Fat Embolism Following Posterior Iliac Graft Harvest for Jaw Reconstruction: Managing the Complications of Major Surgery

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SOMMAIRE

Les spécialistes en chirurgie buccale et maxillofaciale proposent à leurs patients un large éventail de possibilités chirurgicales qui peuvent être classées comme opération mineure ou majeure. Les complications font partie de la chirurgie, mais les opérations lourdes peuvent entraîner des complications qui peuvent mettre la vie en danger et doivent être prises en charge par l'équipe chirurgicale. Le prélèvement de tissus de greffe iliaque pour la reconstruction orale et maxillofaciale présente le risque de complications rares mais graves telles la thrombose veineuse profonde et le syndrome d'embolie graisseuse. Nous présentons ici les complications postopératoires relatives à l'embolie graisseuse chez un patient subissant une opération majeure courante de reconstruction orale et maxillofaciale. Une discussion sur les facteurs entraînant l'embolie graisseuse pendant ou après les opérations chirurgicales vise à aider les chirurgiens à éviter cette complication.

Mots clés MeSH : ambulatory surgical procedures; embolism, fat/etiology; ilium/surgery

Harvesting graft tissue from the anterior and posterior hip to reconstruct deficient parts of the oral and maxillofacial complex has become an established technique for treating advanced maxillary and mandibular atrophy.[1] The relative ease of the surgical approach and predictable recovery time make iliac grafts an attractive option. Surgeons seek to minimize the risks associated with hip graft harvesting by using appropriate surgical techniques[2] and postoperative measures.[3] Several potential complications have been documented (**Box 1**).[4]

One possible complication of iliac graft harvesting is deep vein thrombosis (DVT). Clots can dislodge, becoming emboli, and pass from the veins in the extremities or from the long bones and pelvis to occlude smaller but significant vessels in the lungs.[5] This leads to an alteration in the balance of blood flow supplying vital tissues, which may cause tissue injury, infarction and possibly death. The emboli involved in this pathological process can consist of bone debris, thrombi or fat. Venous thromboembolic disease is a major complication associated with invasive procedures, such as total hip arthroplasty.[5] In oral and maxillofacial surgery, its incidence is low, as less surgical manipulation is required and immobilization time is shorter. In the absence of prophylactic measures, surgical manipulation of the hip can result in a significant risk

Box 1: Possible complications of iliac crest harvesting

Postoperative pain at donor site
Lateral femoral cutaneous nerve paresthesia or
  dysesthesia
Hematoma
Seroma
Gait disturbance
Contour deformity of donor site
Infection
Abdominal herniation
Adynamic ileus
Stress fracture
Keloid scar

Source: Adapted from Freilich and Sándor.[4]

of DVT with a further risk of fatal pulmonary embolism (PE).[5] This most commonly occurs following the second day after surgery. Currently, there is no information on risk assessment regarding such major complications for intraoral or extraoral maxillofacial graft harvesting.[3]

Another significant embolic complication of hip surgery is fat embolism syndrome (FES). This is an uncommon but well-described complication of skeletal trauma characterized by both pulmonary and systemic fat embolism.[4,6,7] FES occurs 2–48 hours following hip surgery or pelvic and long-bone fractures.[6]

A better understanding of the factors that stimulate thrombogenesis and fat embolism during or following surgical procedures might provide a more focused approach to prevention of both venous and fat embolism.

Case Report

A 48-year-old man with no significant medical history was referred by his general dentist when he complained of ill-fitting dentures. He had been partially edentulous since losing teeth to decay in his twenties. The patient was taking no medications, reported no drug allergies and had never been hospitalized for major surgery. Physical examination was normal. Intraoral examination revealed partially edentulous maxilla and mandible with inadequate bony width for the placement of dental implants. Panoramic imaging showed inadequate bony height for implant placement in the posterior maxilla due to pneumatization of the sinuses bilaterally (**Fig. 1**).

The patient consented to bilateral sinus floor augmentation with onlay grafting of the posterior maxilla and posterior mandible. The planned treatment was carried out in the operating room under general anesthesia. A 3 cm × 3 cm cortical block of posterior iliac crest and approximately 55 mL of cancellous bone were harvested from the left hip. The intraoral procedures were completed as planned. There

were no intraoperative complications, and after the procedure the patient was extubated and taken to the recovery unit.

While in recovery, the patient was found to have a blood pressure of 205/110 mmHg measured through an arterial line. This hypertension was treated with intravenous hydralazine and it resolved within 2 hours. The patient was transferred to the ward in a normotensive state. He had been given prophylactic intravenous antibiotics and was scheduled to begin subcutaneous heparin the following morning. On the first postoperative morning, the patient complained of shortness of breath and exhibited confusion, tachycardia and declining oxygen saturation levels. This rapidly progressed to respiratory failure and the patient required intubation.

