Case Report

Turning the young and healthy patient into an elderly faster: A cascade of complications related to immobility

Luiz Alberto Cerqueira Batista Filho[1], Mario Correa Netto Pacheco Junior[2], Alexandre Toledo Maciel[1], Larissa Seraphim Medeiros[3]

From [1]Critical Care Consultant, Brazilian Board in Critical Care Medicine, Member of the Imed Group, [2]Orthopedic Surgeon, Brazilian Board in Orthopedics, Knee Specialist, Hospital Copa D'Or, Rio de Janeiro, [3]Physician, General Practioner, Research Department, Imed Group, São Paulo, Brazil

ABSTRACT

A 42-year-old individual presented with a ruptured left patellar ligament after playing basketball. After undergoing reconstructive orthopedic surgery, he sustained a posterior shoulder dislocation while using crutches, necessitating additional surgery. This combination of orthopedic injuries rendered the patient immobile and bedridden for several months, and immobility-related complications began to develop. Immobilism is a condition that physicians typically associate with elderly and neuropathic patients; however, little is said about this pathology when young and healthy patients who have experienced acute diseases are considered. With a review of the relevant literature, this article describes a peculiar succession of immobility-related complications.

Key words: Bedrest, Cardiovascular deconditioning, Contact dermatitis, Gall stones, Immobilization, Muscular atrophy, Patellar tendon

Immobilism is a condition that affects the lives of elderly and neuropathy patients substantially [1]. This disorder's repercussions on youth are rarely discussed. Polytrauma and significant locomotor system damage generate this syndrome, which is undervalued in young patients. Burns, fractures, muscle and ligament injuries, acute pain, and psychological illnesses might immobilize young patients. Young patients have a better prognosis and clinical condition; therefore, their immobility is often overlooked, with no strategy for a safe hospital exit or for dealing with potential implications while in the hospital. A patient with a severe left patellar ligament injury [2] developed a cascade of complications related to immobility. This case report shows how immobility can cause long-term or fatal implications for the young.

CASE REPORT

An obese 42-year-old former basketball player had mild left patellar tendon soreness for years. Sports caused five anterior left and one anterior right shoulder dislocations between 14 and 35 years. His body mass index was 33.5. A recent bioimpedance evaluated the patient's fat proportion at 21%. Weightlifted 4 times a week at the gym. After 15 years away from basketball, he felt a popping sensation in his left knee on a layup and fell to the ground in severe pain, unable to actively extend the knee and with a left thigh bone deformity.

Upon arrival at the hospital, the patient was alert, oriented, and quite stressed due to pain. There were no pulmonary alterations, except for hyperventilation. Blood pressure was 160×90 mmHg, and there were no abdominal changes. No signs of anemia or dehydration were present. Orthopedic knee radiographs showed patella alta (Fig. 1). Ultrasound showed a complete rupture of the left patellar tendon at the inferior pole osseotendon junction. Distal tendon retraction and patellar superior migration were 5.25 cm. Next day, thick non-absorbable surgical threads were used for direct ligament suturing using the Krackow technique. The patient left the hospital with a plastered left knee the day following surgery.

The day after his release and 2 days after surgery, the patient heard a cracking sound on his right shoulder while using axillary crutches and felt excruciating pain. Anteroposterior radiographs showed the humeral head around the glenoid and below the acromion. A shoulder CT scan revealed posterior humeral head dislocation, requiring surgery the next morning. The patient had an open latarjet coracoid bone block due to glenohumeral anterior instability and numerous shoulder dislocations. Screws anchored the coracoid bone. Slings immobilized right shoulder.

Contact dermatitis develops on immobile limbs (Fig. 2a) and spread to the whole body. The rash was contained with

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Correspondence to: Luiz Alberto Cerqueira Batista, Critical Care Consultant, Hospital Copa D'Or, Rio de Janeiro, Brazil. Filholuiz.cerqueira@imedgroup.com.br

corticosteroids once the plaster splint was removed. A knee brace with lateral stabilizers replaced the plaster splint. Because his left leg could not bear weight and his right arm could not use a cane or crutches, getting the patient out of bed was difficult. The immobile limb swelled 11 days after knee surgery. Left lower limb venous Doppler showed a significant deep venous thrombosis. Percutaneous aspiration thrombectomy and enoxaparin anticoagulation were effective. The multidisciplinary team helped the patient sit on a couch and start physical therapy, reducing bedtime. The patient went home 22 days after shoulder surgery. He had lost 16 kg and had severe muscular atrophy, especially in his immobile limbs. There was also a mild anemia, with hemoglobin of 10.9 g/dL.

