Embolie Graisseuse post traumatique
Introduction

• Dg difficile
• Définition
  – Obstruction des capillaires par gouttelettes graisseuses au niveau
    – Pulmonaire
    – Systémique
  – Syndrome embolie graisseuse
Circonstance d’apparition

• Embolie graisseuse
  – 1°) avec traumatisme
    • Autopsie pulmonaire 90%
      – Dépend du nombre et de la localisation des fractures

  – 2°) sans traumatisme
    • Autopsie 6%

• Syndrome d’embolie graisseuse
  – Fréquence SEG :
    • 0.3% à 17% ; mortalité de 1 à 20%
Bouffard Y, Guillaume C, Perrot D, Delafosse B, Motin J.

Between 1977 and 1982, fifty cases of post-traumatic fat embolism were treated in a general intensive care unit. Average age of patients was 25.5 +/- 13 years; there was no male majority. Mean free interval was 39 +/- 27 h. 12 cases (24%) had single fractures and 38 (76%) multiple fractures. Forty-four patients had a fractured femur. Thirty-two patients presented the complete clinical syndrome with general, respiratory, neurological and cutaneous signs. Thrombocytopaenia and hypocholesterolaemia were the biological signs most often seen. In forty-four patients, orthopaedic treatment consisted of immediate immobilization, usually with traction. Twenty-six patients were reoperated on: intramedullary nail for twenty patients, plate for the other six. Fat embolism appeared in spite of surgery in six cases; it worsened after surgery in six others. Seven patients had per- or postanaesthetic problems. Fourteen per cent of patients died. The decrease in mortality was mainly due to an improvement in mechanical ventilation techniques. Early surgical fixation remained the rule if there was no serious respiratory distress or haemodynamic instability, although it did not seem to change the mortality rate in this group of patients.
Fréquence embolie graisseuse
Diagnostic de SEG

- **Dg** : 3 critères principaux associés à 4 critères secondaires et à une macroglobulinémie lipidique

  C Forster  Forum Suisse Med 2002

- **Critères principaux**
  - Signes respiratoires
  - Signes neurologiques
  - Signe cutanés

- **Critères secondaires**
  - Tachycardie, fièvre, FO, ictère, trouble rénaux

- **Laboratoires**
  - Thrombopénie, anémie, VS, macroglobulinémie lipidique
<table>
<thead>
<tr>
<th>Critères principaux</th>
<th>Pétéchies</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Troubles respiratoires: tachypnée, dyspnée, râles inspiratoires bilatéraux, hémoptysies, opacités diffuses bilatérales à la radiographie du thorax</td>
</tr>
<tr>
<td></td>
<td>Troubles neurologiques: confusion, obnubilation, coma</td>
</tr>
<tr>
<td>Critères secondaires</td>
<td>Tachycardie (&gt;120/min)</td>
</tr>
<tr>
<td></td>
<td>Fièvre &gt;39,4°C</td>
</tr>
<tr>
<td></td>
<td>Lésions de la rétine (corps gras ou hémorragies, v. fig. 2)</td>
</tr>
<tr>
<td></td>
<td>Ictère</td>
</tr>
<tr>
<td></td>
<td>Troubles rénaux (anurie ou oligurie)</td>
</tr>
<tr>
<td>Laboratoire</td>
<td>Thrombopénie (plaquettes &lt;150 × 10⁹/l)</td>
</tr>
<tr>
<td></td>
<td>Hémoglobine abaissée (&gt;20% de la valeur initiale)</td>
</tr>
<tr>
<td></td>
<td>Vitesse de sédimentation &gt;71 mm/h</td>
</tr>
<tr>
<td></td>
<td>Macroglobulinémie lipidique</td>
</tr>
</tbody>
</table>
# Fréquence des symptômes

