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Amniotic Fluid Embolism

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1. Amniotic fluid embolism: Pathophysiology from the perspective of pathology

Author(s): Tamura N.; Farhana M.; Oda T.; Itoh H.; Kanayama N.

Source: Journal of Obstetrics and Gynaecology Research; Apr 2017; vol. 43 (no. 4); p. 627-632

Publication Date: Apr 2017

Publication Type(s): Article

Available in full text at Journal of Obstetrics and Gynaecology Research - from John Wiley and Sons

Abstract: Amniotic fluid embolism (AFE) is recognized as a type of syndrome characterized by the abrupt onset of hypoxia, hypotension, seizures, or disseminated intravascular coagulopathy (DIC), occurring during labor, delivery, or immediately postpartum, caused by the inflow of amniotic components into the maternal circulation. AFE is a rare condition but one of the most serious obstetrical complications, resulting in a high mortality rate among pregnant women. Despite earlier recognition and intensive critical management, we often encounter patients who unfortunately do not recover from the exacerbation of AFE-related conditions. A major concern is that there are no effective evidence-based therapies for AFE, because its pathophysiology is still not well understood. This article reviewed AFE, focusing on the pathology and currently proposed pathophysiology.Copyright © 2017 Japan Society of Obstetrics and Gynecology

Database: EMBASE

2. Amniotic fluid embolism

Author(s): Tuffnell D.J.; Slemeck E.

Source: Obstetrics, Gynaecology and Reproductive Medicine; Mar 2017; vol. 27 (no. 3); p. 86-90

Publication Date: Mar 2017

Publication Type(s): Review

Abstract: Amniotic fluid embolism (AFE) is a rare but severe complication of pregnancy characterised by a catastrophic systemic dysfunction involving the respiratory, cardiovascular and haematological systems. Its incidence in the UK is approximately 1 in 59,000 maternities, but despite its overall rarity AFE is responsible for a significant proportion of the maternal deaths across the developed world and was the third leading direct cause of maternal deaths ascertained by the UK confidential enquiry 2009-2011. However, it should no longer be considered as resulting in inevitable mortality, and increasing evidence shows that good supportive care can result in improved outcomes for mother and baby. Current data puts case fatality rates for AFE in the UK at around 19%, much lower than previously thought. This review collates the latest literature looking at how and when AFE occurs, its presentation, diagnosis and management.Copyright © 2017 Elsevier Ltd

3. Risk factors for fatality in amniotic fluid embolism: a systematic review and analysis of a data pool.

Author(s): Indraccolo, Ugo; Battistoni, Caterina; Mastrantonio, Irene; Di Iorio, Romolo; Greco, Pantaleo; Indraccolo, Salvatore Renato

Source: The journal of maternal-fetal & neonatal medicine : the official journal of the European Association of Perinatal Medicine, the Federation of Asia and Oceania Perinatal Societies, the International Society of Perinatal Obstetricians; Mar 2017 ; p. 1-5

Publication Date: Mar 2017

Publication Type(s): Journal Article

Abstract:PURPOSEInvestigating risk factors for amniotic fluid embolism (AFE)-induced fatality.METHODSA systematic review of cases of AFE available on PubMed, Scielo, Scopus and AJOL databases that occurred from 1990 to 2015 was carried out. After careful reading of titles, abstracts and full texts, case reports of AFE were reviewed. Risk factors for AFE were considered as independent variables in logistic regression models. The first model was built on the whole data pool. The second model was built on typical cases of AFE, according to the classical triad of symptoms (heart, lungs, coagulopathy). The dependent variable was fatality in both models.RESULTS177 cases of AFE were assessed in the first model, while 121 typical cases of AFE were assessed in the second model. Among typical cases of AFE, only oxytocin infusion during labour increases the likelihood of death (odds ratio 2.890, 95% confidence interval 1.166-7.164, p = 0.022). No risk factors for fatality were found in the whole data pool.CONCLUSIONSFurther research on national registries should focus on the behaviour of oxytocin infusion during labour in AFE cases.

Database: Medline

4. Successful treatment of life-threatening hemorrhaging due to amniotic fluid embolism

Author(s): Aurini L.; Rainaldi M.P.; White P.F.; Borghi B.

Source: Minerva Anestesiologica; Nov 2016; vol. 82 (no. 11); p. 1238-1239

Publication Date: Nov 2016

Publication Type(s): Letter

Available in full text at Minerva Anestesiologica - from Free Access Content

Database: EMBASE

5. Amniotic Fluid Embolism with Isolated Coagulopathy: A Report of Two Cases.

Author(s): Liao, Chi-Yuan; Luo, Fuh-Jinn

Source: Journal of clinical and diagnostic research : JCDR; Oct 2016; vol. 10 (no. 10); p. QD03

Publication Date: Oct 2016

Publication Type(s): Journal Article

Available in full text at Journal of Clinical and Diagnostic Research : JCDR - from National Library of Medicine

Abstract: Amniotic Fluid Embolism (AFE) is a catastrophic complication of pregnancy with high mortality rate. The most common clinical presentation is an abrupt onset of cardiopulmonary collapse. Here, we present an uncommon variant involving isolated disseminated intravascular coagulation that developed without antecedent cardiopulmonary disturbances. Both patients developed symptoms soon after delivery. Blood test was sent at 14 minutes postpartum for the second patient due to suspected amniotic fluid embolism. Fetal components were observed in the uterine veins of the lower uterine segments in both cases. Amniotic fluid embolism with disseminated intravascular coagulopathy typically progresses faster than disseminated intravascular coagulopathy associated with other causes and symptoms. It usually develops within two hours of delivery. Prompt recognition and treatment of this entity is crucial to survival.

Database: Medline

6. Amniotic fluid embolism after intrauterine fetal demise.

Author(s): Kristensen, Karl; Langdana, Fali; Clentworth, Howard; Hansby, Chu; Dalley, Paul

Source: The New Zealand medical journal; Sep 2016; vol. 129 (no. 1441); p. 87-88

Publication Date: Sep 2016

Publication Type(s): Case Reports Journal Article

Available in full text at New Zealand Medical Journal, The - from ProQuest

Abstract: We present a case of the successful treatment of severe amniotic fluid embolism in a 41-year-old woman undergoing emergency caesarean section at 36 weeks of gestation for placental abruption and intrauterine fetal demise. The treatment included prolonged cardiopulmonary resuscitation, emergency hysterectomy, re-operation with intra-abdominal packing and intra-aortic balloon pump insertion. The patient made a remarkable recovery and to date has minimal residual morbidity. Amniotic fluid embolism syndrome (AFES) is a rare and often fatal obstetric condition that remains one of the main causes of maternal mortality in developed countries. The incidence varies from 2 to 6 per 100,000 and suggested mortality rates exceed 60%.1-2 The classic triad of sudden hypoxia, hypotension and coagulopathy with acute onset during labour or immediately after delivery forms the hallmark of the AFES diagnosis, however AFES is primarily a clinical diagnosis of exclusion. We present a case of successful maternal outcome following severe amniotic fluid embolism after placental abruption and intrauterine fetal demise.

Database: Medline

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

Author(s): Shen F.; Wang L.; Yang W.; Chen Y.

Source: Archives of Gynecology and Obstetrics; Feb 2016; vol. 293 (no. 2); p. 329-334

Publication Date: Feb 2016

Publication Type(s): Article

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 examine population-based regional data from Suzhou, China. Based on the analysis of all available case reports, we put forward an outline of atypical AFE and investigate whether any variation identified could be ascribed to methodology. Methods: Retrospective study from January 2004 to December 2013, 53 cases was identified from the database of Center for Disease Control (CDC) in the city of Suzhou. We investigated the presentations of atypical AFE and maternal characteristics with potential factors underlying AFE. Multiple-regression analysis was used to calculate adjusted odds ratios (ORs) and 95 % confidence intervals (CIs). Results: The incidence of AFE was 6.91 per 100,000 deliveries (53/766,895). Seventeen deaths occurred, a mortality rate of 32 %. Atypical AFE may as the earlier stage or mild form of AFE, there was no death case in the study with timely remedy. The atypical AFE appear is obstetric hemorrhage and/or pulmonary and renal dysfunction postpartum. Hyperfibrinolysis and coagulopathy may 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 AFE is critical to turn it to a remediable disease. Patient complaints such as breathlessness, chest pain, feeling cold, distress, panic, a feeling of nausea, and vomiting should elicit close attention. The management of a suspected episode of amniotic fluid embolism is generally considered to be supportive. Hysterectomy must be performed if there is further progression of symptoms. Due to advances in acute care, mortality has decreased in recent years, highlighting the importance of early detection and treatment.

Database: EMBASE

8. Incidence, risk factors, management and outcomes of amniotic-fluid embolism: a population-based cohort and nested case-control study.