Two days home, the patient had significant upper abdominal pain. He was hospitalized again, and an abdominal ultrasound revealed acute cholecystitis. Laparoscopic cholecystectomy revealed a necrotic gallbladder with microcalculi (Fig. 2b). He returned home 3 days after surgery.

Six weeks following surgery, extending the left leg and palpating the patellar tendon were difficult. Knee radiographs showed patella alta again. MRI showed a poorly visible, diffusely tapered, and uneven patellar tendon with post-surgical magnetic susceptibility anomalies between and in the adjacent soft tissues, extensive hyperintense foci permeating its residual fibers, and liquid infiltration (Fig. 3), suggesting new rupture. Second knee surgery was done 50 days later after the initial procedure, using hamstring tendon autografts this time [3]. Two weeks after surgery, knee continuous passive motion (CPM) rehabilitation began (Fig. 4). Seven weeks after the second surgery, the brace was removed. The patient took his first steps with a cane after 9 weeks of physical therapy to strengthen his lower and upper limbs. Six months after the second knee operation, the patient's active knee range of motion is 110° and the left lower limb is reversing the extreme sarcopenia from immobilization. Resistance workouts are helping shoulder rehabilitation.

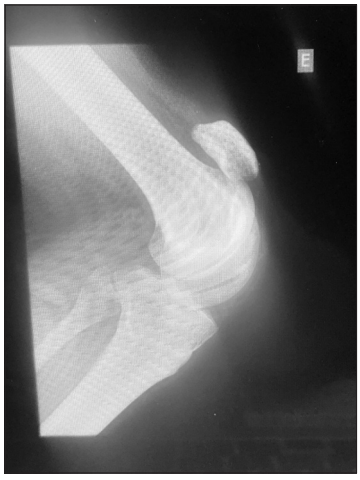

Figure 1: A radiograph of the left knee reveals patella alta

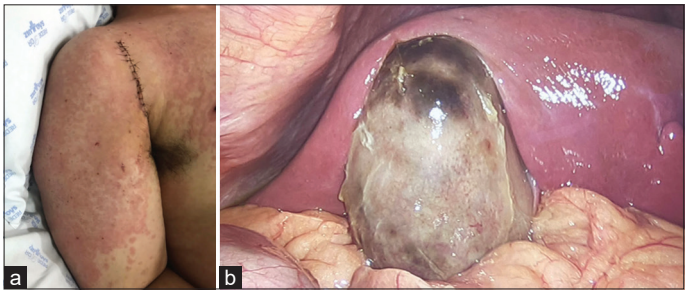

Figure 2: (a) Contact dermatitis on the right arm and chest. Surgical shoulder wound with sutures is shown; (b) Laparoscopic imaging reveals a cholecystitis-related necrotic gallbladder

DISCUSSION

Prolonged immobilization can be secondary to neurological, orthopedic, infectious, or psychiatric causes. This pathology causes cardiovascular, dermatological, respiratory, musculoskeletal, gastrointestinal, neuroendocrine, urinary, and psychological repercussions in stroke, multiple trauma, sepsis, and severe depression patients (Fig. 5). These consequences, which can last for years or hinder rehabilitation, cause many of the problems long-term inpatient patients face.

