Risk Stratification for the Development of Fat Embolism Syndrome in Patients with Long Bone or Pelvic Fracture

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BACKGROUND

Fat Embolism Syndrome (FES) is a poorly defined clinical entity characterized by fat emboli that classically results in neurologic, dermatologic, and pulmonary manifestations. Diagnosis is usually that of exclusion due to a lack of definitive testing for the syndrome. Clinical criteria have been used to assist in diagnosis, but the lack of pathophysiologic mechanism causes difficulty with risk stratification. Two prevailing theories for the development of FES are the mechanical theory and the biochemical theory. The mechanical theory states that vessel occlusion by fat emboli released from the bone marrow due to increased intramedullary pressure are the main factor contributing to development. The biochemical theory states the predominant role of inflammation in syndrome development. Patients at risk for FES may be predisposed due to worsening of factors related to these theories. Treatment of FES is with supportive care to minimize downstream complications. Early diagnosis with early intervention is critical to help prevent life-threatening sequelae in patients at risk for them.

METHODS

Patients were selected based on the ICD10 code for diagnosis of FES. The remaining patients with FES were diagnosed based on meeting Gurd and Wilson's criteria. Patients were matched in a 3:1 method based on bone fracture, age, and sex. Retrospective chart review was performed for patient analysis of comorbidities and clinical characteristics

RESULTS

18 patients with FES who met inclusion criteria were identified. Mean age was 41.16 ± 20.97. Mean BMI was 27.80 ± 5.99. Respiratory symptoms, neurological dysfunction, and thrombocytopenia were associated with FES patients as previously reported. Other statistically significant differences seen between FES and Controls included hypomagnesemia, hyperphosphatemia, and hypoalbuminemia.

Figure 1: Specific Variable Prevalence in FES and non-FES Patients

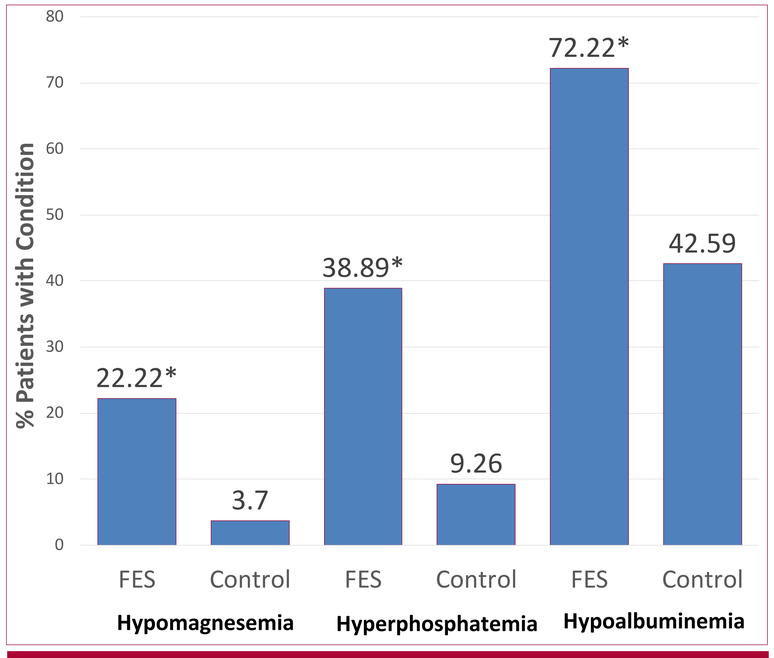

Table 1: Comorbidity Chi Square Analysis

Comorbidity	P-value
Hypomagnesemia	0.014
Hyperphosphatemia	0.008
Hypoalbuminemia	0.041

DISCUSSION AND CONCLUSION

The clinical and laboratory findings in our data were often consistent with previously reported FES diagnostic criteria. Additional findings of this study suggest patients with hypomagnesemia, hyperphosphatemia, and hypoalbuminemia are at increased risk for the development of FES. Decreased magnesium has been shown to cause an inflammatory response, inducing immune and oxidative stress on vasculature. Elevated phosphate has been associated with vascular calcification and involved in disrupting smooth muscle mineralization. Finally, hypoalbuminemia is an indication of an inflammatory state, and has a major role in the disruption of physiologic oncotic pressure. Therefore, further investigation of these variables is necessary for the evaluation and prevention of FES. Furthermore, the role of hypomagnesemia and hyperphosphatemia suggests an inflammatory component to the underlying pathophysiology while the fluid dysregulation of hypoalbuminemia suggests a mechanical component to the pathophysiology. These findings support the hypothesis that the pathophysiology of FES is likely a combination of both theories.

REFERENCES

1. George J, George R, Dixit R, Gupta RC, Gupta N. Fat embolism syndrome [published correction appears in Lung India. 2017 Jan-Feb;34(1):114]. *Lung India*. 2013;30(1):47-53. doi:10.4103/0970-2113.106133
2. Mazur A, Maier JAM, Rock E, Gueux E, Nowacki W, Rayssiguier Y. Magnesium and the inflammatory response: Potential physiopathological implications. *Archives of Biochemistry and Biophysics*. 2007;458(1):48-56. doi: https://doi.org/10.1016/j.abb.2006.03.031.
3. Jono S, McKee MD, Murry CE, et al. Phosphate regulation of vascular smooth muscle cell calcification. *Circulation Research*. 2000;87(7):e10. http://circres.ahajournals.org/cgi/content/abstract/87/7/e10.
4. Soeters, P.B., Wolfe, R.R. and Shenkin, A. (2019), Hypoalbuminemia: Pathogenesis and Clinical Significance. Journal of Parenteral and Enteral Nutrition, 43: 181-193. doi:10.1002/jpen.1451
5. Kwiatt, Michael E, and Mark J Seamon. "Fat embolism syndrome." *International journal of critical illness and injury science* vol. 3,1 (2013): 64-8. doi:10.4103/2229-5151.109426