Author(s): Fitzpatrick, K E; Tuffnell, D; Kurinczuk, J J; Knight, M

Source: BJOG : an international journal of obstetrics and gynaecology; Jan 2016; vol. 123 (no. 1); p. 100-109

Publication Date: Jan 2016

Publication Type(s): Multicenter Study Journal Article Observational Study

Available in full text at BJOG: An International Journal of Obstetrics and Gynaecology - from John Wiley and Sons

Abstract:OBJECTIVETo describe the incidence, risk factors, management and outcomes of amniotic-fluid embolism (AFE) over time.DESIGNA population-based cohort and nested case-control study using the UK Obstetric Surveillance System (UKOSS).SETTINGAll UK hospitals with obstetrician-led maternity units.POPULATIONAll women diagnosed with AFE in the UK between February 2005 and January 2014 (n = 120) and 3839 control women.METHODSProspective case and control identification through UKOSS monthly mailing.MAIN OUTCOME MEASURESAmniotic-fluid embolism, maternal death or permanent neurological injury.RESULTSThe total and fatal incidence of AFE, estimated as 1.7 and 0.3 per 100 000, respectively, showed no significant temporal trend over the study period and there was no notable temporal change in risk factors for AFE. Twenty-three women died (case fatality 19%) and seven (7%) of the surviving women had permanent neurological injury. Women who died or had permanent neurological injury were more likely to present with cardiac arrest (83% versus 33%, P < 0.001), be from ethnic-minority groups (adjusted odds ratio [OR] 2.85, 95% confidence interval [95% CI] 1.02-8.00), have had a hysterectomy (unadjusted OR 2.49, 95% CI 1.02-6.06), had a shorter time interval between the AFE event and when the hysterectomy was performed (median interval 77 minutes versus 248 minutes, P = 0.0315), and were less likely to receive cryoprecipitate (unadjusted OR 0.30, 95% CI 0.11-0.80).CONCLUSIONThere is no evidence of a temporal change in the incidence of or risk factors for AFE. Further investigation is needed to establish whether earlier treatments can reverse the cascade of deterioration leading to severe outcomes.

Database: Medline

9. Amniotic fluid embolism: Despite progress, challenges remain

Author(s): Balinger K.J.; Hon H.H.; Stawicki S.P.; Chu Lam M.T.; Anasti J.N.

Source: Current Opinion in Obstetrics and Gynecology; 2015; vol. 27 (no. 6); p. 398-405

Publication Date: 2015

Publication Type(s): Review

Available in full text at Current Opinion in Obstetrics and Gynecology - from Ovid

Abstract: Purpose of review This article reviews the incidence, pathophysiology, risk factors, diagnosis, and management of amniotic fluid embolism (AFE). Recent findings AFE is a leading cause of maternal morbidity and mortality despite an incidence of approximately 7 to 8 per 100 000 births. Recent reevaluation of AFE suggests that the presence of fetal tissue in maternal circulation alone is not sufficient to cause the clinical syndrome, but rather an individual's response to this fetal tissue. The 'anaphylactoid reaction' associated with AFE shares many clinical and metabolic aspects of septic shock. Acute dyspnea followed by cardiovascular collapse, coagulopathy, and neurological symptoms, such as coma and seizures may all be associated with the clinical AFE syndrome. Specific biochemical markers have been described, but are of limited clinical value because of the rapid progression of the disease process. Treatment is based on an interdisciplinary approach that consists

of a combination of prompt, aggressive hemodynamic resuscitation, provision of end-organ support, correction of hemostatic disorders, and delivery. Summary Although AFE cannot be prevented, early diagnosis and intervention may lead to better outcomes for both the mother and the fetus. Clinical suspicion, traditional laboratory data, or intravascular cellular debris (demonstrated only in 50% of patients) are insufficient to make a definitive diagnosis of AFE. An evolving array of novel biomarkers may help differentiate AFE from other conditions, but none of them currently provide sufficient 'early warning' ability to make real-time impact on diagnosis and/or treatment of AFE.

Database: EMBASE

10. Amniotic fluid embolism after uterine artery embolization for uterine fibroids.

Author(s): Jeanneteau, Pauline; Legendre, Guillaume; Rousseau, Audrey; Descamps, Philippe; Sentilhes, Loïc

Source: European journal of obstetrics, gynecology, and reproductive biology; Aug 2015; vol. 191 ; p. 144-145

Publication Date: Aug 2015

Publication Type(s): Letter Case Reports

Database: Medline

11. Amniotic components in the uterine vasculature and their role in amniotic fluid embolism.

Author(s): Nakagami, Hiroko; Kajihara, Takeshi; Kamei, Yoshimasa; Ishihara, Osamu; Kayano, Hidekazu; Sasaki, Atsushi; Itakura, Atsuo

Source: The journal of obstetrics and gynaecology research; Jun 2015; vol. 41 (no. 6); p. 870-875

Publication Date: Jun 2015

Publication Type(s): Journal Article

Available in full text at Journal of Obstetrics and Gynaecology Research - from John Wiley and Sons

Abstract:AIMTo evaluate whether the presence of amniotic components in the maternal uterine vasculature could be a specific pathological indicator for amniotic fluid embolism (AFE).METHODSMedical records of patients treated between January 2006 and March 2013 were retrospectively examined to identify patients who underwent post-partum hysterectomy or autopsy due to maternal death. Three subjects with AFE with disseminated intravascular coagulation (DIC)-type post-partum hemorrhage (PPH), and 13 non-AFE subjects were included in analysis. Histochemical staining using hematoxylin-eosin (HE) and alcian blue, and immunohistochemical staining for sialyl-Tn were conducted to detect amniotic components in the maternal uterine vasculature.RESULTSAlcian blue was positive for amniotic components in the uterine vasculature of all subjects with AFE and of several subjects without AFE. Similarly, HE and sialyl-Tn were negative in some AFE subjects and positive in some non-AFE subjects.CONCLUSIONSThe presence of maternal intravascular fetal material is not a specific indicator for AFE with DIC-type PPH. Therefore, the presence of fetal components in the uterine vasculature is unlikely to be a definitive indicator for AFE.

Database: Medline

12. Amniotic embolism with complement activation in a lupic pregnant woman.

Author(s): Campanharo, F F; Santana, E F M; Araujo Júnior, E; Sarmento, S G P; Fernandes, F C; Sun, S Y; Mattar, R; Moron, A F

Source: Journal of obstetrics and gynaecology : the journal of the Institute of Obstetrics and Gynaecology; May 2015; vol. 35 (no. 4); p. 416

Publication Date: May 2015

Publication Type(s): Case Reports Journal Article

Database: Medline

13. Activation contact system (ACS) and tissue factor (TF) in human amniotic fluid: measurements of ACS components and TF, and some implications on the pathophysiology of amniotic fluid embolism.

Author(s): Uszyński, Waldemar; Żekanowska, Ewa; Uszyński, Mieczysław; Kieszkowski, Przemysław

Source: Thrombosis research; Apr 2015; vol. 135 (no. 4); p. 699-702

Publication Date: Apr 2015

Publication Type(s): Journal Article

Abstract:BACKGROUND/AIMIt is believed that the amniotic fluid-derived TF, in the case of amniotic fluid embolism (AFE), contributes to acute disseminated intravascular coagulation (DIC) and obstetric shock in the mother. However, the role of amniotic fluid-derived contact phase coagulation factors that irrupt into the bloodstream simultaneously with TF is still unknown. Our study objective was to identify and measure the concentrations of CAS components and TF in amniotic fluid.MATERIAL AND METHODSThe study group consisted of 30 healthy parturients with uneventful pregnancy and birth. Amniotic fluid (AF) and maternal blood were sampled at the end of the first stage of labor. The components of ACS, i.e. factors XII and XI (FXII, FXI), prekallikrein (PK), high molecular weight kininogen (HMWK), and tissue factor (TF) were measured by immunoenzymatic method (ELISA).RESULTSAll ACS components were detected in AF; their levels were higher in AF than in the maternal plasma: FXII--29.17 ng/mg protein vs. 0.94 ng/mg protein (medians); FXI--27.28 ng/mg protein vs. 0.92 ng/mg protein (medians); PK--88442.04 ng/mg protein vs. 113.44 ng/mg protein (medians); HMWK--4253.82 ng/mg protein vs. 2857.96 ng/mg protein (medians). The concentration of TF in amniotic fluid was 39.46 pg/mg protein (median) vs. 0.41 pg/mg protein (median) in blood plasma.CONCLUSIONS1.The ACS components, i.e. FXII, FXI, PK and HMWK, are the constituents of amniotic fluid. 2.The concentrations of the amniotic fluid-derived factors having a coagulation initiation potential, i.e. TF and contact phase coagulation factors, are higher in amniotic fluid than in mother's blood plasma.

Database: Medline

14. Amniotic fluid embolism pathophysiology suggests the new diagnostic armamentarium: beta-tryptase and complement fractions C3-C4 are the indispensable working tools

Author(s): Paolo Busardo F.; Frati P.; Zaami S.; Fineschi V.

