týden
Austrálie	6,1/100 000 > 20. týden

Chybí případy, které neskočily smrtí...

*Knight M, Berg C, Brocklehurst P, Kramer M, Lewis G, Oats J, Roberts CL, Spong C,
Sullivan E, van Roosmalen J, Zwart J.*

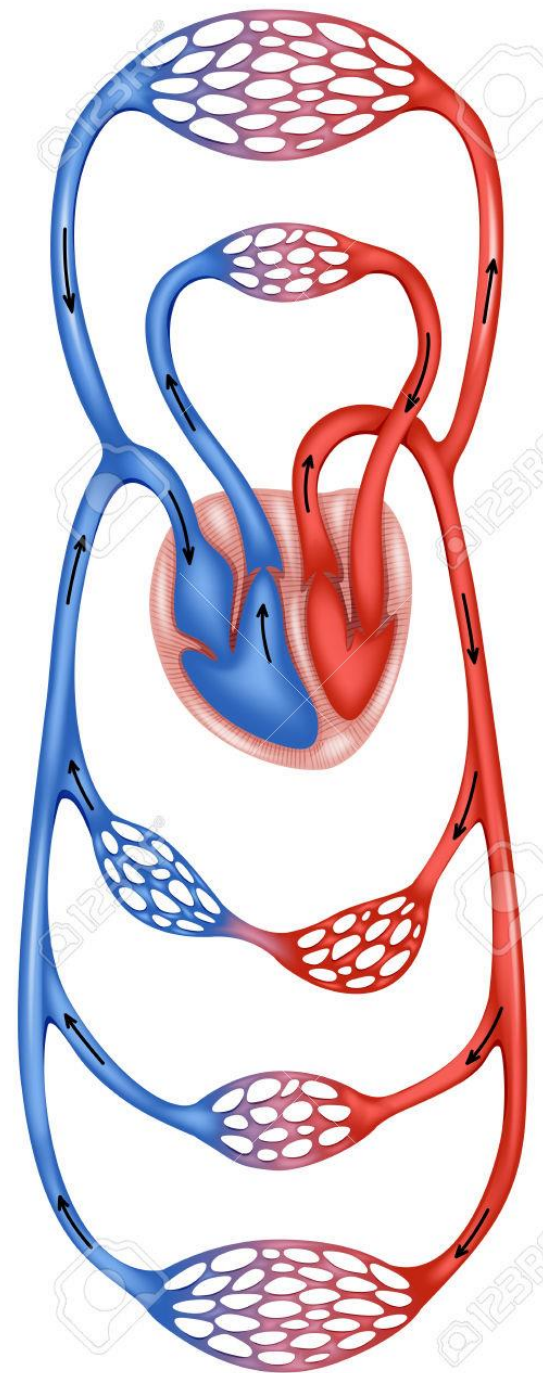
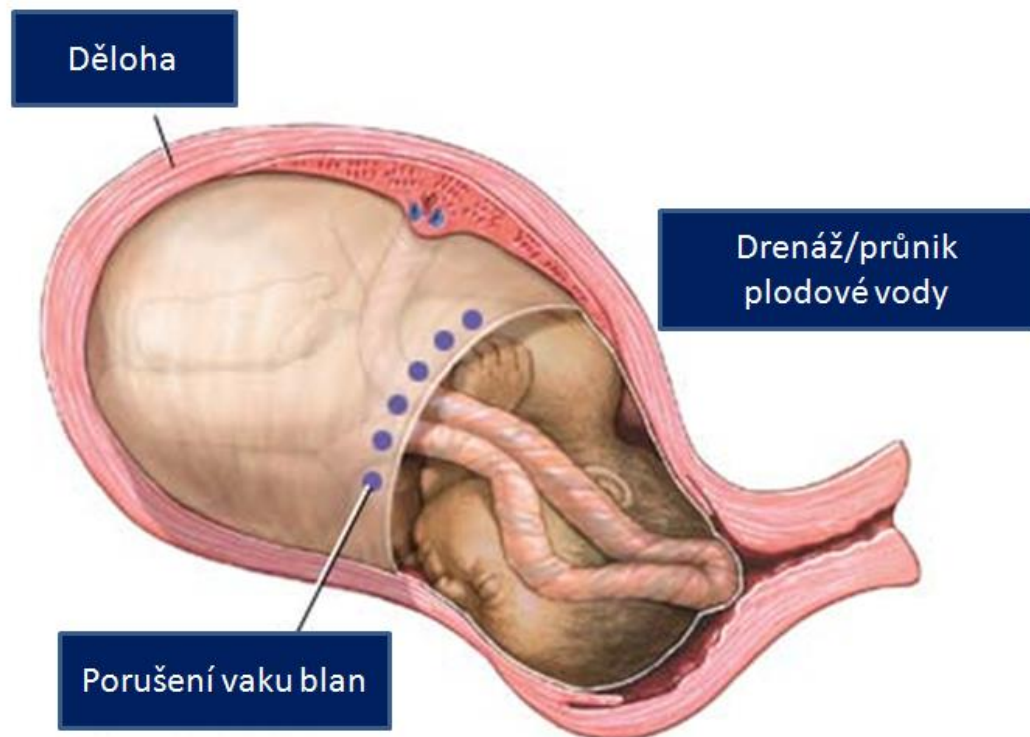
*Amniotic fluid embolism incidence, risk factors and outcomes: a review and
recommendations.*

BMC Pregnancy Childbirth. 2012 Feb 10;12:7.