The patient was moved to the intensive care unit (ICU) and a full diagnostic work-up was carried out. Computed tomography (CT) showed bilateral pulmonary emboli (**Fig. 2**). Doppler studies of the lower extremities were negative for DVT. A cardiac echo revealed borderline concentric left ventricular hypertrophy. The patient's thromboembolic work-up was negative for the presence of antithrombin III, antiphospholipid A and proteins C and S. A diagnosis of bilateral fat embolism was made and the patient was treated with intravenous heparin.

Two days later, the patient was extubated and transferred from the ICU to the ward. The patient's heparin dose was decreased and he was started on coumadin. When the coumadin became therapeutic, the patient was discharged from the hospital with appointments for follow-up with his family doctor and the thromboembolism clinic. At his 6-month visit, no evidence of morbidity was associated with the donor or grafted sites and there were no long-term effects from his postoperative complication.

Discussion

The occurrence of FES associated with elective oral and maxillofacial surgical procedures has not been previously reported, although reviews, analyses and case reports of fat emboli resulting from pelvic and long-bone fractures or hip arthroplasties abound in the orthopedic literature.[8–10] More than 90% of patients with long-bone fractures or who undergo placement of a hip prosthesis have detectable fat emboli.[6,11–13] Instrumentation of the fatty marrow space found at the corticocancellous harvest site in the posterior hip could also result in the intravasation of fat globules into the marrow vasculature.[9]

Fat embolism occurs when fat globules enter the lung parenchyma or peripheral circulation.[12] Fat emboli are thought to originate from either mechanical or biochemical sources.[10] The mechanical theory infers that they are released into damaged marrow vasculature as a result of bone and marrow manipulation and increased intramedullary pressure during instrumentation. The intravasation of fat globules into the lumena of blood vessels requires the loss of
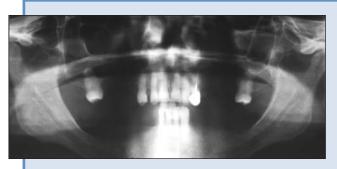
Figure 1: Preoperative panoramic radiograph showing atrophic partially edentulous maxilla and mandible with well-pneumatized maxillary sinuses requiring bone-graft reconstruction.

Figure 2: Computed tomography scan of lungs consistent with bilateral fat emboli.

integrity of marrow vessels, the availability of particulate fat and marrow and the pressurization of the medullary space.[14] The presence of marrow fat has been demonstrated in the femoral vein within seconds of intramedullary manipulation, as has the presence of echogenic material passing through the right ventricle during instrumentation of the medullary cavity of the femur.[15] These emboli can travel and lodge in vessel-rich areas, producing local ischemia, inflammation and platelet aggregation. The biochemical theory is based on the premise that hormonal changes due to trauma or sepsis induce the release of free fatty acids from plasma as chylomicrons, which are stimulated to coalesce.[16,17] FES is believed to be caused by the toxic effects of free fatty acids liberated at the endothelial layer which cause capillary disruption, perivascular hemorrhage and edema.[18,19]

Although fat embolism has been shown to be very common, only a small proportion of affected individuals will develop clinically evident symptoms. Studies have shown that 0.5% to 2% of patients with a single long-bone fracture and 5% to 10% of patients with multiple long-bone fractures, with or without pelvis fractures, will develop FES.[13,20,21]

FES consists of a triad of clinical signs in addition to the fat embolism: respiratory distress or hypoxia, altered mental status and possibly a petechial rash.[13,20,21] The clinical course varies considerably. A severe presentation with rapid onset (within 12 hours) is associated with a poor prognosis. The onset may also be progressive (over 24–72 hours), with a broad range of presentations including subclinical.[13]

The diagnosis of FES is one of exclusion and is based primarily on clinical signs.[13,19,22] Definitive diagnosis is difficult as many of the symptoms are not specific and may mimic other, more common conditions. Major diagnostic criteria include petechial rash, respiratory symptoms and neurologic signs consisting of confusion, drowsiness and coma. Minor criteria include tachycardia, pyrexia with an elevated temperature (> 39.4°C), retinal petechiae, jaundice and renal signs, such as anuria or oliguria.[23]

No specific investigation allows definitive diagnosis of FES. Standard blood work may reveal anemia, thrombocytopenia and poor arterial oxygen saturation. Screening blood and urine for the presence of fat is also nonspecific. Bronchoalveolar lavage has been proposed as a nonspecific and invasive diagnostic tool, as the lavage fluid may show macrophages with fat globules within their cytoplasm.

Various imaging modalities have been proposed to aid in diagnosis. Infiltrates commonly noted on chest radiographs cannot rule out other potential etiologies for pulmonary dysfunction. Chest ventilation–perfusion scans are helpful in ruling out PE but fat emboli are often too small to be detected.[11] CT scans have been used with limited success. Cerebral CT scans are usually negative or may show generalized edema or high-density spots, but are generally of little diagnostic value.[16,19,22] High-resolution CT scans (HRCT) of the lungs have demonstrated ground-glass opacities and thickening of the interlobular septa in patients with FES.[20] Although HRCT may aid in early detection of mild FES, diagnosis remains primarily clinically based.[20]

FES has not been previously reported as a complication of elective oral and maxillofacial reconstructive procedures. Manipulation of the posterior hip marrow cavity may have resulted in bilateral pulmonary fat emboli in this patient. The rapid timeline of the postoperative events, together with the negative coagulation studies and negative Doppler studies of the legs suggest that the patient's pulmonary distress and confusion were the result of fat emboli. These emboli may have intravasated into the circulation as a result of instrumentation of the posterior hip marrow cavity. Prompt recognition by the surgical team together with resuscitative and supportive measures resulted in successful treatment of this life-threatening complication. ✦

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References

1. Bloomquist DS, Feldman, GR. The posterior ilium as a donor site for maxillo-facial bone grafting. *J Oral Maxillofac Surg* 1980; 8(1):60–4.