Source: International Journal of Molecular Sciences; Mar 2015; vol. 16 (no. 3); p. 6557-6570

Publication Date: Mar 2015

Publication Type(s): Review

Available in full text at International Journal of Molecular Sciences - from National Library of Medicine

Available in full text at International Journal of Molecular Sciences - from Free Access Content

Abstract: Amniotic fluid embolism (AFE) is an uncommon obstetric condition involving pregnant women during labor or in the initial stages after delivery. Its incidence is estimated to be around 5.5 cases per 100,000 deliveries. Therefore, this paper investigated the pathophysiological mechanism, which underlies AFE, in order to evaluate the role of immune response in the development of this still enigmatic clinical entity. The following databases (from 1956 to September 2014) Medline, Cochrane Central, Scopus, Web of Science and Science Direct were used, searching the following key words: AFE, pathophysiology, immune/inflammatory response, complement and anaphylaxis. The main key word "AFE" was searched singularly and associated individually to each of the other keywords. Of the 146 sources found, only 19 were considered appropriate for the purpose of this paper. The clinical course is characterized by a rapid onset of symptoms, which include: acute hypotension and/or cardiac arrest, acute hypoxia (with dyspnoea, cyanosis and/or respiratory arrest), coagulopathies (disseminated intravascular coagulation and/or severe hemorrhage), coma and seizures. The pathology still determines a significant morbidity and mortality and potential permanent neurological sequelae for surviving patients. At this moment, numerous aspects involving the pathophysiology and clinical development are still not understood and several hypotheses have been formulated, in particular the possible role of anaphylaxis and complement. Moreover, the detection of serum tryptase and complement components and the evaluation of fetal antigens can explain several aspects of immune response.

Database: EMBASE

15. Treatment of amniotic fluid embolism associated DIC in a labor patient with recombinant factor VIIa

Author(s): Aluyen J.N.; Li H.; Wen T.S.; Vadhera R.B.

Source: Anesthesia and Analgesia; Mar 2015; vol. 120 (no. 3)

Publication Date: Mar 2015

Publication Type(s): Conference Abstract

Available in full text at Anesthesia and Analgesia - from Ovid

Abstract:INTRODUCTION: Amniotic fluid embolism (AFE) is a rare syndrome that can complicate pregnancy and labor. It often has debilitating and lethal consequences. One serious sequela of AFE is disseminated intravascular coagulation (DIC). Presentation of a parturient with sudden cardiopulmonary arrest during labor, directly associated with amniotic fluid embolism with subsequent DIC which responded to treatment with Recombinant Factor VIIa. CASE REPORT: A laboring 32 year old G2P1 Hispanic woman was found unresponsive in her room with frothy discharge from her mouth. She was emergently intubated with subsequent forceps assisted vaginal delivery for fetal distress. After delivery, bleeding related to uterine atony was initially managed with fundal massage, pitocin, hemabate, and cytotec. Arterial and central venous catheters were placed for resuscitation and a bedside transthoracic echocardiogram showed adequate filling of the left

ventricle. Bleeding from the intravascular access sites as well as initial labs showing an elevated INR and low fibrinogen suggested DIC related to an AFE. She remained coagulopathic with persistent uterine bleeding despite multiple transfusions of PRBCs, FFP, and cryoprecipitate prompting ligation of the uterine arteries in the operating room. She received more blood products intraoperatively along with Recombinant Factor VIIa (1000 mcg). Repeat labs showed a normalized INR, coagulopathy improved, and vaginal bleeding stopped after ligation of the uterine arteries. She was discharged 17 days later from the ICU after full recovery with no residual neurological deficits. DISCUSSION: The routine use of recombinant activated factor VIIa in cases of massive hemorrhage is debatable but has been shown, in some cases, to reverse DIC and be successful. The use of recombinant activated factor VIIa should be considered in patients with massive obstetric hemorrhage in whom standard measures of stabilization are unsuccessful. In addition to all the traditional therapeutic means, Recombinant Factor VIIa may be an option for patients with amniotic fluid embolism associated DIC unresponsive to conventional treatment.

Database: EMBASE

16. Amniotic fluid embolism: The known and not known

Author(s): Benson M.D.

Source: Obstetric Medicine; 2014; vol. 7 (no. 1); p. 17-21

Publication Date: 2014

Publication Type(s): Article

Available in full text at Obstetric Medicine - from National Library of Medicine

Abstract: Amniotic fluid embolism was first recognized in 1926, in a Brazilian journal case report, on the basis of large amounts of fetal material in the maternal pulmonary vasculature at autopsy. The first English language description appeared in 1941 and consisted of eight parturients dying suddenly in which, once again, fetal material was seen in the pulmonary vasculature. A control group of 34 pregnant women dying of other recognized causes did not have fetal material in their lungs. The incidence of recognized, serious illness is on the order of two to eight per 100,000, with a mortality rate ranging from 13% to 35%. The diagnosis rests largely on one or more of four clinical signs: circulatory collapse, respiratory distress, coagulopathy, and seizures/ coma. The only confirmatory laboratory test remains autopsy findings although serum tests for fetal antigen, insulin-like growth factor binding protein-1, and complement are currently being investigated. One of the paradoxes of diagnosis is that fetal material in the pulmonary circulation at autopsy is specific for amniotic fluid embolism, while the same finding in the living is not. The mechanism of disease remains uncertain although the best available evidence suggests that complement activation might have a role. In contrast, mast cell degranulation probably is not a mechanism, so amniotic fluid embolism is not an anaphylaxis or anaphylactoid reaction as has been occasionally suggested. Perhaps the greatest unknown is not why 1 in 50,000 pregnant women develop what appears to be an immune response to their fetus, but rather why the other 49,999 do not?.

Database: EMBASE

17. Amniotic fluid embolism induces uterine anaphylaxis and atony following cervical laceration

Author(s): Nagai H.; Maeda H.; Kuroda R.-H.; Nakajima M.; Igarashi A.; Yoshida K.-I.; Tamura N.; Kanayama N.

Source: Gynecologic and Obstetric Investigation; 2014; vol. 78 (no. 1); p. 65-68

Publication Date: 2014

Publication Type(s): Article

Available in full text at Gynecologic and Obstetric Investigation - from ProQuest

Abstract: Amniotic fluid embolism (AFE) is a rare, high-risk obstetric complication primarily found in the lungs and potentially related to anaphylaxis. Tryptase release from the mast cell reflects anaphylaxis. Case report and findings: A female, aged over 40 years, presented with uterine atony and lethal hemorrhage after induced vaginal labor. Cervical laceration was accompanied by severe hemorrhage. Stromal edema and myometrial swelling were consistent with uterine atony. Alcian blue staining and zinc coproporphyrin immunostaining disclosed AFE, which was more prominent in the uterus than in the lungs. Tryptase immunostaining was diffuse and prominent around the activated mast cells (halos) in the uterus, including the cervix. Similar distribution of findings on the AFE markers, tryptase halos, complement receptor C5aR, and atony in the uterus suggested the causality of AFE to anaphylaxis, complement activation and atony. It is probable that disseminated intravascular coagulation (DIC), induced by AFE, uterine atony and cervical laceration, caused the lethal hemorrhage. It is likely that AFE, in association with cervical laceration, induces uterine anaphylaxis, complement activation, atony, DIC and lethal hemorrhage.

Database: EMBASE

18. Incidence, diagnosis and pathophysiology of amniotic fluid embolism

Author(s): Ito F.; Akasaka J.; Koike N.; Uekuri C.; Shigemitsu A.; Kobayashi H.

Source: Journal of Obstetrics and Gynaecology; Oct 2014; vol. 34 (no. 7); p. 580-584

Publication Date: Oct 2014

Publication Type(s): Article

Abstract: Amniotic fluid embolism (AFE) is a rare clinical entity, sometimes fatal. A review was conducted to describe the frequency, diagnosis and pathophysiology of AFE. The reported incidences ranged from 1.9 cases per 100,000 maternities (UK) to 6.1 per 100,000 maternities (Australia), which can vary considerably, depending on the period, region of study and the definition. Although the development of amniotic fluid-specific markers would have an impact on early diagnosis, definition of AFE based on these markers is not widely accepted. To date, immunological mechanisms, amniotic fluid-dependent anaphylactic reaction and complement activation, have been proposed as potential pathogenetic and pathophysiological mechanisms. Immune cell activation induced through complement activation may be associated with the mechanism that immediately initiates maternal death, only in susceptible individuals. This review will focus on advances in the field of AFE biology and discuss the prevalence, diagnosis and pathophysiology of AFE.

Database: EMBASE

19. Clinical characteristics of amniotic fluid embolism: an experience of 29 years.

Author(s): Yoneyama, Koichi; Sekiguchi, Atsuko; Matsushima, Takashi; Kawase, Rieko; Nakai, Akihito; Asakura, Hirobumi; Takeshita, Toshiyuki

Source: The journal of obstetrics and gynaecology research; Jul 2014; vol. 40 (no. 7); p. 1862-1870

Publication Date: Jul 2014

Publication Type(s): Research Support, Non-u.s. Gov't Multicenter Study Journal Article

Available in full text at Journal of Obstetrics and Gynaecology Research - from John Wiley and Sons

Abstract:AIMThe aim of this study was to elucidate the clinical characteristics and risk factors for amniotic fluid embolism (AFE).METHODSWe performed a retrospective case study analysis of patients using medical records and autopsy records. The diagnosis of AFE was based on the presence of clinical symptoms using Clark's criteria and autopsy results. We analyzed patient records from a 29-year period in three hospitals affiliated with the Nippon Medical School in Japan.RESULTSTen diagnoses of AFE were found in the records. First, we classified AFE patients into two types based on the initial presenting symptoms: post-partum hemorrhage and cardiopulmonary collapse. Fifty percent of the patients initially presented with post-partum hemorrhage and disseminated intravascular coagulation. Most were diagnosed with post-partum hemorrhage or uterine atony at AFE onset. Similarly, 50% presented with cardiopulmonary arrest or pulmonary arrest as initial symptoms, and most were diagnosed with eclampsia. Second, risk factors for AFE included advanced maternal age, multiparity, increased intrauterine pressure and disruptions of the uterine vasculature. Third, the case fatality rate was 70%. Fourth, squamous cells were observed in maternal central venous blood of five patients.CONCLUSIONAFE patients were classified into two types based on presenting signs and symptoms. Knowledge of the various initial symptoms of AFE enables a correct diagnosis.