Many reviews describe the dangers of protracted decubitus, but almost no case reports exist. The odd sequence of events in a young, healthy patient makes this instance remarkable. Mostly,

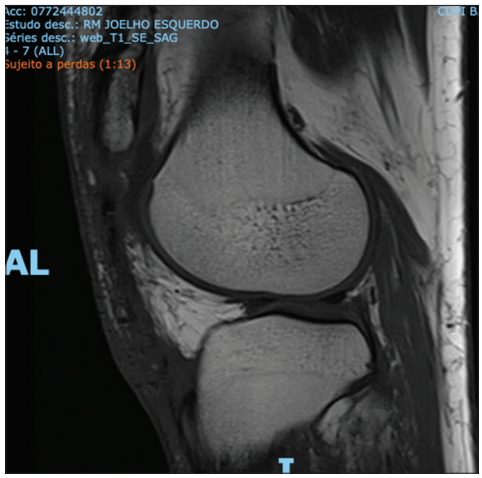

Figure 3: A magnetic resonance imaging of the left knee, showing new patellar tendon rupture

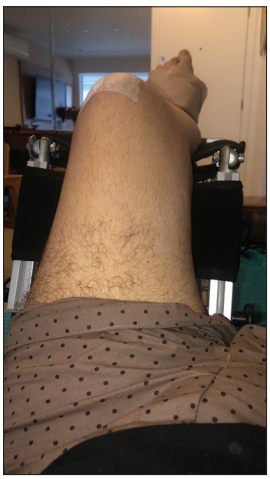

Figure 4: Rehabilitative device utilizing continuous passive motion. Manifest sarcopenia of the left lower extremity

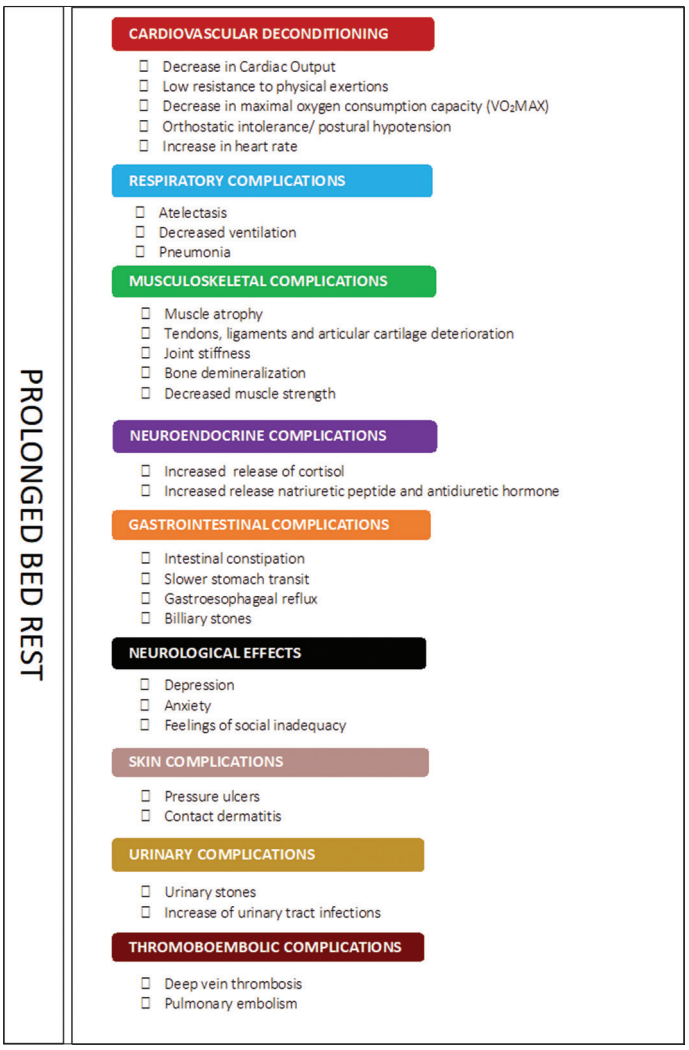

Figure 5: Complications related to prolonged bedrest

the literature describes the unfavorable consequences of lengthy hospitalizations in bedridden patients. These reviews discuss older individuals with various comorbidities, neuropathies, and intensive care unit acquired weakness or septic encephalopathy, which does not suit this case completely.

Extended bed rest is controversial since ancient times. Hippocrates wrote: "If the whole body is rested much more than usual, there is no immediate increase in strength. In fact, a lengthier time of inactivity followed by a quick resumption to exercise will cause deterioration" [4]. Due to war and aerospace medicine advances, immobile patients started to be mobilized early in the second half of the 20th century. Bedrest was used as a proxy for space weightlessness [5]. Due to prolonged sheet and plaster contact, this patient acquired contact dermatitis [6]. Corticoids and avoiding triggers improved his contact dermatitis.