Mortalita matek

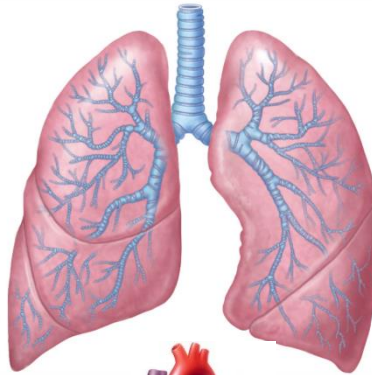
UK	19% (11-30%)
Holandsko	11% (3-45%)
USA	21% (18-22%)
Kanada	13% (9-19%)
Austrálie	14% (9-19%)

*Knight M, Berg C, Brocklehurst P, Kramer M, Lewis G, Oats J, Roberts CL, Spong C, Sullivan E, van Roosmalen J, Zwart J.
Amniotic fluid embolism incidence, risk factors and outcomes: a review and recommendations.
BMC Pregnancy Childbirth. 2012 Feb 10;12:7.*





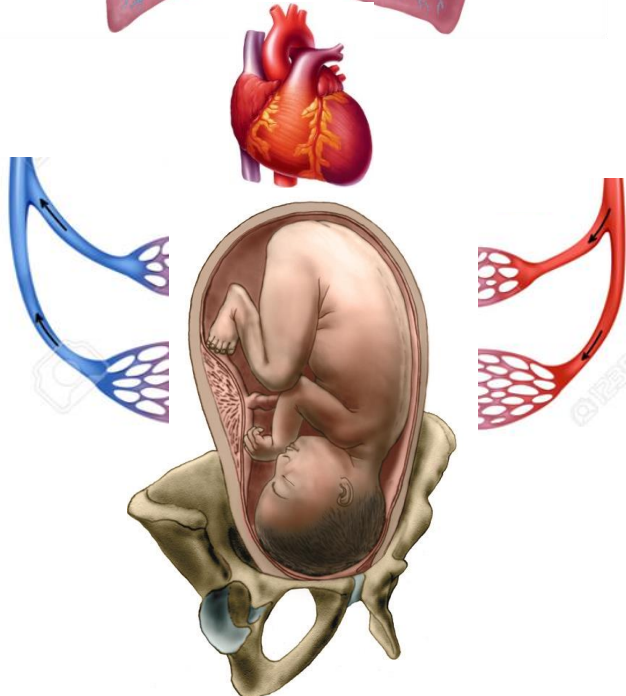
Dušnost



Plodová voda přechází
do plicní cirkulace

Působky do velké oběhu

Plodová voda přechází
do mateřského oběhu



Pokles perfúze
Hypoxie plodu

Embolie plodovou vodou - fáze

1. fáze

- spasmus plicních cév
- porucha plicní ventilace/perfúze
- selhávání pravého srdce
- hypoxie a hypotenze matky

po cca 30 min.

2. fáze

- selhávání levého srdce
- plicní edém

3. DIC

- srdeční zástava matky → přežití nepravděpodobné

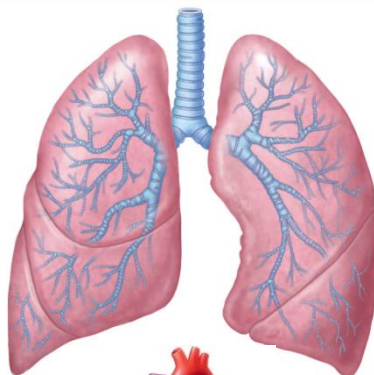
Trvalý neurologický
deficit



Hypoxie mozku
Křeče

Dušnost
Cyanóza

Plicní edém
- kardiogenní
- non-kardiogenní
Bronchokonstrikce
Bronchospasmus
Desaturace

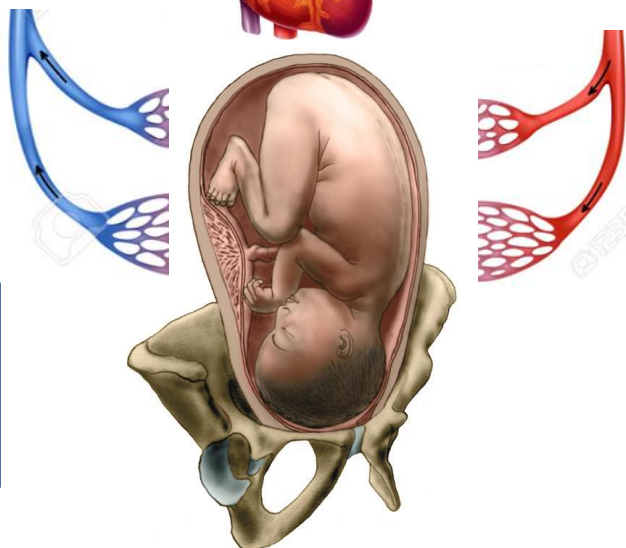


Snížená perfúze srdce
Snížení inotropie
Snížený srdeční výdej
Plicní kongesce

Hypotenze, hypoperúze
Šok

DIC
Krvácení

Snížení perfúze dělohy
Hypotonie/atonie
PŽOK



Hypoxie plodu
Bradykardie
Smrt dítěte

Kritéria pro diagnostiku AFE

A. Mrtví = pitevní nález (histochemicko-patologické vyšetření)
Přeživší = klinické projevy

B. Klinická manifestace:

1. Příznaky

- srdeční zástava (akutní hypoxie a hypotenze)
- zástava dýchání (dyspnoe)
- konzumpční koagulopatie (DIC)

2. Nástup klinické manifestace:

- těhotenství
- spontánní porod
- operační porod císařský řez (do 12 hodin od porodu)

3. Absence jiné nemoci/patologie,

která by mohla vysvětlit popsané klinické příznaky



6:46
11. 5. 2001

Diagnostika....obtížná u přeživších

Chybí: jednoduchý, neinvazivní, specifický a senzitivní test

- zinek kopropofyrin (sérum) ...produkt mekónia
- tryptáza (sérum) ...degranulace mastocytů
- insulin-like growth factor binding protein-1
- IL 8
- Sialyl-Tn
- squamózní buňky v centrální venózní krvi
- squamózní buňky v a. pulmonalis
- zbytky částic amniové tekutiny a. pulmonalis
- zbytky částic amniové tekutiny v bronchiálním sekretu (bronchoalveolární laváž)