Database: Medline

20. Amniotic fluid embolism: Pathophysiology and new strategies for management

Author(s): Kanayama N.; Tamura N.

Source: Journal of Obstetrics and Gynaecology Research; Jun 2014; vol. 40 (no. 6); p. 1507-1517

Publication Date: Jun 2014

Publication Type(s): Article

Available in full text at Journal of Obstetrics and Gynaecology Research - from John Wiley and Sons

Abstract: The registry program of amniotic fluid embolism (AFE) in Japan started in 2003. More than 400 hundred clinical diagnosed amniotic fluid embolism has been accumulated. Those data showed that there were two etiologies of AFE: the fetal materials create physical obstructions in the maternal microvessels in various organs, such as the lung; and (ii) the liquids cause an anaphylactoid reaction that leads to pulmonary vasospasm and activation of platelets, white blood cells and/or complements. The clinical findings showed that AFE was characterized mainly by cardiopulmonary collapse, the other involves the presence of disseminated intravascular coagulation (DIC) and atonic bleeding. Zinc coproporphyrin-1, sialyl Tn antigen (STN), complement C3, C4 and interleukin-8 have been used as serum markers of AFE. The levels of zinc coproporphyrin-1 and STN were increased in cardiopulmonary collapse type AFE, and a marked reduction of C3 and C4 was observed in DIC type AFE. At the primary medical institution, initial treatments for shock airway management, vascular management, fluid replacement, administration of anti-DIC therapy such as antithrombin, and administration of fresh frozen plasma should be provided. C1 esterase inhibitor activity in AFE cases was significantly lower than those of normal pregnant women. C1 esterase inhibitor may be a

promising candidate of treatment of AFE. © 2014 The Authors. Journal of Obstetrics and Gynaecology Research © 2014 Japan Society of Obstetrics and Gynecology.

Database: EMBASE

21. Amniotic fluid embolism

Author(s): Clark S.L.

Source: Obstetrics and Gynecology; Feb 2014; vol. 123 (no. 2); p. 337-348

Publication Date: Feb 2014

Publication Type(s): Article

Available in print at Patricia Bowen Library and Knowledge Service West Middlesex university Hospital - from Obstetrics and Gynecology

Available in full text at Obstetrics and Gynecology - from Ovid

Abstract: Amniotic fluid embolism remains one of the most devastating conditions in obstetric practice with an incidence of approximately 1 in 40,000 deliveries and a reported mortality rate ranging from 20% to 60%. The pathophysiology appears to involve an abnormal maternal response to fetal tissue exposure associated with breaches of the maternal-fetal physiologic barrier during parturition. This response and its subsequent injury appear to involve activation of proinflammatory mediators similar to that seen with the classic systemic inflammatory response syndrome. Progress in our understanding of this syndrome continues to be hampered by a lack of universally acknowledged diagnostic criteria, the clinical similarities of this condition to other types of acute critical maternal illness, and the presence of a broad spectrum of disease severity. Clinical series based on population or administrative databases that do not include individual chart review by individuals with expertise in critical care obstetrics are likely to both overestimate the incidence and underestimate the mortality of this condition by the inclusion of women who did not have amniotic fluid embolism. Data regarding the presence of risk factors for amniotic fluid embolism are inconsistent and contradictory; at present, no putative risk factor has been identified that would justify modification of standard obstetric practice to reduce the risk of this condition. Maternal treatment is primarily supportive, whereas prompt delivery of the mother who has sustained cardiopulmonary arrest is critical for improved newborn outcome. © 2014 by The American College of Obstetricians and Gynecologists.

Database: EMBASE

22. Amniotic fluid embolism: What level of scientific evidence can be drawn? a systematic review

Author(s): Frati P.; Zaami S.; Busardo F.P.; Foldes-Papp Z.

Source: Current Pharmaceutical Biotechnology; 2013; vol. 14 (no. 14); p. 1157-1162

Publication Date: 2013

Publication Type(s): Article

Abstract: Amniotic fluid embolism (AFE) is a rare and severe obstetric emergency and a significant cause of maternal mortality in developed countries and its incidence varies according to different studies. Presently, advances in the understanding of this pathology continue to be slowed down for the absence of generally accepted diagnostic criteria, the clinical analogies of this entity to other types of acute dangerous maternal illnesses and the presence of a wide range of disease severity. The aim of this review has been to evaluate the incidence of AFE, the role of possible risk factors, the clinical presentation (signs and symptoms) and outcome. Secondly the authors reviewed the management of these very difficult patients, including treatments and interventions in order to

extrapolate sharable recommendations for the management of these complicated patients. ©2013 Bentham Science Publishers.

Database: EMBASE

23. Incidence, risk factors, and consequences of amniotic fluid embolism.

Author(s): Kramer, Michael S; Abenhaim, Haim; Dahhou, Mourad; Rouleau, Jocelyn; Berg, Cynthia

Source: Paediatric and perinatal epidemiology; Sep 2013; vol. 27 (no. 5); p. 436-441

Publication Date: Sep 2013

Publication Type(s): Research Support, Non-u.s. Gov't Journal Article

Available in full text at Paediatric and Perinatal Epidemiology - from John Wiley and Sons

Abstract:BACKGROUNDAmniotic fluid embolism (AFE) is a rare but serious cause of maternal mortality whose aetiology remains obscure. Previous population-based studies have reported associations with labour induction and caesarean delivery.METHODSWe updated a previous analysis based on the US Nationwide Inpatient Sample from 1999 to 2008. We adapted a diagnostic validation algorithm to minimise false-positive diagnoses, along with statistical methods that account for the stratified random sampling design.RESULTSOf the 8 571 209 deliveries recorded in the database, 276 met our case definition of AFE, of which 62 (22.9% of the 274 with known vital status) were fatal. Significant associations with AFE were observed for medical induction {adjusted odds ratio [aOR] = 1.7 [95% confidence interval (CI) 1.2, 2.5]}, caesarean delivery [aOR = 15.0; 95% CI 9.4, 23.9], instrumental vaginal delivery [aOR = 6.6; 95% CI 4.0, 11.1], and cervical/uterine trauma [aOR = 7.4; 95% CI 3.6, 14.9]. AFE was associated with increases in risk of stillbirth, hysterectomy, maternal death, and prolonged maternal length of delivery hospital stay.CONCLUSIONSAFE remains an extremely serious obstetric complication with high risks of maternal and fetal mortality. The increased risks of AFE associated with labour induction and caesarean delivery have implications for elective use of these interventions.

Database: Medline

24. Acute hypotension associated with intraoperative cell salvage using a leukocyte depletion filter during management of obstetric hemorrhage due to amniotic fluid embolism.

Author(s): Rogers, William Kirke; Wernimont, Sarah A; Kumar, Girish C; Bennett, Eliza; Chestnut, David H

Source: Anesthesia and analgesia; Aug 2013; vol. 117 (no. 2); p. 449-452

Publication Date: Aug 2013

Publication Type(s): Case Reports Journal Article

Available in full text at Anesthesia and Analgesia - from Ovid

Abstract: Amniotic fluid embolism (AFE) is a rare but catastrophic obstetric complication that can lead to profound coagulopathy and hemorrhage. The role of cell salvage and recombinant human Factor VIIa (rFVIIa) administration in such cases remains unclear. We present a case of AFE and describe our experience with the use of cell salvage and rFVIIa administration during the resuscitation. Cell salvage and transfusion through a leukocyte depletion filter was attempted after the diagnosis of AFE was made, but the attempted transfusion was immediately followed by hypotension and a worsening of hemodynamics. rFVIIa, on the contrary, was used with clinical improvement in coagulopathy and without apparent adverse thrombotic effect.

Database: Medline

25. Pathogenesis and management of peripartum coagulopathic calamities (disseminated intravascular coagulation and amniotic fluid embolism).

Author(s): Levi, Marcel

Source: Thrombosis research; Jan 2013; vol. 131

Publication Date: Jan 2013

Publication Type(s): Journal Article

Abstract: Acute coagulopathic peripartum calamities are relatively rare but contribute importantly to maternal morbidity and mortality in the Western world. Abruptio placenta, amniotic fluid embolism, and retained fetal or placental material may lead to fulminant intravascular activation of coagulation which results in thromboembolic complications and consumption coagulopathy causing severe hemorrhage. The central underlying pathophysiological pathway in the coagulopathy associated with these syndromes is the occurrence of tissue factor, released from the placenta and amniotic fluid, in the circulation, in combination with low levels of physiological anticoagulant factors during pregnancy. The diagnosis of DIC may be made trough conventional composite scoring systems employing routine coagulation tests, whereas for the diagnosis of amniotic fluid embolism measurement of insulin like growth factor binding protein-1 seems promising. Therapy is aimed at removing the precipitating factor combined with supportive adjunctive treatment options.

