Fat Embolism Syndrome: a Condition Unfamiliar to the Dermatologist

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To the Editor:

The fat embolism syndrome was described by Zenker in 1861, although the triad of confusional state, dyspnea, and petechiae had already been mentioned in the German literature by Von Bergman. Despite being described more than 100 years ago, the diagnosis and specific treatment of this syndrome are still a subject of debate. With the 2 cases presented here, we aim to draw attention to a dermatological disorder which is widely discussed in the medical and surgical literature but which is unfamiliar to the dermatologist.

The first patient was an 18-year-old man who had suffered a motorbike accident and presented a major lung contusion and multiple long-bone fractures (Figure 1). The fractures were immobilized with plaster casts and the patient was admitted to hospital for routine surgery. He was asymptomatic for 48 hours, but before the surgical intervention, small papules a few millimeters in diameter appeared; they were of reddish color, did not blanch on pressure, and were distributed over the anterior aspect of the thorax, base of the neck, and conjunctivae, and in the axillas (Figure 2). This was followed by a rapid onset of neurological signs, consisting of temporospatial disorientation and clouding of consciousness, and acute respiratory failure. The chest radiograph showed bilateral opacities in both lung fields that were not present at the time of admission. No relevant findings except for mild thrombocytopenia were reported in the additional studies performed—complete blood count, biochemistry, and cerebral computed tomography (CT). Despite intensive supportive measures, the patient died a few hours later.

The second patient was a 27-year-old man, also victim of a motorcycle accident. He presented fractures of the right femur and left radius and ulna, and underwent surgical osteosynthesis. After being asymptomatic for 48 hours, he developed fever of up to 39°C associated with psychomotor agitation, severe acute respiratory failure, and pinpoint petechial lesions in the axillas and around the base of the neck.

The complete blood count revealed anemia of 9.5 g/dL and thrombocytopenia of 98 x 10⁹/L; biochemistry and coagulation were normal. Lipiduria was observed in the
fractures or during orthopedic procedures. Fat present in the bone marrow is released, causing embolization of the capillary vessels in the lung parenchyma and in the peripheral circulation. Fat embolism can also occur in other nontraumatic disorders such as pancreatitis and sickle cell anemia, though this is rare. The presence of a fat embolism is a relatively common finding and is usually asymptomatic. However, a small number of patients develop serious signs and symptoms as a result of organ dysfunction that typically involves the skin, central nervous system, and lungs; the term fat embolism syndrome is reserved for these cases. The incidence of this syndrome is estimated at 0.5% of long bone fractures, though the majority of cases are not reported, and remain undetected in the context of a complex clinical situation. Usually, after being asymptomatic for a period of 2 or 3 days (lucid interval), the patient develops the typical clinical triad of respiratory failure, cerebral dysfunction, and petechiae. These patients can also develop fever, anemia, thrombocytopenia, renal failure, jaundice, and tachycardia.

The diagnosis is clinical and requires a high degree of suspicion. Gurd, in 1970, proposed a series of diagnostic criteria (Table). This disorder often passes unnoticed due to its transitory nature and the lack of specificity of some signs.

The fat embolism syndrome is a serious condition that can sometimes follow a fulminant course. An early diagnosis of the condition and the initiation of appropriate therapeutic measures can reduce the number of complications and improve the prognosis. Mortality is currently around 5%-10%.

The skin lesions can sometimes appear before other clinical manifestations. The presence of petechiae with a characteristic distribution, in an appropriate clinical context, should therefore make us think of this disorder.

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Conflicts of interest
The authors declare no conflicts of interest.

References

Table. Gurd Diagnostic Criteria

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<td>Hypoxia</td>
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<td>Tachycardia (&gt;120 beats per minute)</td>
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<td>Fever (temperature &gt; 39°C)</td>
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<td>Unexplained anemia</td>
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<td>Thrombocytopenia (platelet count &gt;15 × 10⁹/L)</td>
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