<table>
<thead>
<tr>
<th>Signes cliniques</th>
<th>Forster</th>
<th>Bouffard</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoxie</td>
<td>96%</td>
<td>76%</td>
</tr>
<tr>
<td>Tachycardie &gt; 120</td>
<td>93%</td>
<td>82%</td>
</tr>
<tr>
<td>Fièvre &gt; 39°C</td>
<td>70%</td>
<td>66%</td>
</tr>
<tr>
<td>Anémie (\downarrow) Hb</td>
<td>67%</td>
<td>46%</td>
</tr>
<tr>
<td>Altération conscience</td>
<td>59%</td>
<td>88%</td>
</tr>
<tr>
<td>Thrombopénie &lt; 150</td>
<td>37%</td>
<td>90%</td>
</tr>
<tr>
<td>Pétéchie</td>
<td>33%</td>
<td>88%</td>
</tr>
<tr>
<td>Hémolyse</td>
<td></td>
<td>16%</td>
</tr>
</tbody>
</table>
Figure 2.
Fond d’œil dans un syndrome d’embolie graisseuse (rétinopathie de Purtscher).
Biologie : lipides

• 1°) diminution
  – Phospholipides, cholestérol

• 2°) augmentation
  – Phospholipase A2, lipase sérique
  – AGL, triglycérides
Post-traumatic blood lipid changes and fat embolism. Relation of post-traumatic blood lipid changes and fat embolism syndrome.

Lepisto PV.

Serum cholesterol, triglycerides, phospholipids, and lipoprotein levels were studied by electrophoresis in 43 trauma patients with multiple fractures of the lower extremities and pelvis. Ten healthy volunteers were used for control studies. The fat embolism syndrome was diagnosed in eight of the 43 patients. The lipid values of all the trauma patients were lower than the control values. A decrease in cholesterol and phospholipids was observed in the fat embolism patients during 4 days; these changes were not observed in the other trauma patients. The triglycerides of all the trauma patients increased slowly over a period of 4 days. Changes in the lipoprotein fractions after the trauma were minor. An early increase in the alpha1 lipoprotein fraction was noted in the fat embolism patients simultaneously with a decrease in the pre-beta lipoprotein fraction; the values of the other trauma patients were at the control levels. Normalization was observed in 6 hours. The lipid concentrations of plasma and serum did not change with filtration with 1.2, 5, and 8mum Millipore filters. No differences were noted between the femoral and cubital vein samples.
LBA et inclusions lipidiques

• LBA et inclusions lipidiques
  – Spécifique
    • Chastre Ann Intern Med 1990
  – Non spécifique
    • JM Vedrinne, C Guillaume Chest 1992
Bronchoalveolar lavage for rapid diagnosis of the fat embolism syndrome in trauma patients.


Hopital Bichat, Paris, France.

OBJECTIVE: To evaluate the usefulness of bronchoalveolar lavage in establishing the diagnosis of the fat embolism syndrome in trauma patients with long-bone fractures. DESIGN: Case series. SETTING: Referral hospital. PATIENTS: Eighteen trauma patients with long-bone fractures, including 5 with definite fat embolism syndrome, 5 in whom the diagnosis had been clinically suspected but was impossible to confirm or exclude before bronchoscopy, and 8 with no clinical evidence of the syndrome. Control groups included 9 patients without previous trauma who developed the adult respiratory distress syndrome for various reasons and 15 normal volunteers. MEASUREMENTS AND MAIN RESULTS: Each patient had fiber-optic bronchoscopy with bronchoalveolar lavage, and the percentage of lavage cells containing intracellular fat droplets stained with oil red 0 were determined. In the five patients with definite fat embolism syndrome, light microscopic study of bronchoalveolar cells stained with oil red 0 showed many large intracellular fat droplets (mean percentage of cells containing fat droplets, 63%; range, 31% to 82%), whereas less than 2% of cells recovered by lavage from trauma patients with no clinical evidence of the syndrome, from patients with the adult respiratory distress syndrome, or from normal volunteers contained such inclusions. Use of the same technique in the five patients with possible fat embolism syndrome permitted the immediate identification of three patients in whom this diagnosis was later confirmed by subsequent autopsy or clinical follow-up. CONCLUSIONS: The identification of fat droplets within cells recovered by bronchoalveolar lavage in trauma patients may be a rapid and specific method for establishing the diagnosis of the fat embolism syndrome.
Bronchoalveolar lavage in trauma patients for diagnosis of fat embolism syndrome.