Database: Medline

26. An overview of amniotic fluid embolism: Past, present and future directions

Author(s): Tsunemi T.; Oi H.; Sado T.; Naruse K.; Noguchi T.; Kobayashi H.

Source: Open Women's Health Journal; 2012; vol. 6 (no. 1); p. 24-29

Publication Date: 2012

Publication Type(s): Article

Abstract: Objective: A recent report has highlighted that amniotic fluid embolism (AFE) is the first among maternal mortality in Japan. The clinical presentation is not the same with respect to symptoms, timing and subsequent course. Methods: This article reviews the English language literature for pathophysiology on AFE based on the clinical and animal studies. Results: First, AFE syndrome may be divided into three subgroups designated the classical subtype, the anaphylactoid subtype and the DIC subtype, each having a distinct pattern of clinical symptoms and disease severity. Second, AFEassociated reactions can be classified as an anaphylactoid reaction or complement activation to fetal antigens or an idiosyncratic reaction. Host idiosyncrasy may be a major cause of hypersensitivity reaction. Third, the AFE reaction may be caused by a combination of immunologic and vasospastic factors. Finally, the development of effective markers for diagnosing entry of amniotic fluid into the maternal circulation would have an impact on early diagnosis and AFE-related mortality. Conclusion: This review summarizes new insights into the pathophysiology of AFE, with a focus on the potential direction of future research. © Tsunemi et al.

Database: EMBASE

27. Current concepts of immunology and diagnosis in amniotic fluid embolism.

Author(s): Benson, Michael D

Source: Clinical & developmental immunology; 2012; vol. 2012 ; p. 946576

Publication Date: 2012

Publication Type(s): Research Support, Non-u.s. Gov't Journal Article Review

Available in full text at Clinical and Developmental Immunology - from National Library of Medicine

Abstract: Amniotic fluid embolism (AFE) is one of the leading causes of maternal mortality and morbidity in developed countries. Current thinking about pathophysiology has shifted away from embolism toward a maternal immune response to the fetus. Two immunologic mechanisms have been studied to date. Anaphylaxis appears to be doubtful while the available evidence supports a role for complement activation. With the mechanism remaining to be elucidated, AFE remains a clinical diagnosis. It is diagnosed based on one or more of four key signs/symptoms: cardiovascular collapse, respiratory distress, coagulopathy, and/or coma/seizures. The only laboratory test that reliably supports the diagnosis is the finding of fetal material in the maternal pulmonary circulation at autopsy. Perhaps the most compelling mystery surrounding AFE is not why one in 20,000 parturients are afflicted, but rather how the vast majority of women can tolerate the foreign antigenic presence of their fetus both within their uterus and circulation?

Database: Medline

28. Amniotic fluid embolism: incidence, risk factors, and impact on perinatal outcome.

Author(s): Kramer, M S; Rouleau, J; Liu, S; Bartholomew, S; Joseph, K S; Maternal Health Study Group of the Canadian Perinatal Surveillance System

Source: BJOG : an international journal of obstetrics and gynaecology; Jun 2012; vol. 119 (no. 7); p. 874-879

Publication Date: Jun 2012

Publication Type(s): Research Support, Non-u.s. Gov't Journal Article

Available in full text at BJOG: An International Journal of Obstetrics and Gynaecology - from John Wiley and Sons

Abstract:OBJECTIVETo extend our previous work on AFE in Canada by including stricter criteria for case identification and by examining risks for stillbirth, neonatal mortality and serious maternal and neonatal morbidity.DESIGNPopulation-based cohort study.SETTINGCanada.POPULATION OR SAMPLEIn all, 4,508,462 hospital deliveries from fiscal year 1991/92 to 2008/09.METHODSTo reduce false-positive diagnoses, we restricted our analysis to AFE cases with cardiac arrest, shock or severe hypertension, respiratory distress, mechanical ventilation, coma, seizure, or coagulation disorder. Linkage of maternal and neonatal records, available since 2001/02, enabled us to examine the effects of AFE on neonatal outcomes. Detailed demographic and clinical data facilitated control for a broad array of potential confounding variables.MAIN OUTCOME MEASURESAmniotic fluid embolism, in-hospital neonatal death, asphyxia, mechanical ventilation, bacterial sepsis, seizure, nonimmune haemolytic or traumatic jaundice and length of hospital stay.RESULTSA total of 292 AFE cases were identified, of which only 120 (40%) were confirmed after applying our additional diagnostic criteria, yielding an AFE incidence of 2.5 per 100,000 deliveries. Of the 120 confirmed cases, 33 (27%) were fatal. Significant modifiable risk factors included medical induction, caesarean delivery, instrumental vaginal delivery, and uterine or cervical trauma. Amniotic fluid embolism was associated with significantly increased risks of stillbirth and neonatal asphyxia, mechanical ventilation, sepsis, seizures and prolonged length of hospital stay.CONCLUSIONSAmniotic fluid embolism remains a rare

but serious obstetric outcome, with several important modifiable risk factors and major implications for maternal, fetal and neonatal health.

Database: Medline

29. Can the presence of amniotic emboli in the myometrial vasculature be interpreted as a sign of amniotic fluid embolism?

Author(s): De l'Aulnoit A.H.; Deruelle P.; Petit S.; Devisme L.

Source: American Journal of Obstetrics and Gynecology; Jan 2012; vol. 206 (no. 1)

Publication Date: Jan 2012

Publication Type(s): Conference Abstract

Abstract:OBJECTIVE: Amniotic fluid embolism (AFE) is a life-threatening complication with a high maternal and neonatal mortality. Definitive diagnosis of AFE is based primarily on demonstrating the presence of fetal debris from the amniotic fluid within the pulmonary vasculature. However, there are more and more reports of patients surviving after prompt and aggressive therapy. In patients with clinical patterns of AFE needing hysterectomy, we observed amniotic fluid debris within the uterine vasculature. We hypothesized that these findings might be histopathological signs of AFE. To test this hypothesis, we analyzed the risk factors and clinical features associated with the presence or absence of amniotic emboli (AE) in the uterine circulation. STUDY DESIGN: A retrospective review of women who underwent peripartum hysterectomy was performed. Histopathological examination aimed to identify placenta accreta, fibrinocruoric thrombi, markers of disseminated intravascular coagulation or fetal debris in the myometrial vasculature. Characteristics that are associated with the presence of AE were examined. RESULTS: 36 patients were included in this study. Nine had intramyometrial vascular AE. When AE were present in myometrial vessels, the patients were more often primipara (44.4 vs. 7.4%, p 8 units of packed red cells (77.7 vs. 22.2%, p<0.01), fibrinogen (88.8 vs. 48.1%, p<0.04) and platelet units (66.6 vs. 18.5%, p<0.02). CONCLUSION: The diagnosis of AFE is currently limited. Our results suggested that histopathological examination of the uterus is an important key in the investigation to confirm AFE. In case of severe peripartum condition, the presence of AE in the myometrial vasculature would be an objective explanation for a possible expertise.

Database: EMBASE

30. Use of recombinant factor VIIa in patients with amniotic fluid embolism: A systematic review of case reports

Author(s): Leighton B.L.; Phillips L.E.; Wall M.H.; Lockhart E.M.; Zatta A.J.

Source: Anesthesiology; Dec 2011; vol. 115 (no. 6); p. 1201-1208

Publication Date: Dec 2011

Publication Type(s): Article

Available in full text at Anesthesiology. - from Ovid

Available in print at Patricia Bowen Library and Knowledge Service West Middlesex university Hospital - from Anesthesiology

Available in full text at Anesthesiology - from Free Access Content

Abstract:BACKGROUND: Patients with amniotic fluid embolism (AFE) (major cardiac and pulmonary symptoms plus consumptive coagulopathy) have high circulating tissue factor concentrations. Recombinant factor VIIa (rVIIa) has been used to treat hemorrhage in AFE patients even though rVIIa can combine with circulating tissue factor and form intravascular clots. A systematic review was

done of case reports from 2003 to 2009 of AFE patients with massive hemorrhage who were and were not treated with rVIIa to assess the thrombotic complication risk. METHODS: MEDLINE was searched for case reports of AFE patients receiving rVIIa (rVIIa cases) and of AFE patients who received surgery to control bleeding but no rVIIa (cohorts who did not receive rVIIa). Additional AFE case reports were obtained from the Food and Drug Administration, the Australian and New Zealand Haemostasis Registry, and scientific meeting abstracts. The risk of a negative outcome (permanent disability or death) in rVIIa cases versus cohorts who did not receive rVIIa was calculated using risk ratio and 95% confidence interval. RESULTS: Sixteen rVIIa cases and 28 cohorts were identified who did not receive rVIIa. All patients had surgery to control bleeding. Death, permanent disability, and full recovery occurred in 8, 6, and 2 rVIIa cases and 7, 4, and 17 cohorts who did not receive rVIIa (risk ratio 2.2, 95% CI 1.4-3.7 for death or permanent disability vs. full recovery). CONCLUSION: Recombinant factor VIIa cases had significantly worse outcomes than cohorts who did not receive rVIIa. It is recommended that rVIIa be used in AFE patients only when the hemorrhage cannot be stopped by massive blood component replacement.