Table 5. Investigational biomarkers for amniotic fluid embolism

New biomarkers

Activin A (transforming growth factor)	Characteristic of maternal serum, cord blood serum, and amniotic fluid Increased with gestational age
Proopiomelanocortin	Characteristic of placental membrane 10× more concentrated in amniotic fluid than maternal serum Inversely related to human chorionic gonadotropin High concentrations at feto-maternal junction
Endothelin	Increased when amniotic fluid injected into circulation Meconium stained amniotic fluid elicited greatest degree of endothelial injury
Procollagen type 1 N-terminal propeptide	Associated with fetal metabolism and development >400 times more concentrated in amniotic fluid than maternal circulation
CK13 (Rab); CK10/13 (Mab)	Strongly expressed in squamous cells from maternal amniotic fluid Brown cytoplasmic granules when immunostained

Zinek - coproporphyrin - 1 (Zn - CP1)

Běžná hodnota < 1,6 pmol /ml

Sialyl - Tn

Běžná hodnota < 46 IU /ml

Interleukin - 8

Běžná hodnota < 20 pg /ml

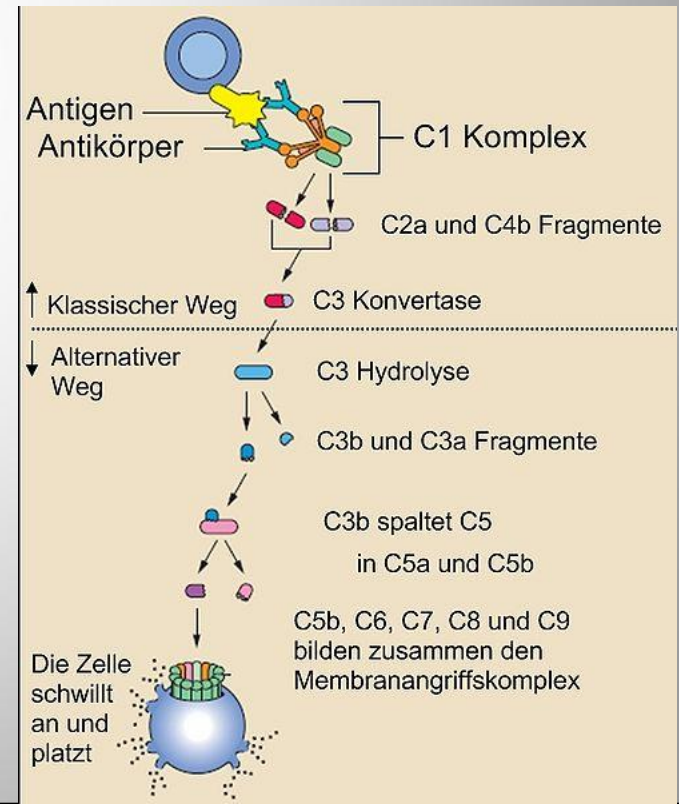
C3, C4 komplement (sérum)
vysoká senzitivita i specificita AFE

Komplement C3 a C4

Normální hodnoty

C3: 80 až 140 mg /dl

C4: 11 až 34 mg /dl



C1 Esterase Inhibitor Activity in Amniotic Fluid Embolism*

Naoaki Tamura, MD, PhD¹; Satoshi Kimura, MD¹; Mustari Farhana, MD¹;
Toshiyuki Uchida, MD, PhD¹; Kazunao Suzuki, MD, PhD¹; Kazuhiro Sugihara, MD, PhD¹;
Hiroaki Itoh, MDSc¹; Tomoaki Ikeda, MD, PhD²; Naohiro Kanayama, MD, PhD¹

Objectives: Amniotic fluid embolism exhibits activation of the complement system and the kallikrein-kinin and coagulofibrinolytic systems. C1 esterase inhibitor is a major inhibitor of C1 esterase and can inhibit plasma kallikrein and also factors XIIa and XIa. Its activity has been shown to be significantly lower in pregnancy and labor than in the nonpregnant state. The purpose of this study was to determine C1 esterase inhibitor activity levels in amniotic fluid embolism.

Design: Retrospective study.

Setting: A single university-based center.

Patients: One hundred six cases with amniotic fluid embolism in a total of 194 singleton pregnant women between January 2010 and December 2011.

Interventions: None.

Measurements and Main Results: One hundred six cases of amniotic fluid embolism had applied to the Japan amniotic fluid embolism registration center in Hamamatsu University School of Medicine between January 2010 and December 2011. In amniotic fluid embolism cases, 85 cases were nonfatal and 21 cases were fatal. Eighty-eight women who delivered without amniotic fluid embolism were regarded as a control. C1 esterase inhibitor activity levels were significantly lower in amniotic fluid embolism patients ($30.0\% \pm 1.8\%$) than in control women ($62.0\% \pm 2.0\%$) ($p < 0.0001$). C1 esterase inhibitor activity levels in fatal amniotic fluid embolism cases ($22.5\% \pm 3.4\%$) were significantly lower than those in nonfatal amniotic fluid embolism cases ($32.0\% \pm 2.1\%$) ($p < 0.05$).

Conclusions: These results demonstrated that low C1 esterase inhibitor activity levels were closely associated with the pathogenesis of amniotic fluid embolism suggesting that C1 esterase inhibitor activity levels have potential as a prognosis factor of amniotic fluid embolism. (*Crit Care Med* 2014; 42:1392–1396)

Key Words: amniotic fluid embolism; C1 esterase inhibitor; disseminated intravascular coagulopathy; kallikrein; postpartum hemorrhage; serpin

Amniotic fluid embolism (AFE) is one of the most serious complications of obstetrics, anesthetics, and critical care. Despite earlier recognition and intensive critical care, the mortality of AFE remains high and has been estimated at between 5% and 15% of all maternal deaths (1). Maternal mortality rates due to AFE have been estimated at between 37% and 80% (2, 3). Maternal death has been decreasing year by year in Japan; however, the prevalence of maternal death due to AFE has remained unchanged. The maternal mortality rate due to AFE has increased to 24.3% in Japan (4).

AFE is recognized as a kind of syndrome characterized by the abrupt onset of hypoxia, hypotension, and disseminated intravascular coagulopathy (DIC) (5). Benson et al (6) reported that maternal complement levels were significantly decreased in AFE. These findings suggested a disorder in the coagulofibrinolytic system as well as the complement system that may play important roles in the pathogenesis of AFE.