Immobility causes substantial sarcopenia. It increases pro-inflammatory cytokines and reactive oxygen species, lowering muscle mass and increasing proteolysis [7]. Resistance training and dietary protein supplementation are the recommended treatment for bedrest-induced sarcopenia [8]. A CPM device was utilized for the rehabilitation of knee motion. However, it still needs further study on its efficacy, and it does not revert to muscle atrophy [9].

Prolonged immobility causes patients to lose torso control and tire easily. The patient loses aerobic conditioning, which lowers his VO_2 max (maximal oxygen consumption capacity). Absolute bed rest decreases VO_2 max by 0.3% each day. Plasma volume, red blood cell mass, vasodilator function, muscle atrophy, and peripheral O_2 diffusing capacity decrease [10]. Cardiovascular deconditioning resembles a space flight. Bedrest reduces stroke volume by 30% and raises resting heart rate, causing orthostatic intolerance within 72 h [11].

Since body weight limits pulmonary expansibility, bedridden individuals have lower tidal volumes. Lung expansion procedures help prevent atelectasis and pneumonia and enhance aeration. These maneuvers require patient consent and cannot be performed on unconscious individuals. In non-cooperative patients, expiratory positive airway pressure and breath-stacking might be used. However, these maneuvers have not been adequately tested in immobile individuals [12].

Severe orthopedic injuries cause intense muscle atrophy and joint stiffness when large joints are involved, which severely limits movement and working capacity, causing psychosocial stress for young patients. As in this scenario, hospitals often discharge these patients without a plan, which can be harmful since dependency can lead to unexpected actions and injuries, especially in young patients. Due to axillary crutch-related glenohumeral dislocation, the patient was unable to use crutches or a cane with his right upper limb, delaying rehabilitation. Chronic glenohumeral dislocation required a careful discharge plan to avoid further injuries, which was not done.

Prolonged immobility also causes cholecystitis. A 6-month abdominal ultrasound indicated no gallbladder changes in this patient. During his second hospitalization, he lost 16 kg in 22 days, possibly causing gallstones and cholecystitis [13]. Prolonged decubitus slows stomach transit time, lowering appetite and peristalsis, and causing gastroesophageal reflux. Bedridden individuals may also have constipation due to decreased stool motility and anorexia-related fiber and fluid deficiency. Constipation is caused by difficulty sitting and weak evacuation muscles [14].

Immobility worsens the Virchow trinity of venous stasis, hypercoagulability, and blood vessel injury, raising DVT risk [15]. Thus, extended bedrest can cause pulmonary embolism (PE). The patient had severe DVT in the knee brace-immobilized limb and was at high risk for PE. Thrombi aspiration followed a vena cava filter with post-procedure filter removal.

Prolonged bedrest is also a risk factor for renal calculi and urinary tract infections. Bone demineralization increases urinary calcium, which in combination with the decreased kidney-to-bladder urine drainage promotes precipitation and crystalloid aggregation which increase renal calculus risk [16]. Bacteria thrive in urine stasis, increasing the risk of urinary tract infections with prolonged decubitus. Sanitation is difficult for immobile patients, increasing infection risk.

Bedrest increases cortisol [17] which breaks down skeletal muscle and causes gluconeogenesis, glucose insulin resistance, and glucose intolerance. After 24 h of bed rest, 1 L of blood is moved from the legs to the belly, thorax, and head, increasing venous return to the heart and natriuretic peptide levels. However, diuresis decreases plasma volume and red cell mass after a few weeks of immobility. Plasma volume decreases, stimulating the renin-angiotensin-aldosterone axis to maintain blood pressure.

CONCLUSION

Immobility syndrome is a serious and potentially life-threatening condition usually undervalued in young patients. With the assistance of a multidisciplinary team, effective recovery planning should be implemented before hospital discharge. Physical therapy should be initiated early to prevent joint stiffness and promote muscle recovery as well as to prevent venous thrombosis. Psychological support is essential in light of the patient's loss of functionality and its consequent depressive potential. The cascade of complications related to prolonged decubitus should never be underestimated, even in young patients, as it could bring an additional burden to the initial disease.

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