Vedrinne JM, Guillaume C, Gagnieu MC, Gratadour P, Fleuret C, Motin J.

Service d'Anesthesie-Reanimation, Hopital Edouard Herriot, Lyon, France.

Fat embolism syndrome (FES) is a rare but serious complication occurring after long bone fractures. Presence of fat droplets in cells obtained by bronchoalveolar lavage has been proposed as a specific tool for FES diagnosis in trauma patients. We evaluated this technique over a 15-month period in 85 patients. Twenty-eight patients were excluded. The remaining 57 patients were divided into three groups: group 1, 26 patients without trauma as control; group 2, 22 patients with trauma but without evidence of FES; and group 3, nine patients with trauma and evidence of FES. Six of 26 patients in group 1 and nine of 22 patients in group 2 exhibited fat droplets in alveolar macrophages, whereas three of nine patients of group 3 had not. This study suggests that (1) presence of fat droplets in alveolar macrophages is not a reliable method for diagnosis of FES after long bone trauma, and (2) many conditions are associated with fat droplets in alveolar macrophages.
Alveolar macrophages fat stain in early diagnosis of fat embolism syndrome.

Reider E, Sherman Y, Weiss Y, Liebergall M, Pizov R.

Department of Anesthesiology and Critical Care Medicine, Hadassah University Hospital, Jerusalem, Israel.

The aim of this study was to assess the contribution of bronchoalveolar lavage (BAL) in the diagnosis of fat embolism syndrome (FES). The presence of fat droplets in alveolar macrophages was addressed in 13 trauma patients with bone fractures and 10 non-trauma patients with acute respiratory distress syndrome (ARDS). The control group was composed of 5 anesthesized patients with ischemic heart disease, immediately prior to cardiac surgery. Two patients with suggestive clinical and laboratory signs of FES had 40% and 24% fat-containing alveolar cells, respectively. The trauma patients without signs of FES displayed a wide variation in the percentage of fat-containing macrophages (from 3% to 95%). Most of the patients with ARDS who were receiving lipid emulsion as part of their parenteral nutrition, had a high percentage (> 85%) of fat-containing macrophages. Patients with normal lungs had no fat-containing macrophages. Our findings suggest that BAL Oil Red O-positive macrophages are frequently observed in trauma patients irrespective of the presence of FES. Therefore, estimation of the percentage of fat-containing macrophages from BAL is an unreliable marker of FES.
Specificity of bronchoalveolar lavage for the diagnosis of fat embolism syndrome.

Stanley JD, Hanson RR, Hicklin GA, Glazier AJ Jr, Ervanian A, Jadali M.

Department of Surgery Education, Iowa Methodist Medical Center, Des Moines 50309.

The diagnosis of fat embolism syndrome (FES) is relatively difficult because simple, quantitative criteria have been lacking. The results of a recent study, however, suggest that the diagnosis of FES can be made if more than 5 per cent of the cells in fluid obtained by bronchoalveolar lavage are lipid-laden. Our study was designed to assess the specificity of this lipid staining test of bronchoalveolar cells for diagnosing FES in a series of patients coming to the pulmonology clinic. Thirty-four consecutive patients with suspected pulmonary diseases, but not FES, underwent routine bronchoscopy. Bronchoalveolar fluid was applied to slides, fixed with formalin, and stained with oil red 0. Three hundred consecutive cells of each specimen were observed for red-staining droplets. More than 5 per cent of bronchoalveolar lavage cells stained for lipids in 25 of the 34 subjects. The calculated specificity, assuming a negative finding is defined as \( < \) or \( \leq \) 5 per cent lipid-laden cells in the sample, was 26.5 per cent. We conclude that staining of bronchoalveolar lavage cells for lipids is not a specific test for FES.
Role of bronchoalveolar lavage in the diagnosis of fat embolism syndrome