Database: EMBASE

31. A noninvasive evaluation analysis of amniotic fluid embolism and disseminated intravascular coagulopathy

Author(s): Liao W.-C.; Jaw F.-S.

Source: Journal of Maternal-Fetal and Neonatal Medicine; Nov 2011; vol. 24 (no. 11); p. 1411-1415

Publication Date: Nov 2011

Publication Type(s): Article

Abstract: Objective. Amniotic fluid embolism (AFE) is a complication of pregnancy with a high mortality rate. The diagnosis of AFE is currently based on clinical findings, acute respiratory distress, cardiovascular collapse during labor and delivery, and immediate massive postpartum hemorrhaging. The serum biological markers are unreliable, and their detection requires a long time intervals for result. An early diagnosis is very important and prompts the clinical management of the condition. Study design and results.We present here a noninvasive method (time frequency, entropy) to analyze heart rate variability (HRV). 3D-Spectrogram and entropy were derived from the RR interval of two pregnant subjects with AFE and disseminated intravascular coagulation (DIC) survived after delivering normal babies admitted to the Taiwan Seventh-day Adventists Hospital, and the entropy values were compared with those of 105 healthy pregnant subjects in the same hospital. Conclusions. We show that these methods can be successfully applied to the diagnosis of AFE and predict the prognosis of DIC. We also show that CT scans can be applied to the diagnosis of pulmonary embolism, eliminating the need to pathology. © 2010 Informa UK, Ltd.

Database: EMBASE

32. Activation of coagulation in amniotic fluid during normal human pregnancy.

Author(s): Sarig, Galit; Klil-Drori, Adi J; Chap-Marshak, Dafna; Brenner, Benjamin; Drugan, Arieh

Source: Thrombosis research; Nov 2011; vol. 128 (no. 5); p. 490-495

Publication Date: Nov 2011

Publication Type(s): Journal Article

Abstract:INTRODUCTIONAmniotic fluid (AF) is an important medium for fetal development which exhibits high procoagulant activities; however, the role of these procoagulants during pregnancy has not been elucidated and might be associated with pregnancy complications. The current study aimed to evaluate factor X (FX) activation and its association with tissue factor (TF), tissue factor pathway inhibitor (TFPI) and coagulation activation markers in AF during normal human pregnancy.METHODSActivation of FX and concentration of TF, free TFPI, D-dimer and prothrombin fragments (F1+2) were evaluated in AF samples obtained for chromosome analysis from 91 women with normal pregnancy: 65 samples were taken from patients at 16-20 weeks of gestation, 9 samples were drawn at 21-30 weeks and 17 samples--after 30 weeks of gestation.RESULTSActivation of FX in AF significantly increased during normal pregnancy (from 65±41 to 205±80 equivalent RVV ng/mg total protein, P<0.0001). TF and TFPI levels in AF also rose with gestational age. In contrast, the AF concentration of D-dimer and F1+2, markers of coagulation activation significantly decreased when expressed per mg total protein. Levels of free TFPI correlated with TF (r=0.5, P<0.001), and were 8-fold higher than those of TF during pregnancy.CONCLUSIONHigh levels of TFPI might be associated with the inhibition of procoagulant activity in amniotic fluid during normal pregnancy, which may account for the rarity of clinical amniotic fluid embolism.

Database: Medline

33. Incidence and risk factors for amniotic-fluid embolism

Author(s): Knight M.; Tuffnell D.; Brocklehurst P.; Spark P.; Kurinczuk J.J.

Source: Obstetrical and Gynecological Survey; Sep 2010; vol. 65 (no. 9); p. 547-548

Publication Date: Sep 2010

Publication Type(s): Note

Available in full text at Obstetrical & gynecological survey. - from Ovid

Database: EMBASE

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

Author(s): Oi H.; Naruse K.; Noguchi T.; Sado T.; Kobayashi H.; Kimura S.; Kanayama N.; Terao T.

Source: Gynecologic and Obstetric Investigation; Aug 2010; vol. 70 (no. 2); p. 138-144

Publication Date: Aug 2010

Publication Type(s): Article

Available in full text at Gynecologic and Obstetric Investigation - from ProQuest

Abstract: Aims: To identify factors leading to fatality of patients with amniotic fluid embolism (AFE). Methods: Patients who had fatal or nonfatal AFE were registered at the Hamamatsu University School of Medicine in the Department of Obstetrics and Gynecology from 1992 to 2006. Data collected included information about demographics and clinical characteristics. The fatal factors among these data were identified using chi2 analysis and the Mann-Whitney test. Results: One hundred and thirty-five patients met the criteria, which included fatal (n = 65) and nonfatal AFE (n = 70). Maternal full-term gestational weeks, multiparous and noncesarean sections were the risk factors for death found in this study ($p < 0.01$). Sialyl Tn levels (mean +/- SD) in the serum of patients with fatal AFE (69.7+/- 126.4 U/ml) were higher compared to those with nonfatal AFE (48.3+/- 161.8 U/ml; $p = 0.003$). Each of three items (cardiac arrest, dyspnea or loss of consciousness) was more common in fatal AFE ($p < 0.01$). Maternal pregnancy and labor complications were not associated with the distinction between fatal and nonfatal AFE. Conclusion: Factors associated with patients with fatal AFE were identified. These included multiparity, noncesarean section at full-term and the three symptoms mentioned above. Sialyl Tn levels could be a possible prognostic fatality factor. © 2010 S. Karger AG, Basel.

Database: EMBASE

35. Amniotic fluid embolism: an evidence-based review

Author(s): Conde-Agudelo A.; Romero R.

Source: American Journal of Obstetrics and Gynecology; Nov 2009; vol. 201 (no. 5); p. 445

Publication Date: Nov 2009

Publication Type(s): Review

Abstract: We reviewed the best evidence on 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 increased risk of AFE include advanced maternal age, placental abnormalities, operative deliveries, eclampsia, polyhydramnios, cervical laceration, and uterine rupture. Hemodynamic response to 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. © 2009.

Database: EMBASE

36. Factor VIIa treatment of DIC as a clinical manifestation of amniotic fluid embolism in a patient with fetal demise.

Author(s): Kahyaoglu, Inci; Kahyaoglu, Serkan; Mollamahmutoglu, Leyla

Source: Archives of gynecology and obstetrics; Jul 2009; vol. 280 (no. 1); p. 127-129

Publication Date: Jul 2009

Publication Type(s): Case Reports Journal Article

Available in full text at Archives of Gynecology and Obstetrics - from Springer Link Journals

Abstract:INTRODUCTIONA pregnant patient, with term intrauterine fetal demise, who developed cardiopulmonary arrest during labor, followed by disseminated intravascular coagulation (DIC) secondary to amniotic fluid embolism (AFE) that was treated with Recombinant Factor VIIa, is presented.CASE REPORTA 22-year-old Turkish woman was admitted to our antenatal clinic at 39 weeks 6 days of gestation with a complaint of decreased fetal movements for the previous 3 days. Shortly after presentation, she was noted to have circumoral cyanosis with shortness of breath and sudden loss of consciousness. After a 3,220 g macerated male fetus was delivered, persistent bleeding occurred in the mother and was managed with Recombinant Factor VIIa at a dose of 90 mcg/kg. She died 8 days after the admission due to multiple organ failure.CONCLUSIONRecombinant Factor VIIa may be a treatment option for hemorrhage in patients with DIC related to AFE.

Database: Medline

37. Incidence and risk factors of amniotic fluid embolisms: a population-based study on 3 million births in the United States

Author(s): Abenhaim H.A.; Leduc L.; Azoulay L.; Kramer M.S.

Source: American Journal of Obstetrics and Gynecology; Jul 2008; vol. 199 (no. 1); p. 49

Publication Date: Jul 2008

Publication Type(s): Article

Abstract: Objective: Amniotic fluid embolism (AFE) is a condition occurring during delivery that can lead to severe maternal morbidity and mortality. Given the rarity of its occurrence, current estimates and predictors of the incidence and outcomes are often difficult to obtain. Study Design: We conducted a population-based cohort study on 3 million birth records in the Healthcare Cost and Utilization Project-Nationwide Inpatient Sample from 1999 to 2003 to estimate the incidence and case fatality of AFEs. Logistic regression was used to calculate the odds ratio (OR) and corresponding 95% confidence intervals (CIs) of demographic and obstetrical determinants of AFEs and fatal AFEs. Results: The overall incidence of AFE was 7.7 per 100,000 births (95% CI 6.7 to 8.7), with a case fatality rate of 21.6% (95% CI 15.5 to 27.6%). AFE was associated with maternal age greater than 35 (OR 2.2, 95% CI 1.5 to 2.1), placenta previa (OR 30.4, 95% CI 15.4 to 60.1), and cesarean delivery (OR 5.7, 95% CI 3.7 to 8.7). Although AFEs were not significantly associated with induction of labor (OR 1.5, 95% CI 0.9 to 2.3), they were associated with preeclampsia, abruptio placentae, and the use of forceps. Among women with an AFE, common demographic or obstetrical determinants were not predictive of maternal mortality. Conclusion: AFE is a rare but serious condition that is associated with advanced maternal age, placental pathologies, and cesarean deliveries. Further research on the treatment of this condition is necessary.