We developed the Japan AFE registration system in 2003 and collected clinical data, maternal serum, and uterine tissue from nearly all cases of fatal AFE in Japan (4, 7). Under the system, maternal serum has been applied to determine mainly the levels of specific amniotic fluid complements such as Sialyl Tn and zinc coproporphyrin 1 (8, 9). These clinical and his-

*See also p. 1548.

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²Department of Obstetrics and Gynaecology, Mie University School, Mie, Japan.

Dr. Kanayama's institution received grant support from Japan Society for

Inhibitor C-1 esterázy

Pokles < 26%

Clinical Case Reports

Open Access

CASE REPORT

Therapeutic application of C1 esterase inhibitor concentrate for clinical amniotic fluid embolism: a case report

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¹Department of Obstetrics & Gynaecology, Hamamatsu University School of Medicine, Hamamatsu, Shizuoka, Japan

²Department of Obstetrics & Gynaecology, Mie University School, Mie, Japan

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Funding Information

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Received: 24 January 2015; Revised: 6 April 2015; Accepted: 21 May 2015

Clinical Case Reports 2015; 3(7): 673–675

Key Clinical Message

We present the successful application of C1 esterase inhibitor (C1INH) concentrate to a patient with clinical amniotic fluid embolism (AFE).

Keywords

Amniotic fluid embolism, C1 esterase inhibitor, disseminated intravascular coagulopathy, kallikrein, uterine atony.

Biologické markery v séru:

- stávající markery (ne)spolehlivé (senzitivita a specificita)
- dlouhý časový interval pro diagnostiku

Včasná diagnostika – kardiologie???:

- analýza variability srdeční frekvence,
- 3D-spektrogram a nukleární kardiologie

Liao WC, Jaw FS

*A noninvasive evaluation analysis of amniotic fluid
embolism and disseminated intravascular*

coagulopathy. J Matern Fetal Neonatal Med. 2011 Nov;24(11):1411-5. Epub 2011 Jan 19.

Table 3. Differential diagnoses by organ system

Differential diagnoses

Respiratory

Pulmonary embolism

Air embolism

Aspiration

Cardiac

Acute myocardial infarction

Peripartum cardiomyopathy

Aortic dissection

Cardiac arrhythmia

Shock

Septic shock

Anaphylactic shock: drug induced

Obstetric

Postpartum hemorrhage

Uterine rupture

Placental abruption

Eclampsia

Embolie plodovou vodou

Historická představa

Průnik plodové vody do mateřského oběhu

Mechanické látky

(fetální skvamózní epitelie, lanugo, vlasy, mázek, mekónium)

Obstrukce plicních kapilár a kardiovaskulární kolaps

Embolie plodovou vodou

Průnik plodové vody do mateřského oběhu

Solubilní látky

(histamin, bradykinin, prostaglandiny, aktivátory komplementu, endotelin, leukotrieny a metabolity kyseliny arachidinové)

Embolie plodovou vodou

Antigenní produkty plodového vejce

Anafylaktoidní těhotenský syndrom

Imunologická reakce

AF + mekonium = toxičtější než AF bez mekonie

Matka zemřela na vzácnou alergii vteřiny poté, co poprvé objala své dítě

Osmadvacetiletá Julie Welshová z britského Derby podlehl silné alergické reakci, která udeřila jen chvíli poté, co porodila zdravého syna Isaaca. Tamější lékaři se s něčím takovým prý ještě nesetkali, informovali však o tom, že podobné riziko hrozí zhruba 18 matkám z miliónu. V asi 80 procentech případů je tato alergická reakce smrtelná.



Zvětšit obrázek

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čtvrtek 4. listopadu 2010, 11:39 - Derby

Julie Welshová měla dostatek času jen na to, aby k

▲ Matka zemřela krátce po
objetí svého novorozeného
chlapce. Ilustrační foto

FOTO: fotopicka Profimedia

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naposled vzletly z letadlové lodi](#)
▶ Video



[Při studentských protestech v
Británii útočily i dívky](#) ▶ Video



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na summitu NATO](#)



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▼ Komerční sdělení

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Katalog 2011 zdarma.](#)



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AFE or Septic Shock?



Maternal death following cardiopulmonary collapse after delivery: amniotic fluid embolism or septic shock due to intrauterine infection?
Romero R, Kadar N, Vaisbuch E, Hassan SS.
Am J Reprod Immunol. 2010 Aug 1;64(2):113-25.

Amniotic fluid embolism: an evidence-based review.
Conde-Agudelo A, Romero R.
Am J Obstet Gynecol. 2009 Nov;201(5):445.e1-13.

Embolie plodovou vodou

Podobnost

- septický šok (*chybí teplota*)
- anafylaktický šok (*chybí kožní projevy*)

41% rodiček s AFE registru trpí alergií nebo atopií

Incidence and risk factors of amniotic fluid embolisms: a population-based study on 3 million births in the United States. Am J Obstet Gynecol. 2008

