FIZIOTERAPIJSKI PRISTUP BOLESNIKU S EKSTENZIVNOM DISEKCIJOM AORTE I PARAPLEGIJOM - PRIKAZ SLUČAJA

Physiotherapy approach to a patient with extensive aortic dissection and paraplegia - a case report

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PRIKAZ SLUČAJA / CASE REPORT
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Abstract

Introduction: Aortic dissection (AD) is a rare, life-threatening condition that usually presents with intense chest pain. The only life-saving treatment is surgical intervention, which should be performed as soon as possible to avoid a fatal outcome. Postoperatively, AD can be accompanied by numerous complications, one of the rarest being paraplegia. This paper aims to present a physiotherapeutic approach to a patient with extensive AD complicated by paraplegia.

Case report: A 64-year-old male patient, recently discharged from Cardiac Surgery where he underwent surgical treatment of an AD type A, which was complicated by paraplegia caused by malperfusion syndrome, and a large stage 4 presacral pressure ulcer, was admitted to the Medical Intensive Care Unit (MICU) with signs of septic shock and multiorgan dysfunction caused by nosocomial bacterial pneumonia. At the beginning of hospitalization the patient was mechanically ventilated and analgosedated, presenting with respiratory and peripheral muscle weakness and hypersecretion. Following the implementation of intensive care measures, including physiotherapy initiated from day one, the patient was successfully weaned from mechanical ventilation after 23 days and continued to breathe spontaneously via an endotracheal cannula, which was removed after one month. At that point, he was able to speak, cough, and swallow. Due to significant respiratory muscle weakness, respiratory physiotherapy was administered daily to promote airway clearance and maintain pulmonary function. After two months of intensive physiotherapy, muscle contractions became evident in the upper limbs and upper trunk, and the patient achieved independent sitting. Following a 108-day stay in our MICU, the patient was discharged home with a prescription for physical therapy, home nursing care, a wheelchair, and an anti-decubitus mattress.

Conclusion: To our knowledge, this is the first case report describing a physiotherapy approach in a patient with Stanford type A AD complicated by paraplegia. This case also highlights the importance of a dedicated, multidisciplinary therapeutic approach in the recovery of chronically critically ill patients.

Key words: aortic dissection, Stanford type A, paraplegia, physiotherapy, rehabilitation

Sažetak

Uvod: Disekcija aorte (DA) predstavlja rijetko, životno ugrožavajuće stanje koje se najčešće prezentira intenzivnom boli u prsima. Jedini način spašavanja života

je hitna kirurška intervencija kojoj je potrebno pristupiti u što kraćem roku kako bi se spriječio smrtni ishod. Postoperativno DA može uzrokovati brojne komplikacije, rijetko uključujući i paraplegiju. Cilj ovog rada je prikazati fizioterapijski pristup bolesniku s ekstenzivnom DA kompliciranom s paraplegijom.

Prikaz slučaja: Muškarac u dobi od 64 godine nakon otpusta s Odjela za kardijalnu kirurgiju gdje je operiran zbog disekcije aorte tipa A komplicirane malperfuzijom leđne moždine s posljedičnom paraplegijom i razvojem dekubitusa četvrtog stupnja u presakralnom području, hospitaliziran je na Odjelu za intenzivnu medicinu zbog razvoja septičkog šoka i višestrukog zatajenja organa uzrokovanih nozokomijalnom bakterijskom pneumonijom. Početkom hospitalizacije bio je mehanički ventiliran i analgosediran, uz izraženu respiratornu i perifernu mišićnu slabost, te hiperprodukciju bronhijalnog sekreta. Uz provođenje mjera intenzivnog liječenja, uključujući i fizioterapiju koja je započela prvog dana, nakon 23. dana bolesnik je uspješno odvojen od mehaničke ventilacije te je nastavio spontano disati putem endotrahealne kanile, koja je uklonjena mjesec dana kasnije čime je omogućen govor, kašalj i gutanje. Zbog izražene slabosti respiracijskih mišića svakodnevno se provodila respiratorna fizioterapija s ciljem eliminacije sekreta i očuvanja plućnog kapaciteta. Nakon dva mjeseca intenzivne fizioterapije mišićne kontrakcije su vidljive u gornjim ekstremitetima i gornjem dijelu trupa te je omogućeno samostalno sjedenje. Nakon 108 dana hospitalizacije, pacijent se otpušta kući uz propisanu fizikalnu terapiju, zdravstvenu njegu, invalidska kolica i antidekubitalni madrac.