Database: EMBASE

38. Suspected amniotic fluid embolism following amniotomy: a case report.

Author(s): Mato, Jampierre

Source: AANA journal; Feb 2008; vol. 76 (no. 1); p. 53-59

Publication Date: Feb 2008

Publication Type(s): Case Reports Journal Article Review

Available in full text at AANA Journal - from EBSCOhost

Available in full text at AANA Journal - from ProQuest

Abstract: Amniotic fluid embolism (AFE), also referred to as anaphylactoid syndrome of pregnancy, is a rare obstetric emergency that may manifest itself at any time during pregnancy. AFE is believed to occur when the constituents of amniotic fluid enter the maternal circulation, leading to varying degrees of multiorgan compromise. AFE was first described in 1926, gaining widespread recognition in 1941. This article describes the pathogenesis of AFE, including theories of its immunological mediation available in the literature. The most current diagnostic and treatment modalities are discussed, including several novel therapies. A case report of a 40-year-old parturient who suffered probable AFE following amniotomy, with the development of cardiopulmonary compromise, neurologic involvement, fetal distress, and coagulopathy, is outlined. The patient survived emergency cesarean delivery and hysterectomy with no residual physiologic deficits.

Database: Medline

39. A hypothesis regarding complement activation and amniotic fluid embolism

Author(s): Benson M.D.

Source: Medical Hypotheses; 2007; vol. 68 (no. 5); p. 1019-1025

Publication Date: 2007

Publication Type(s): Article

Abstract: Amniotic fluid embolism, a rare, sudden and often fatal illness of pregnancy may not be a true embolic event resulting from the physical obstruction of the pulmonary vasculature. The high degree of variability in symptoms, the lack of characteristic findings on radiological exam, the absence of a dose-response effect on symptoms, and the occasional occurrence of coagulopathies are not entirely consistent with a physical block to the circulation as the main mechanism of disease. Alternatively, it might be the result of complement activation initiated by fetal antigen leaking into the maternal circulation. This rare immune response may be initiated by a rare pathological antigen, or by common antigens presented uncommonly-in amount, timing, or frequency of entry into the maternal circulation. Some very early evidence in AFE patients supports this hypothesis but is not conclusive. Complement levels remain well within the normal range during uncomplicated parturition. A prior theory that AFE might be a result of maternal anaphylaxis to fetal antigen has much less evidence to support it. The disseminated intravascular coagulation often seen in this and other serious obstetrical illnesses may be a secondary result of complement activation rather than the direct introduction of pro-coagulants into the maternal circulation although the link between the complement and coagulation pathways, if any, remains poorly defined. Through currently available laboratory testing, both the complement hypothesis and the anaphylaxis mechanism are able to be assessed. Direct measurement of serum complement as well as serum tryptase and urinary histamine are readily obtained tests in community hospitals as well as tertiary care hospitals. If the hypothesis proves true, this investigation may be of profound importance to understanding immune tolerance. Rather, than asking why one pregnant woman in 20,000 develops a violent immune reaction to the fetus, a better question is why do not all pregnant women reject the fetus which is a large collection of foreign antigens?

Database: EMBASE

40. Amniotic fluid embolism after blunt abdominal trauma.

Author(s): Ellingsen, Christian Lycke; Eggebø, Torbjørn Moe; Lexow, Kristian

Source: Resuscitation; Oct 2007; vol. 75 (no. 1); p. 180-183

Publication Date: Oct 2007

Publication Type(s): Case Reports Journal Article

Abstract: Amniotic fluid embolism (AFE) is a rare, but potentially fatal complication of pregnancy, with an incidence between 1 in 8000 and 1 in 80,000 pregnancies. The pathogenesis is not fully understood, but the generally accepted belief is that amniotic fluid enters the mother's circulation, most commonly via tears in the lower uterine segment. In the fluid there are substances with pro-inflammatory, vasospastic and pro-coagulative properties. AFE after blunt trauma is very rare, only described a few times in the literature. We report a case of fatal AFE after probable minor blunt trauma to the abdomen and give a review of the literature.

Database: Medline

41. Amniotic fluid embolism after surgical trauma: two case reports and review of the literature.

Author(s): Pluymakers, Christine; De Weerdt, Annick; Jacquemyn, Yves; Colpaert, Cecile; Van de Poel, Els; Jorens, Philippe G

Source: Resuscitation; Feb 2007; vol. 72 (no. 2); p. 324-332

Publication Date: Feb 2007

Publication Type(s): Case Reports Journal Article

Abstract: Amniotic fluid embolism (AFE) is a relatively rare condition usually occurring during or shortly after pregnancy and is catastrophic in most cases. The classical description is a sudden onset of dyspnoea, cyanosis and hypotension out of proportion to the blood loss, followed quickly by cardiorespiratory arrest. Up to 20% of patients will have seizures and up to 40% will have consumptive coagulopathy. If the patient survives the initial phase, a non-cardiogenic pulmonary oedema will follow in up to 70% of all cases. We report on two cases of severe and near fatal amniotic fluid embolism during pregnancy. Surgical trauma, caused by a blow in the stomach and a surgical intervention, was considered to be the aetiology.

Database: Medline

42. Placenta previa and accreta complicated by amniotic fluid embolism.

Author(s): Mathelier, Amedee C; Karachorlu, Kirkor

Source: International journal of fertility and women's medicine; 2006; vol. 51 (no. 1); p. 28-32

Publication Date: 2006

Publication Type(s): Case Reports Journal Article

Abstract:BACKGROUNDThe simultaneous occurrence of placenta previa and placenta accreta in patients who had previous low transverse cesarean delivery is presently well established. However, the sequence of previous cesarean delivery followed by placenta previa and accreta in a patient who also experiences a premature rupture of membranes as well as amniotic fluid embolism (AFE) is a rare obstetric phenomenon.CASEA 24-year-old woman, para 2 with two previous cesarean deliveries, at 32 weeks' gestation by last menstrual period, was admitted with premature rupture of

membranes. A repeat cesarean delivery (CD) was done. Excessive hemorrhage occurred, necessitating a hysterectomy. Also, the patient developed an amniotic fluid embolism.CONCLUSIONPlacenta previa and placenta accreta may be observed in patients who have a previous CD scar and in whom AFE develops suddenly and unexpectedly. AFE, a condition with complex pathogenesis, presents a number of challenges, with the patient undergoing serious complications that may include massive hemorrhage, disseminated intravascular coagulopathy, and death. The obstetrician should be alert to the symptoms of AFE, and if they occur should begin prompt and aggressive treatment.

Database: Medline

43. Amniotic-fluid embolism and medical induction of labour: a retrospective, population-based cohort study.

Author(s): Kramer, Michael S; Rouleau, Jocelyn; Baskett, Thomas F; Joseph, K S; Maternal Health Study Group of the Canadian Perinatal Surveillance System

Source: Lancet (London, England); Oct 2006; vol. 368 (no. 9545); p. 1444-1448

Publication Date: Oct 2006

Publication Type(s): Research Support, Non-u.s. Gov't Journal Article

Available in full text at Lancet, The - from ProQuest

Available in print at Patricia Bowen Library and Knowledge Service West Middlesex university Hospital - from The Lancet

Abstract:BACKGROUNDAmniotic-fluid embolism is a rare, but serious and often fatal maternal complication of delivery, of which the cause is unknown. We undertook an epidemiological study to investigate the association between amniotic-fluid embolism and medical induction of labour.METHODSWe used a population-based cohort of 3 million hospital deliveries in Canada between 1991 and 2002 to assess the associations between overall and fatal rates of amniotic-fluid embolism and medical and surgical induction, maternal age, fetal presentation, mode of delivery, and pregnancy and labour complications.FINDINGSTotal rate of amniotic-fluid embolism was 14.8 per 100,000 multiple-birth deliveries and 6.0 per 100,000 singleton deliveries (odds ratio 2.5 [95% CI 0.9-6.2]). Of the 180 cases of amniotic-fluid embolism in women with singleton deliveries during the study period, 24 (13%) were fatal. We saw no significant temporal increase in occurrence of amniotic-fluid embolism for total or fatal cases. Medical induction of labour nearly doubled the risk of overall cases of amniotic-fluid embolism (adjusted odds ratio 1.8 [1.3-2.7]), and the association was stronger for fatal cases (crude odds ratio 3.5 [1.5-8.4]). Maternal age of 35 years or older, caesarean or instrumental vaginal delivery, polyhydramnios, cervical laceration or uterine rupture, placenta previa or abruption, eclampsia, and fetal distress were also associated with an increased risk.INTERPRETATIONMedical induction of labour seems to increase the risk of amniotic-fluid embolism. Although the absolute excess risk is low, women and physicians should be aware of this risk when making decisions about elective labour induction.

Database: Medline

44. Amniotic fluid embolism with isolated coagulopathy: A case report

Author(s): Yang J.-I.; Kim H.-S.; Chang K.-H.; Ryu H.-S.; Joo H.-J.