2. Schult M, Kuchle R, Hofmann A, Schmidt-Brakling T, Ortmann C, Wassermann E, and others. Pathophysiological advantages of rinsing-suction-reaming (RSR) in a pig model for intramedullary nailing. *J Orthop Res* 2006; 24(6):1186–92.

3. Marx RE, Morales MJ. Morbidity from bone harvest in major jaw reconstruction: a randomized trial comparing the lateral anterior and posterior approaches to the ilium. *J Oral Maxillofac Surg* 1988; 46(3):196–203.

4. Freilich MM, Sándor GK. In-office iliac crest bone harvesting for peri-implant jaw reconstruction. *J Can Dent Assoc* 2006; 72(6):543–7.

5. Levy DL. The fat embolism syndrome. A review. *Clin Orthop Relat Res* 1990; (261):281–6.

6. Pitto RP, Hamer H, Fabiani R, Radespiel-Troeger M, Koessler M. Prophylaxis against fat and bone-marrow embolism during total hip arthroplasy reduces the incidence of postoperative deep-vein thrombosis: a controlled, randomized clinical trial. *J Bone Joint Surg Am* 2002; 84-A(1):39–48.

7. Fenger C, Salisbury JH. Diffuse multiple capillary fat embolism in the lungs and brain is a fatal complication in common fractures. *J Chicago Med Exam* 1879; 39:587–95.

8. Pell AC, Hughes D, Keating J, Christie J, Busuttil A, Sutherland GR. Fulminating fat embolism syndrome caused by paradoxical embolism through a patent foramen ovale. *N Engl J Med* 1993; 329(13):926–9.

9. Tronzo RG, Kallos T, Wyche MQ. Elevation of intramedullary pressure when methylmethacrylate is inserted in total hip arthroplasty. *J Bone Joint Surg Am* 1974; 56(4):714–8.

10. Orsini EC, Richards RR, Mullen JM. Fatal fat embolism during cemented total knee arthroplasty: a case report. *Can J Surg* 1986; 29(5):385–6.

11. Fabian TC, Hoots AV, Stanford DS, Patterson CR, Mangiante EC. Fat embolism syndrome: prospective evaluation in 92 fracture patients. *Crit Care Med* 1990; 18(1):42–6.

12. Koessler MJ, Fabiani R, Hamer H, Pitto RP. The clinical relevance of embolic events detected by transesophageal echocardiography during cemented total hip arthroplasty: a randomized clinical trial. *Anaesth Analg* 2001; 92(1):49–55.

13. Bulger EM, Smith DG, Maier RV, Jurkovich GJ. Fat embolism syndrome. A 10-year review. *Arch Surg* 1997; 132(4):435–9.

14. Byrick RJ. Fat embolism and postoperative coagulopathy. *Can J Anaesth* 2001; 48(7):618–21.

15. Manning JB, Bach AW, Herman CM, Carrico CJ. Fat release after femur nailing in the dog. *J Trauma* 1983; 23(4):322–6.

16. Harris AC, Torreggiani WC, Lyburn ID, Zwirewich CV, Ho SGF, Munk PL. CT and sonography of traumatic fat embolism in the common femoral vein. *AJR Am J Roentgenology* 2000; 175(6):1741–2.

17. Baker PL, Pazell JA, Peltier LF. Free fatty acids, catecholamines, and arterial hypoxia in patients with fat embolism. *J Trauma* 1971; 11(12):1026–30.

18. Glover P, Worthley LI. Fat embolism. *Crit Care Resusc* 1999; 1(3):276–84.

19. Mellor A, Soni N. Fat embolism. *Anaesthesia* 2001; 56(2):145–54.

20. Malagari K, Economopoulos N, Stoupis C, Daniil Z, Papiris S, Muller NL, and other. High-resolution CT findings in mild pulmonary fat embolism. *Chest* 2003; 123(4):1196–1201.

21. Richards RR. Fat embolism syndrome. *Can J Surg* 1997; 40(5):334–9.

22. Georgopoulos D, Bouros S. Fat embolism syndrome: clinical examination is still the preferable diagnostic method. *Chest* 2003; 123(4):982–3.

23. Muller C, Rahn BS, Pfister U, Meining RP. The incidence, pathogenesis, diagnosis, and treatment of fat embolism. *Orthop Rev* 1994; 23(2):107–17.