N. Roger*, A. Xaubet*, C. Agustí*, E. Zabalza**, E. Ballester*, A. Torres*, C. Picado*, R. Rodríguez-Roisín*

Fig. 1. – Individual (O) and mean±SD (■) of macrophages oil red O positive in bronchoalveolar lavage of the different groups. FES: fat embolism syndrome. *: p<0.001, with respect to the other groups.
Contribution of bronchoalveolar lavage to the diagnosis of posttraumatic pulmonary fat embolism.


Service d'Anesthesie-Reanimation, Universite de Paris Sud, Hopital de Bicetre, France.

OBJECTIVE: To verify whether the determination of the percentage of cells recovered by bronchoalveolar lavage and containing fat inclusions is a useful diagnostic tool of posttraumatic pulmonary fat embolism. DESIGN: Prospective study. SETTING: Surgical Intensive Care Units in two university hospitals. PATIENTS: 56 successive trauma patients needing prolonged postinjury mechanical ventilation, including 4 with clinical definite fat embolism syndrome, 5 in whom the diagnosis had been clinically suspected but was impossible to confirm or exclude before bronchoscopy, and 47 with no clinical evidence of the syndrome. Control groups included 8 patients without previous trauma who developed ARDS and 6 healthy surgical patients. METHODS: Bronchoalveolar lavage was performed within the first post-traumatic 3 days in trauma patients, at the beginning of the pulmonary disease in non trauma ARDS patients and just after anesthetic induction in healthy ortopedic patients. The magnitude of lipid content in alveolar cells was compared with the clinical pattern of the pulmonary fat embolism syndrome retrospectively evaluated at the seventh day postinjury in trauma patients. RESULTS: All the patients with definite fat embolism syndrome had more than 70% of lavage cells containing fat droplets. The group of patients in whom the diagnosis of the fat embolism syndrome was suspected had percentages of fat cells above 30% in 4 out of 5 patients. A percentage of fat cells above 30% was only observed in 7 out of the 47 patients without clinical evidence of the syndrome. The percentage varied between 0% to 35% in the group of non trauma ARDS patients and between 0 to 5% in healthy surgical patients. CONCLUSION: Lipid inclusions in alveolar cells are common during traumatic and non-traumatic respiratory failure. Determination of the percentage of cells recovered by bronchoalveolar lavage and containing fat droplets may contribute to the diagnosis of the fat embolism syndrome in mechanically-ventilated trauma patients with respiratory failure provided that the significant threshold would be 30%.
Fat embolism syndrome and pulmonary microvascular cytology.

Castella X, Valles J, Cabezuelo MA, Fernandez R, Artigas A.

Intensive Care Medicine Service, Hospital de Sabadell, Spain.

Pulmonary microvascular cytology consists of analysis of capillary blood sampled while a Swan-Ganz catheter is in the wedge position. This technique has proved to be useful in the diagnosis of lymphangitic spread of carcinoma in the lungs and there are case reports of their use in amniotic fluid embolism. Its usefulness in diagnosing fat embolism syndrome has been shown only rarely. We report a new case in which pulmonary microvascular cytologic study allowed a definite diagnosis of fat embolism syndrome. We suggest obtaining routinely samples of capillary blood when a pulmonary catheter is in place and fat embolism is suspected on a clinical basis.
Diagnostic : LBA et dosage lipides

- Dosage des lipides neutres dans LBA : cholestérol et ses esters

- G Karagiorga biochemical parameters of bronchoalveolar lavage fluid in fat embolism
  Int Care Med 2006; 32
Diagnostic : tomographie gamma camera et 99 m Tc phytate

- Moelle osseuse embolisée renferme des cellules réticuloendothéliale phagocytant le m 99 Tc phytate

- W Bruce novel imaging strategy for the detection of fat embolism after arthroplasty
  ANZ J.Surg 2004 74
Diagnostic : Embolie graisseuse et IRM spectroscopie cérébrale

- En IRM les images obtenues sont peu spécifiques, lésions dispersés dans la substance blanche dans les zones péri ventriculaires et subcorticales.