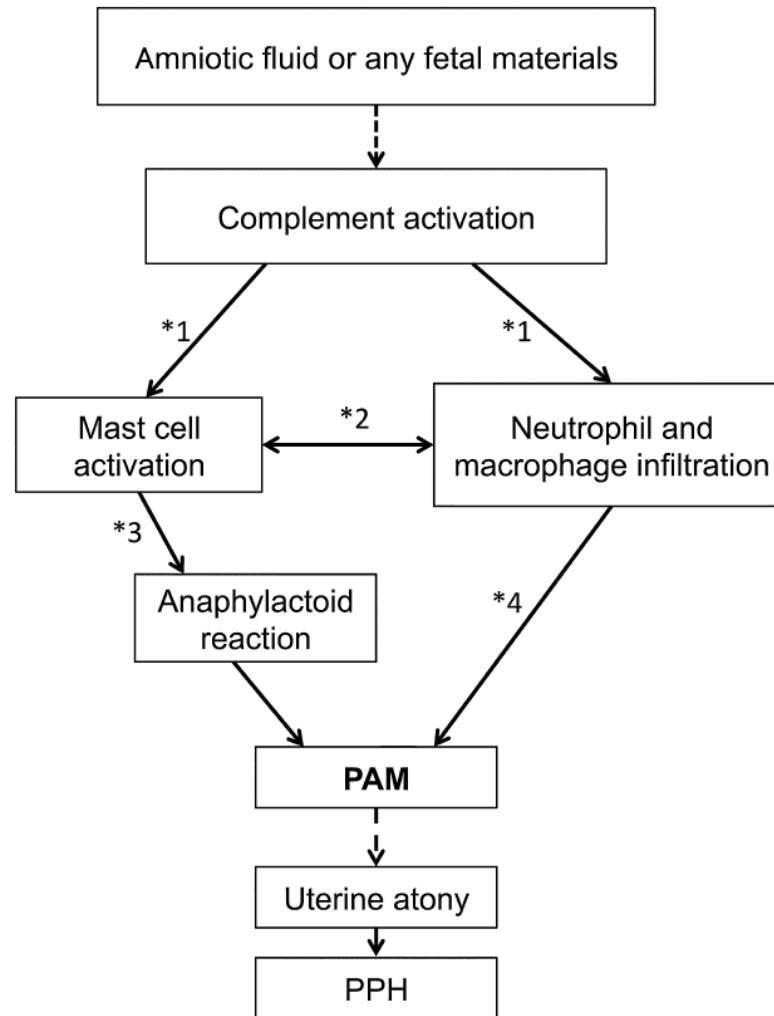


Fig. 4. Scheme indicating the mechanism of pathogenesis in PPH of unknown etiology. * 1: Anaphylatoxin (C3a, C5a), * 2: IL-1, IL-8, TNF- α , * 3: Degranulation of mast cells and * 4: Matrix metalloproteinase and free radicals were possible components. PAM: Postpartum acute myometritis.

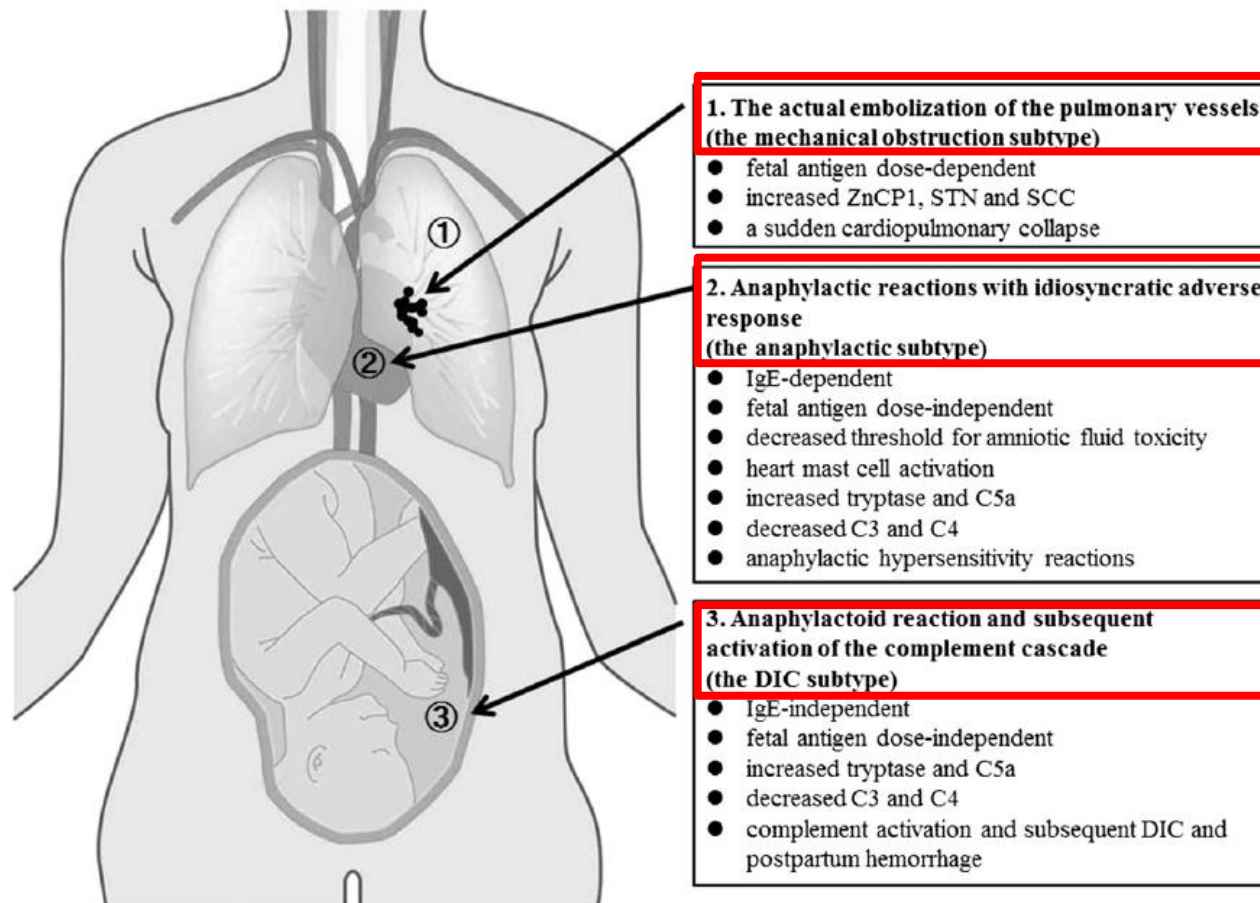


FIG. 1. Pathophysiological classification of AFE. Amniotic fluid embolism can be classified as (1) the mechanical obstruction subtype, (2) the anaphylactic subtype, and (3) the DIC subtype. This subclassification may be beneficial in the diagnosis and the personalized treatment of AFE.

