Peritoneal Calcification and axis

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Dear Sir,

Recently Marichal et al. [1] reported 2 patients with 'progressive calcifying peritonitis'. Both were treated with continuous ambulatory peritoneal dialysis. In 1 patient there was evidence of secondary hyperparathyroid-ism (HPT). Both were hyperphosphatemic. The pathog-enesis of the calcification remained elusive.

Selye [3] proposed a theory called calciphylaxis for the etiologic explanation of such conditions. Essential for calciphylaxis is a state of sensitization caused by HPT, application of vitamin D₂, or tachysterine to provoke a calcification mediated by different challengers in distinct organs. These mediators are often metals (cations). Peritoneal calcification may be produced by ferri-albumi-nate, alum [AlK(SO₄)₂, AlNH₄(SO₄)₂], and thorotrast [3].

After sensitization of female Wistar rats (weighing 150 g) by vitamin D₂ (2 mg i.m./100 g body mass) and oral administration of ZLL-ethionine (55 mg), we have injected intraperitoneally 2.5 ml of Fe-III saccharate (corresponding 50 mg Fe⁺⁺). Five days later, the general status of the animals deteriorated. At that time the peritoneal cavity in autopsied rats showed a hard and whitish glittering calcification (fig. 1). Histological examination verified the macroscopic appearance (v. Kóssa).

Similar results were obtained after sensitization by ferric carboxymethyldextran (Ursoferran; Jenapharm Serumwerk, Bernburg, GDR). This method is particularly useful to produce routinely calcification of pancreatic parenchyma [2]. In experiments using intraperitoneal administration of calciphylactic agents, the participation of ferric ions and a disturbance of the protein metabolism were essential. We believe that in cases of peritoneal calcification associated with HPT or vitamin D administration contact with ferric ions perhaps from blood or hemoglobin must be considered in the discussion of its pathogenesis.

Fig. 1. Rat abdominal wall showing calcification.

References

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