- La spectroscopie augmente la spécificité de l’IRM en mettant en évidence la présence anormale d’acide gras libre.

Pathogénie

- 1°) Théorie mécanique
  - Autopsie
  - ETO
  - FOP
  - DTC
- 2°) Théorie biochimique
ETO

• Présence d’embols graisseux en ETO : jusqu’à 93% des pts ayant une prothèse de hanche suivant la technique de pose

• MJ Koessler Anesth Analg 2001 ; 92
Doppler trans cranien

- détection en per opératoire (prothèse hanche et genoux) d’embols graisseux sur l’ACM chez 50% des pts

Pathogénie

• 1°) Théorie mécanique

• 2°) Théorie biochimique
  – AGL
  – Déposition des émulsions lipidiques
Acide Gras Libre

• AGL responsable
  – Vascularite et OAP hémorragique
  – Lésions pulmonaires

• AGL proviendraient de l’hydrolyse des triglycérides
Les Lipases

- **Action**
  - Triglycérides → glycérol
  - Acide gras libre

- **Augmentation de la lipase sérique de 50 à 70% dans traumatisme avec fracture**

- **Facteurs influençant la lipolyse** :
  - Augmentée par : stéroïdes, cathécholamine
  - Freinée par : glucose, insuline, betamimétique, alcool
La Phospholipase A2 (PLA2)

Activité PLA2 augmente lors de traumatisme
Augmente les médiateurs inflammatoires
Proteine C réactive (CRP)

• CRP dans traumatisme et sepsis
• Responsable de l’agglutination de chylomicrons et de lipoprotéines de très basse densité (VLDL) présents dans le sang sous forme d’émulsions
• Flag A2
  – 1 Flag A2 >0 sur 13 dossiers
Choc et coagulation

- Thrombocytes
- Gouttelettes graisseuses

Piégés dans capillaires pulmonaires

Lésions endothéliales

Activation de la coagulation
Prévention

- 1°) correction hypovolémie et hypoxémie
- 2°) sédation
- 3°) immobilisation
The role of rehydration in the prevention of fat embolism syndrome

I.D. McDermott*, P. Culpan, M. Clancy, J.F. Dooley

Hillingdon Hospital, 30 Park Way, Ruislip, Middlesex HA4 8NU, UK

Accepted 6 December 2001

Abstract

We encountered three cases of young sportsmen developing fat embolism syndrome (FES) after sustaining isolated tibial shaft fractures whilst playing football. All fractures were treated with intra-medullary nails and all three patients were kept nil-by-mouth pre-operatively without intravenous fluids.

Correction of shock is often quoted as an important factor in the prevention of FES. However, animal studies have shown that dehydration, as opposed to hypovolaemia, may also be of great importance. We therefore examined the specific gravity of the urine of 20 patients with musculoskeletal injuries sustained during sport. The mean urinary specific gravity was significantly higher than that of a control group of 10 members of staff.

We emphasise the importance of adequate pre-operative rehydration, especially if injuries were sustained during heavy exercise, as this may reduce the risk of developing FES.

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Traitement médical

• 1°) symptomatique
  – Ventilation protectrice

• 2°) autres
  – Corticoides à petites doses
  – albumine
  – Alcool
  – Héparine
The use of methylprednisolone and hypertonic glucose in the prophylaxis of fat embolism syndrome.

Stoltenberg JJ, Gustilo RB.