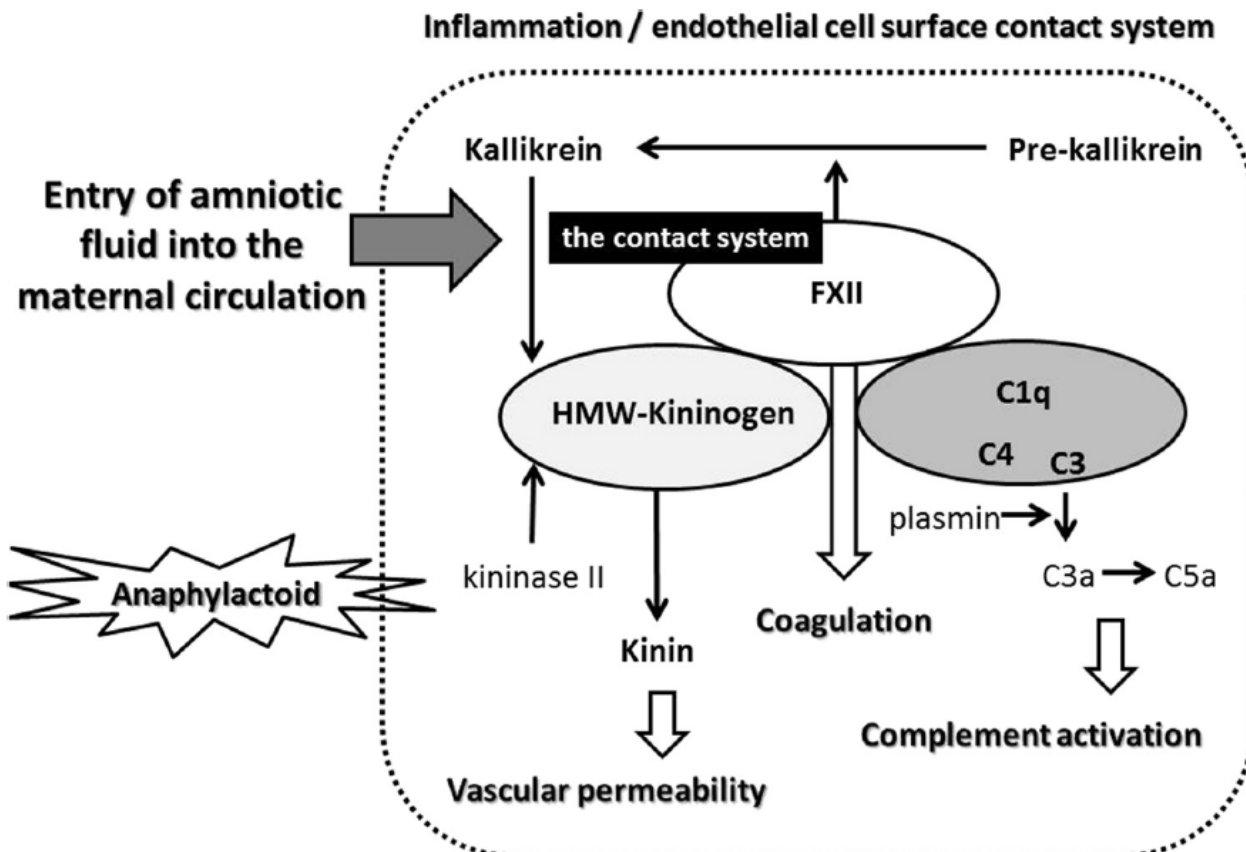


FIG. 2. Endothelial cell surface contact system. The entry of amniotic fluid into the maternal circulation activates endothelial cell surface contact system. Bradykinin is proteolytically cleaved from high-molecular-weight (HMW) kininogen through the contact system cascade involving coagulation factor XII and kallikrein.^{19,41} The plasma contact activation system is involved in coagulation (activated factor XII), complement activation (C1q), and vascular permeability (local generation of kinin). The involvement of the anaphylactoid reaction in the process of the contact system initiates the activation of coagulation, fibrinolysis, complement, and kinin cascade. The beneficial effects of kallikrein and kininase II are attributed to prolongation of the half-life of bradykinin.

TABLE 4

Differential diagnosis of amniotic fluid embolism

	Amniotic fluid embolism	Hemorrhage	Sepsis	Anesthetic accident	Pulmonary thromboembolism	Systemic anaphylaxis
Hypotension	+++	+++	+++	+++	++	+++
Hypoxia	+++	+/-	+	+++	+++	+++
Coagulopathy	+++	+	+	No	No	No
Sudden onset	Yes	No	No	Yes	Yes	Yes
Prior fever	No	No	Yes	No	No	No
Recognized antecedent event	No	Hemorrhage	Chorioamnionitis	Anesthetic administration	No	Medication administration

Clark. Case definition of amniotic fluid embolism to improve quality of clinical and translational research. *Am J Obstet Gynecol* 2016.

Embolie plodovou vodou

Steiner a Lushbaugh = více než 70 let

Rozpaky

Skepse

Frustrace

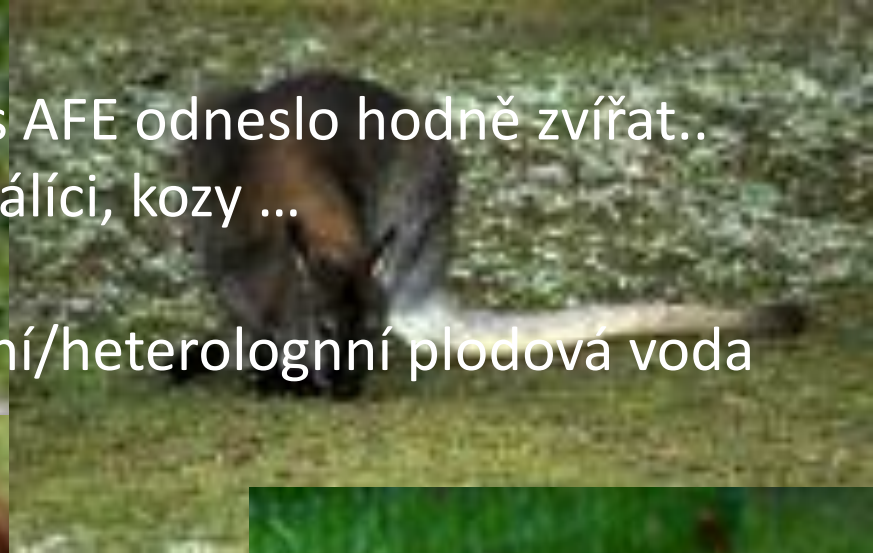
Zmar

Patofyziologie

multifaktoriální proces → chudé znalosti

Pokusy s AFE odneslo hodně zvířat..
krysy, králíci, kozy ...

autologní/heterologní plodová voda



Embolie plodovou vodou

Proč nižší výskyt?

