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Figure 1. Demarcation of residual facial plethora.



Figure 2. Shoulder petechiae.



Figure 3. Subconjunctival hemorrhage. Used with permission of Edward T. Dickinson, MD, Department of Emergency Medicine, University of Pennsylvania School of Medicine, Philadelphia, PA.

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A 49-year-old man sustained a crushing injury to the torso as a lawn tractor rolled over him while he was driving it up the ramp of a flatbed trailer. He was briefly apneic while pinned in a jack knife position. He appeared cyanotic over his face and neck with scattered petechiae over his upper torso and face (Figures 1 and 2). His examination also revealed bilateral subconjunctival hemorrhages (Figure 3). He remained neurologically intact despite an anterior compression fracture of the L2 vertebral body.

*For the diagnosis and teaching points, see page 167.
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DIAGNOSIS:

High-pressure paint injection injury and unrelated Dupuytren's disease. Despite the innocuous early appearance of many high-pressure injection injuries, they are associated with a high rate of significant functional impairment and amputation.¹ The key to treatment is recognition of the severe nature of this injury and urgent surgical decompression and debridement to avoid extensive tissue destruction. The likelihood of amputation increases if debridement is delayed more than 6 hours.² Our patient was taken urgently to the operating room for surgical decompression, debridement, and irrigation. A significant amount of latex paint was debrided from the soft tissues of the palm (Figure 2). The patient's Dupuytren's disease was excised incidentally to facilitate the debridement. The patient did well postoperatively and was discharged home, with arrangements for outpatient hand therapy.

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DIAGNOSIS:

Traumatic asphyxia. Traumatic asphyxia, or Perthes syndrome, is characterized by the presence of subconjunctival hemorrhage, cervical/facial petechiae and edema, and cyanosis secondary to severe compressive forces applied to the torso (farm- or work-related crush injuries).¹⁻³

The pathophysiology of traumatic asphyxia involves distention of the superior vena cava and its tributary veins, with resulting capillary paresis, with or without capillary rupture.¹ The predominant involvement of the head, neck, face, and upper thorax has been postulated to be due to the incompetent valves of the innominate and jugular veins.^{1,4}

Other clinical findings observed in patients with traumatic asphyxia include sore throat, hoarseness, hemoptysis, hemotympanum, dizziness, numbness, and headaches.³ In general, the skin discoloration and subconjunctival hemorrhage associated with traumatic asphyxia usually resolve within 1 month.³

It is not unusual for patients with traumatic asphyxia to have associated significant head (67%), thoracic (58% to 79%), or abdominal (50%) injuries.^{2,3,5-7} Cerebral injury may be present after a prolonged period of asphyxia and is usually secondary to cerebral hypoxia.⁸

Death is rarely due directly to the traumatic asphyxia itself, and the treatment and outcomes of patients with traumatic asphyxia is usually based on associated injuries.^{1,3} In cases of isolated traumatic asphyxia, oxygen supplementation and head elevation to 30 degrees constitute the mainstay of treatment.³

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