In a prospective randomized trial of prophylactic therapy in fat embolism syndrome (FES) 64 patients with femoral and/or tibial shaft fractures uncomplicated by other significant injuries were treated with hypertonic glucose (A), methylprednisolone (B), and placebo (C). Clinical findings and several laboratory parameters were used to establish the diagnosis. Two of the 23 patients in the placebo group (C), 3 of the 21 patients in the hypertonic dextrose (A) group and none of the 20 patients in the methylprednisolone (B) group developed clinical fat embolism syndrome. All patients with fat embolism syndrome were hypoxemic (pO2 less than 65 mm Hg) and exhibited central nervous system symptoms. Four out of 5 were hypocalcemic and 3 patients had thrombocytopenia. There was a high incidence of subclinical hypoxemia or subclinical fat embolism syndrome (29% in A Group, 15% in B Group and 39% in C Group). A comparison between groups of the mean pO2 for each patient demonstrated a statistically significant difference between the methylprednisolone group and the control group (p less than 0.025) as was the comparison of proportions of pO2 less than 70 mm Hg (methylprednisolone versus control p less than 0.01, glucose versus control p less than 0.03). Methylprednisolone given prophylactically may reduce the incidence of fat embolism syndrome and can reduce the degree of hypoxemia associated with long bone fractures of the lower extremity.
Blood alcohol and fat embolism syndrome.

Myers R, Taljaard JJ.

In an analysis of the cases of 100 consecutive patients with diaphyseal fractures in the major bones of the lower limb, the incidence of fat embolism syndrome was 17 per cent. The blood alcohol level was determined at the time of admission. A raised level of alcohol in the blood was associated with a lower incidence of fat embolism.
Traitement chirurgicale

• 1°) Réduction précoce

• 2°) Technique opératoire
  – Enclouage ou pas
  – Alésage ou pas
    • Augmentation des taux IL 6

• 3°) Interruption chirurgie
Conclusions

• Le diagnostic d’embolie graisseuse est un diagnostic d’élimination difficile, et sous estimé
• Les déterminants de la pathogénie reste encore discutés
  – Embolie
  – biochimique
• Les causes de la diminution de fréquence du syndrome d’embolie graisseuse sont multiples
EG : réflexion

• Au bloc
  – DTC et ETO pdt PTH
  – Si + : LBA et quantification ?
    • Inclusion macrophagique au rouge soudan
    • Etude chimique

• En réa
  – Contexte + 2 signes cardinaux
    • FO, EEG
    • LBA ; rouge congo, chimie
    • Lipase, cholestérol, phospholipides, triglycéride
    • FlagA2, hémolyse (haptoglobin, LDH)
    • IRM cérébrale si signe neuro, scanner thoracique si hypoxémie
    • Alcoolémie initiale
Les embolies graisseuses se présentent souvent après des traumatismes, mais elles peuvent aussi apparaître dans d’autres circonstances. Le diagnostic de syndrome d’embolie graisseuse est rarement posé.

Le syndrome d’embolie graisseuse se définit par la triade suivante: pétéchies, troubles respiratoires et troubles neurologiques. Il survient généralement 12 à 36 heures après un traumatisme. C’est le tableau clinique qui permet de suspecter un syndrome d’embolie graisseuse, mais le fond d’œil est un examen complémentaire important.

La pathogénèse n’est pas complètement établie mais les phénomènes suivants y participent: embolisation de graisse et de graisse médullaire, instabilité des émulsions lipidiques, lésions endothéliales et activation des facteurs de la coagulation.