Nesnižuje se incidence...

...zlepšují se metody intenzivní medicíny
při řešení život ohrožujících stavů.

Hlavní rizika

Table 1. Risk factors for amniotic fluid embolism

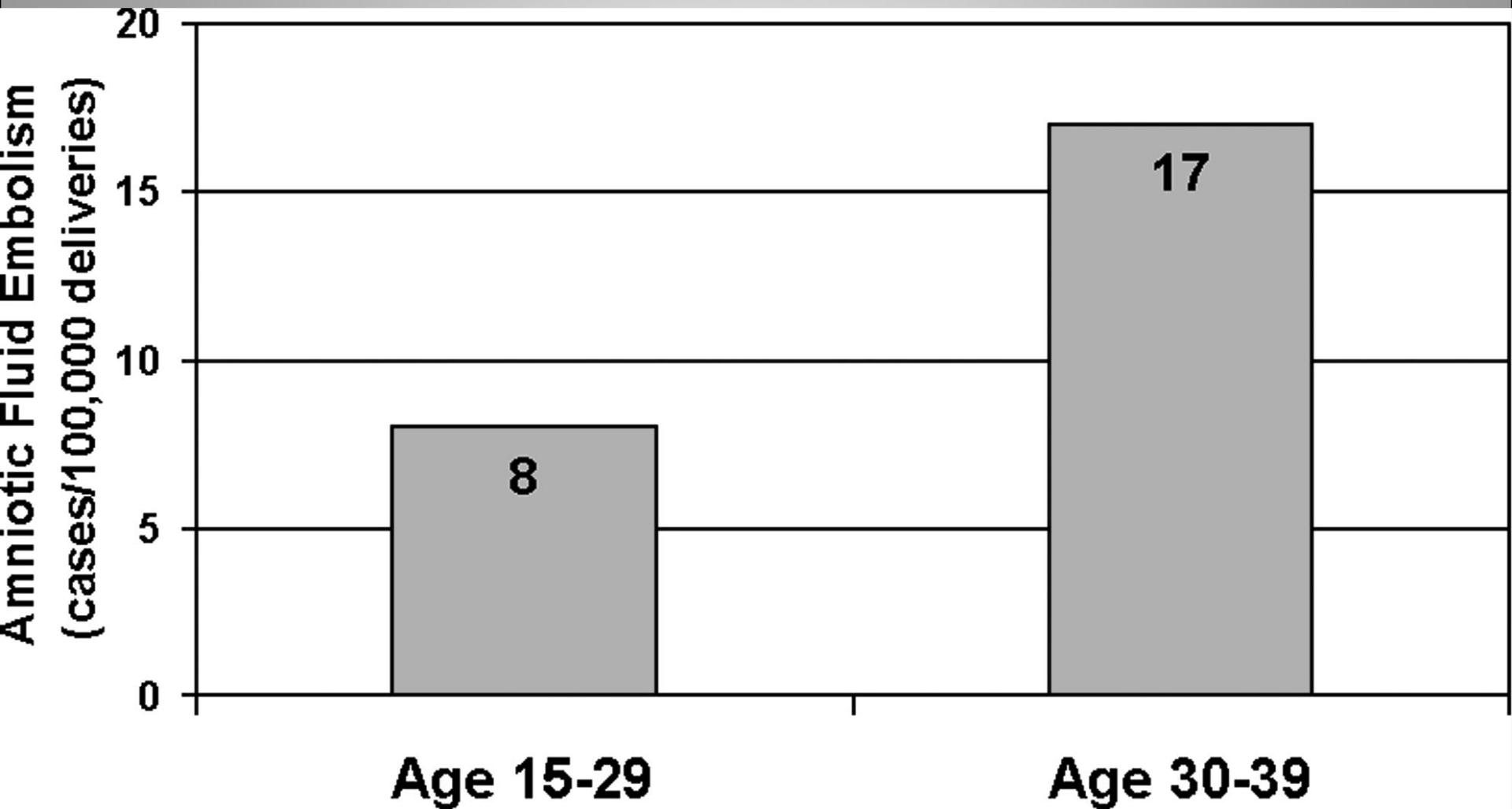
Fetal factors	Maternal factors	Obstetrical factors
Male gender	Advanced maternal age >35	Cesarean section
Placenta previa	Multiparity	Cervical laceration
Placenta accreta	Diabetes	Induction of labor
Placental abruption		Instrumental delivery: forceps and vacuum-assisted vaginal deliveries
Fetal distress		Uterine rupture
Polyhydramnios		Eclampsia
Fetal macrosomia		Oxytocin use
Premature rupture of membranes		
Intrauterine death		