Source: Journal of Reproductive Medicine for the Obstetrician and Gynecologist; Jan 2006; vol. 51 (no. 1); p. 64-66

Publication Date: Jan 2006

Publication Type(s): Article

Abstract:BACKGROUND: Amniotic fluid embolism is a life-threatening complication of pregnancy accompanied by a high mortality rate. The common clinical presentation is sudden onset of dyspnea, hypotension inappropriate to the volume of blood loss, and hypoxia, followed by cardiopulmonary arrest. Recently, cases of amniotic fluid embolism with isolated coagulopathy as an atypical presentation have been reported. CASE: A 27-year-old multigravida presented with continuous postpartum oozing after an uneventful vaginal delivery at 38 weeks of gestation. Laboratory evidence revealed disseminated intravascular coagulopathy. Despite good uterine contractions and massive blood component therapy, vaginal bleeding continued and finally led to emergency laparotomy. Histopathologic examination showed a deep cervical laceration in the endocervix, and multiple areas of amniotic fluid debris were demonstrated in the laceration site vasculature of the endocervix. After hysterectomy, the patient recovered fully, without sequelae. CONCLUSION: This case represents atypical symptoms and signs: clinical hemorrhage in the initial presentation rather than the classical pattern of cardiopulmonary collapse. In cases of suspected amniotic fluid embolism with an atypical presentation, a thorough histologic examination of the uterus, including the cervix, is critical to making the diagnosis of amniotic fluid embolism. © Journal of Reproductive Medicine, Inc.

Database: EMBASE

45. The syndrome of amniotic fluid embolism: a potential contribution of bradykinin.

Author(s): Robillard, Josée; Gauvin, France; Molinaro, Giuseppe; Leduc, Line; Adam, Albert; Rivard, Georges E

Source: American journal of obstetrics and gynecology; Oct 2005; vol. 193 (no. 4); p. 1508-1512

Publication Date: Oct 2005

Publication Type(s): Research Support, Non-u.s. Gov't Case Reports Journal Article

Abstract:OBJECTIVEAmniotic fluid embolism is a potentially fatal complication of pregnancy; although several hypotheses have been formulated, the pathophysiology of this condition is not well known. An exaggerated release of bradykinin, which is activated by products of the amniotic fluid that enter the maternal circulation, could explain the symptoms that are present in amniotic fluid embolism. The objective of this study was to assess whether bradykinin is involved in amniotic fluid embolism.STUDY DESIGNThe plasma bradykinin-generating capacity was measured serially in a patient who experienced amniotic fluid embolism.RESULTSThe plasma bradykinin-generating capacity was found to be very low at the time of the initial clinical manifestations, which were characterized by severe hypotension, cardiorespiratory arrest, and coagulopathy.CONCLUSIONThis study suggests a potential role for bradykinin release in the pathophysiology of amniotic fluid embolism.

Database: Medline

46. Presumed antepartum amniotic fluid embolism.

Author(s): Kent, Kristen J; Cooper, Brian C; Thomas, Karl W; Zlatnik, Frank J

Source: Obstetrics and gynecology; Sep 2003; vol. 102 (no. 3); p. 493-495

Publication Date: Sep 2003

Publication Type(s): Case Reports Journal Article

Available in print at Patricia Bowen Library and Knowledge Service West Middlesex university Hospital - from Obstetrics and Gynecology

Available in full text at Obstetrics and Gynecology - from Ovid

Abstract:BACKGROUNDAmniotic fluid embolism is seldom recognized in nonperipartum patients. The pathophysiology is uncertain and diagnosis imprecise, making management after stabilization difficult.CASEA 37-year-old woman at 28 weeks' gestation presented with signs and symptoms consistent with amniotic fluid embolism including disseminated intravascular coagulopathy. A ventilation-perfusion scan demonstrated unmatched perfusion defects, but other radiographic studies were negative; the patient was treated with heparin. Four days after presentation she had spontaneous rupture of membranes followed by hypoxemia, necessitating cesarean delivery. A pulmonary arteriogram after the operation showed multiple filling defects; the patient was discharged on warfarin.CONCLUSIONAmniotic fluid embolism is a difficult diagnosis to make, at best. Anticoagulation may be a therapeutic option.

Database: Medline

47. Amniotic fluid embolism and isolated coagulopathy: atypical presentation of amniotic fluid embolism.

Author(s): Awad, I T; Shorten, G D

Source: European journal of anaesthesiology; Jun 2001; vol. 18 (no. 6); p. 410-413

Publication Date: Jun 2001

Publication Type(s): Case Reports Journal Article

Abstract:A 41-year-old multigravida presented at 32 weeks of gestation with polyhydramnios and an anencephalic fetus. Abnormal bleeding as a result of disseminated intravascular coagulation complicated an emergency Caesarean section for severe abdominal pain thought to be due to uterine rupture. Massive transfusion with blood products was necessary and the abdomen packed to control bleeding. The patient was transferred to the intensive care unit where she made a slow but complete recovery. Amniotic fluid embolism with atypical presentation of isolated coagulopathy is the likely diagnosis in this case. The case serves to demonstrate that amniotic fluid embolism may present with symptoms and signs other than the classical pattern of dyspnoea, cyanosis and hypotension.

Database: Medline

48. Amniotic fluid embolism in a patient with SC sickle cell disease.

Author(s): Sanders, G M

Source: Anaesthesia; Jun 1999; vol. 54 (no. 6); p. 614-616

Publication Date: Jun 1999

Publication Type(s): Letter Case Reports

Available in full text at Anaesthesia - from John Wiley and Sons

Database: Medline

49. Amniotic fluid embolism following blunt abdominal trauma in pregnancy.

Author(s): Judich, A; Kuriansky, J; Engelberg, I; Haik, J; Shabtai, M; Czerniak, A

Source: Injury; Jul 1998; vol. 29 (no. 6); p. 475-477

Publication Date: Jul 1998

Publication Type(s): Case Reports Journal Article

Available in print at Patricia Bowen Library and Knowledge Service West Middlesex university Hospital - from Injury

Database: Medline

50. Amniotic fluid embolism associated with castor oil ingestion.

Author(s): Steingrub, J S; Lopez, T; Teres, D; Steingart, R

Source: Critical care medicine; Jun 1988; vol. 16 (no. 6); p. 642-643

Publication Date: Jun 1988

Publication Type(s): Case Reports Journal Article

Abstract: We report a case of an amniotic fluid embolism (AFE) causing a cardiorespiratory arrest associated temporally with ingestion of castor oil in a full-term normal pregnancy. Risk factors usually associated with AFE were not found in this patient.

Database: Medline

Strategy 220462

#	Database	Search term	Results
2	Medline	exp "EMBOLISM, AMNIOTIC FLUID"/	1011
3	Medline	("amniotic fluid embol*").ti,ab	934
4	Medline	(2 OR 3)	1237
5	Medline	((fetus OR foetal OR fetal) ADJ3 (haemorrhag* OR hemorrhag* OR bleed*)).ti,ab	1148
6	Medline	(4 AND 5)	7
7	Medline	exp "FETOMATERNAL TRANSFUSION"/	1176
8	Medline	(4 AND 7)	3
9	Medline	exp "UTERINE HEMORRHAGE"/	19372
10	Medline	(4 AND 9)	123
11	EMBASE	*"AMNION FLUID EMBOLISM"/	839
12	EMBASE	exp "FETOMATERNAL TRANSFUSION"/	12360
13	EMBASE	(11 AND 12)	2
14	EMBASE	exp "DISSEMINATED INTRAVASCULAR CLOTTING"/	20981
15	EMBASE	(12 AND 14)	21
16	EMBASE	exp "FETAL HEMORRHAGE"/	297
17	EMBASE	(11 AND 16)	0
18	EMBASE	exp "AMNION FLUID	1491

		EMBOLISM"/	
19	EMBASE	(16 AND 18)	3
20	EMBASE	(11 AND 14)	194
21	EMBASE	exp "ANTEPARTUM HEMORRHAGE"/	1228
22	EMBASE	(11 AND 21)	2
23	EMBASE	*"AMNION FLUID EMBOLISM"/et	148
24	EMBASE	exp SYMPTOMATOLOGY/	853026
25	EMBASE	(11 AND 24)	111
26	EMBASE	exp "FETUS DEATH"/	35664
27	EMBASE	(11 AND 26)	33
28	EMBASE	(12 AND 14 AND 18)	2
29	EMBASE	(haemorrhag* OR hemorrhag*).ti,ab	286175
30	EMBASE	(11 AND 29)	99
31	EMBASE	(12 AND 18)	9
32	EMBASE	exp "AMNION FLUID"/	21776
33	EMBASE	(12 AND 32)	513
34	Medline	(symptom*).ti,ab	909619
35	Medline	(4 AND 34)	79
36	Medline	exp "FETAL DEATH"/	27582
37	Medline	(4 AND 36)	75
38	Medline	exp "EMBOLISM, AMNIOTIC FLUID"/et,pp	267

39	Medline	exp ANEMIA/	149786
40	Medline	(4 AND 39)	9
41	EMBASE	exp ANEMIA/	315987
42	EMBASE	(11 AND 41)	9