La prévention s’articule autour d’une prise en charge rapide des fractures et de techniques opératoires soignées. Le traitement est symptomatique. Les corticoïdes auraient un effet bénéfique.
<table>
<thead>
<tr>
<th>Lésions tissulaires</th>
<th>Moelle osseuse</th>
<th>Maladies</th>
<th>Facteurs exogènes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lésions des tissus mous</td>
<td>Fracture</td>
<td>Pancréatite aiguë</td>
<td>Alimentation parentérale</td>
</tr>
<tr>
<td>Réduction mécanique des graisses (liposuccion)</td>
<td>Transplantation de moelle osseuse</td>
<td>Obésité</td>
<td>Perfusion de propofol</td>
</tr>
<tr>
<td>Brûlures</td>
<td>Greffe de moelle osseuse</td>
<td>Drépanocytose</td>
<td>Chimiothérapie à hautes doses</td>
</tr>
<tr>
<td>Nécrose hépatique</td>
<td>Mal aigu des montagnes</td>
<td></td>
<td>Facteurs de croissance granulocytaire (G-CSF)</td>
</tr>
<tr>
<td>Rhabdomyolyse</td>
<td>Lupus érythémateux disséminé</td>
<td>Corticoïdes en fortes doses</td>
<td></td>
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<tr>
<td></td>
<td>Diabète sucré</td>
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<td>Lymphographie</td>
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<td>Hépatites virales</td>
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<td></td>
<td>Infarctus du myocarde</td>
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<td></td>
<td>Dystrophie musculaire</td>
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<td>Carcinomes mucineux</td>
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<td></td>
<td>Stéatose hépatique</td>
<td></td>
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</table>
Tableau 3. Fréquence des symptômes et signes.

<table>
<thead>
<tr>
<th>Signes cliniques</th>
<th>Unité</th>
<th>Fréquence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoxie</td>
<td></td>
<td>96%</td>
</tr>
<tr>
<td>Tachycardie</td>
<td>&gt;120/min</td>
<td>93%</td>
</tr>
<tr>
<td>Fièvre</td>
<td>&gt;39 °C</td>
<td>70%</td>
</tr>
<tr>
<td>Anémie inexpliquée</td>
<td>Hémoglobine abaissée &gt;20%</td>
<td>67%</td>
</tr>
<tr>
<td>Altération de la conscience</td>
<td></td>
<td>59% (jusqu’à 80%)</td>
</tr>
<tr>
<td>Thrombopénie</td>
<td>&lt;150 × 10⁹/l</td>
<td>37%</td>
</tr>
<tr>
<td>Pétéchies</td>
<td></td>
<td>33% (jusqu’à 60%)</td>
</tr>
</tbody>
</table>

Les valeurs de ce tableau proviennent d’études cliniques. Leur corrélation avec les données fournies par des autopsies n’a pas été analysée.
The role of secretory phospholipase A2 in acute chest syndrome.

Kuypers FA, Styles LA.

Children's Hospital Oakland Research Institute, Oakland, CA, USA. fkuypers@chori.org

Acute chest syndrome (ACS) is the leading cause of death in sickle cell disease. Severe ACS often develops in the course of a vasoocclusive crisis (VOC), and frequently involves pulmonary fat embolism. Secretory phospholipase A2 (sPLA2), a potent inflammatory mediator, is elevated in ACS, and sPLA2 levels in serum or plasma predict impending ACS. In addition sPLA2 may play a major role in the actual damage to the lung resulting in a new pulmonary infiltrate on chest radiography, respiratory symptoms, and ultimately alveolar collapse and the impairment of gas exchange. The data indicate that measurement of sPLA2 can be useful in alerting the clinician to patients with impending ACS, and suggest that instituting early therapies based on sPLA2 levels, including inhibition of sPLA2 activity, may be useful to prevent or reduce the clinical morbidity of ACS in sickle cell disease.
Les Lipases

• Action
  • Triglycérides
    lipase
    glycérol
    Acide gras libre

• Augmentation de la lipase sérique de 50 à 70% dans traumatisme avec fracture

• Nature en fonction de la localisation
  – Pancréas
  – Endothélium tissu graisseux et musculaire : lipoprotéine lipase
  – Endothélium hépatique : triglycéride-lipase hépatique

• Facteurs influençant la lipolyse
Circonstance d’apparition

• 1°) avec traumatisme
  – Autopsie pulmonaire 90%
    • Fracture os longs
    • Fractures de cotes multiples
    • Enclouage centro médullaires
    • autres

• 2°) sans traumatisme
  – Autopsie 6%

• 3°) Syndrome d’embolie graisseuse
  – ETO per enclouage
    • 93% EG
    • 12% SEG
  – Fréquence SEG :
    • 0.3% à 17% ; mortalité de 1 à 20%