REVIEW



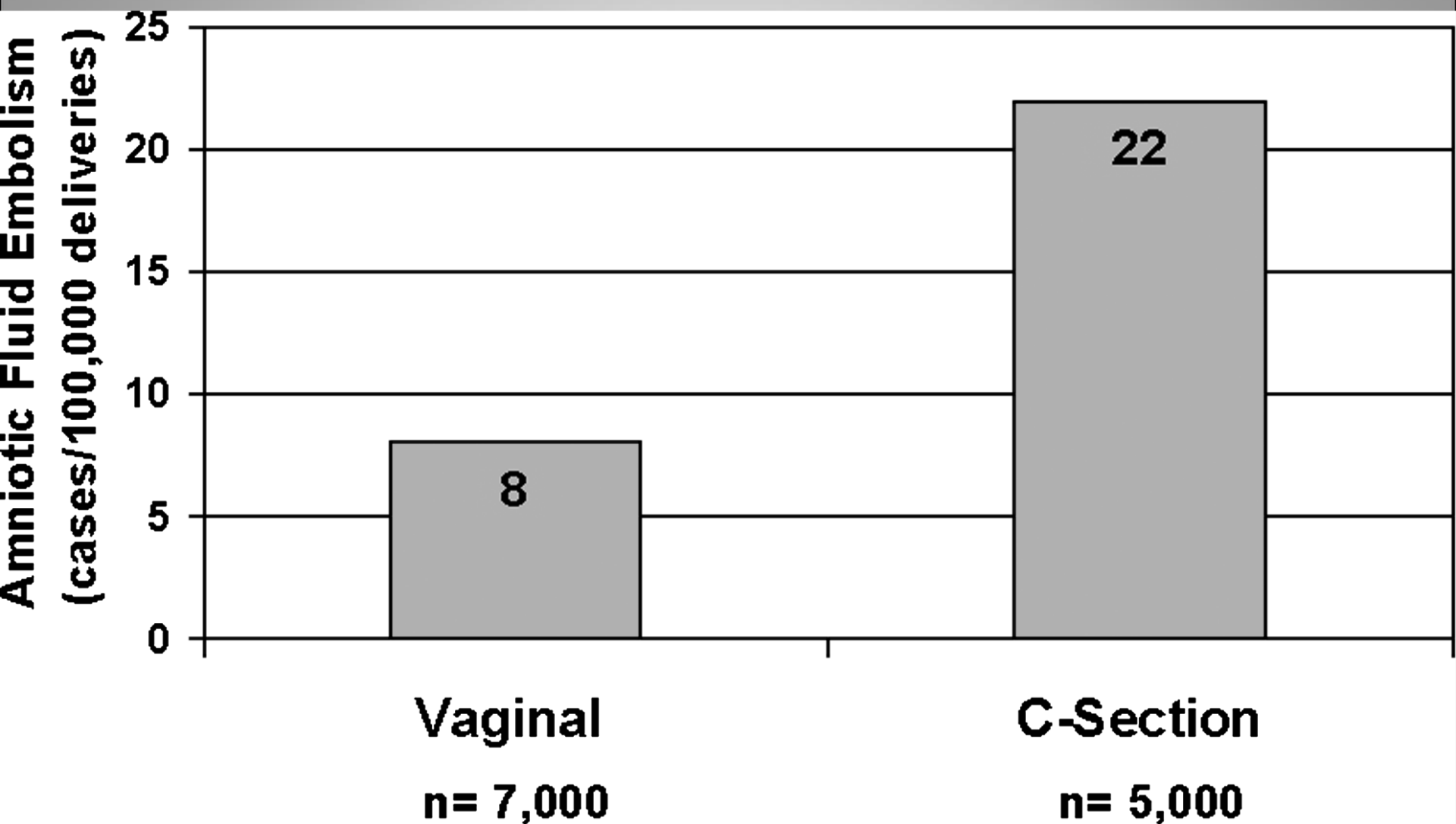
Amniotic fluid embolism: despite progress, challenges remain

Kathryn J. Baling^a, Melissa T. Chu Lam^b, Heidi H. Hon^a, Stanislaw P. Stawicki^a, and James N. Anast^b



Paul D. Stein, M.D.,^{1,2} Fadi Matta, M.D.,¹ and Abdo Y. Yaekoub, M.D.¹

Incidence of Amniotic Fluid Embolism: Relation to Cesarean Section and to Age. JOURNAL OF WOMEN'S HEALTH, Volume 18, Number 3, 2009.



*Paul D. Stein, M.D.,^{1,2} Fadi Matta, M.D.,¹ and Abdo Y. Yaekoub, M.D.¹
Incidence of Amniotic Fluid Embolism: Relation to Cesarean Section and to Age. JOURNAL OF WOMEN'S HEALTH, Volume 18, Number 3, 2009.*

**Rozhoduje rychlost
a reakce v rozhodování...**

Embolie plodovou vodou

Pouhé podezření

Agresivní terapie

Mezioborová spolupráce

Porodník

Anesteziolog

Anesteziolog/intenzivista

Hematolog

event. patolog

Podezření na AFE



Okamžitá akce

- zajistit dýchací cesty
- dvě silné i.v. kanyly/přístupy
- okamžitě hematologické odběry
- okamžitě objednání krevních náhrad
- zajistit léky/vasopresory
- příprava operačního sálu



Akutní císařský řez/emergent SC



Pooperační péče

- Jednotka intenzivní péče
- resuscitace tělního oběhu/oběmu
- seriál/monitoring
hematologických/biochemických vyšetření
- použití vazopresorů

Úmrtnost žen na AFE u císařského řezu
byla nižší, než v ostatních případech...

Vysvětlení:

přítomnost anesteziologa,
možnost resuscitace...

*Oi H, Naruse K, Noguchi T, Sado T, Kimura S, Kanayama N, Terao T,
Kobayashi H.*

*Fatal factors of clinical manifestations and laboratory testing in patients
with amniotic fluid embolism.*

Gynecol Obstet Invest. 2010;70(2):138-44. Epub 2010 Apr 24.



Mimotělní oxygenace u těhotných s AFE

Embolie plodovou vodou

- STATIM ukončit těhotenství
- symptomatická léčba – zajištění vitálních funkcí
- statim koagulační vyšetření – očekáváme DIC
- statim deleukotizovaná Ery-masa, FFP
- centrální žilní katétr/a.pulmonalis

Národní sběr dat

www.AFE.porodnice.cz

Kritéria pro diagnostiku embolie plodové vody

1.

Příznaky se objevily v průběhu těhotenství nebo do 12 hod. po porodu.

2.

Byl proveden některý lékařský postup pro léčbu jednoho nebo více následujících symptomů:

- srdeční zástava
- život ohrožující krvácení neznámého původu do 2 hod po porodu (≥ 1500 ml)
- disseminovaná intravaskulární koagulace
- respirační selhání

3.

Pokud uvedené symptomy nelze vysvětlit jinou příčinou/nemocí





Rath W. Postpartum Haemorrhage (PPH):

"too little is done too late"!

Z Geburtshilfe Neonatol. 2011 Oct;215(5):177-81.