Zaključak: Prema našim saznanjima ovo je prvi prikaz slučaja koji opisuje fizioterapijski pristup pacijentu s DA tipa A komplicirane paraplegijom. Ovaj prikaz naglašava važnost multidisciplinarnog terapijskog pristupa u rehabilitaciji kronično kritičnih bolesnika.

Ključne riječi: disekcija aorte, Stanford tip A, paraplegija, fizioterapija, rehabilitacija

Introduction

Aortic dissection (AD) is a life-threatening condition caused by a tear in the intimal layer of the aorta or bleeding within the aortic wall, resulting in separation (dissection) of its layers. The incidence is estimated at 5–30 cases per million people per year, occurring more frequently in the male population (1,2). Mortality in AD depends on several factors, primarily on the type of dissection. Although AD occurs more frequently in men, women experience higher mortality rates, with the average age of symptom onset being 72 years in women compared to 64 years in men. According to data from the International Registry of Acute Aortic Dissection (IRAD), women with AD are, on average, hospitalized later than men and more frequently present with severe clinical conditions such as coma and cardiac tamponade, along with a higher incidence of pre-hospital mortality (3).

The clinical classification of AD is based on the affected segments of the aorta. The Stanford classification defines type A dissections as those involving the ascending aorta, with possible extension into the aortic arch, regardless of whether the descending aorta is also affected. Type B dissections are limited to the descending thoracic and/or abdominal aorta (4). In addition to the Stanford classification, the DeBakey classification is also commonly used in the literature to categorize AD into three types: Type I, which involves both the ascending and descending segments of the aorta; Type II, confined to the ascending aorta; and Type III, limited to the descending aorta (5). Type III is further subdivided into Type IIIa, which is limited to the thoracic aorta, and Type IIIb, which extends into the abdominal aorta (6).

Risk factors for the development of AD include pre-existing structural weakness of the aortic wall and high arterial pressure (4). This risk is further exacerbated by advanced age and dyslipidemia. The increased incidence of AD among individuals with high blood pressure highlights the importance of preventive measures, timely treatment of dyslipidemia, as well as systematic monitoring and optimization of blood pressure (1,7).

The most frequent presenting symptom of AD is a sudden onset of intense chest pain with a sharp, stabbing character, often radiating to the interscapular region, but it can also occur in the back or abdominal area (8). In cases where dissection extends to the carotid arteries, neurological symptoms such as syncope, confusion, and focal neurological deficits may occur due to decreased cerebral perfusion resulting from systemic hypoperfusion or direct extension into the extracranial arteries supplying the brain. Given that AD can present with various cardiac and neurological symptoms, it may go unrecognized. Thus, it must be considered in the differential diagnosis when such clinical signs are present (9).

If there is clinical suspicion of AD, an integrated approach combining clinical evaluation, laboratory testing, and advanced imaging modalities is essential to confirm the diagnosis and determine appropriate management. Standard chest radiography is insufficient to confirm AD, and therefore, computed tomography (CT), transesophageal echocardiography (TEE), and magnetic resonance imaging (MRI) are more appropriate (10).

The treatment plan for AD depends on the type of dissection. In Stanford type A, the only life-saving treatment option is surgical intervention, which must be undertaken as quickly as possible (8). Surgical treatment includes endovascular aortic surgery, open-heart surgery, and hybrid methods.

The goals of surgical interventions are to prevent or treat aortic rupture, eliminate the entry into the false lumen and reconstruct the true lumen with a graft, stop retrograde propagation of the dissection toward the aortic root, prevent its further anterograde extension into distal segments, and reduce organ malperfusion syndromes (2,11).

The postoperative period may be marked by numerous complications such as multiple organ failure, stroke, myocardial infarction, and, in rare cases, paraplegia caused by organ malperfusion, which further increases mortality (12). It can be explained by the fact that the lower half of the spinal cord is supplied by direct branches from the aorta, which includes the intercostal, lumbar, iliolumbar, and sacral arteries (11).

This paper aims to present a physiotherapeutic approach to the management of a patient with extensive Stanford type A aortic dissection, treated with thoracic endovascular aortic repair (TEVAR), whose postoperative course was complicated by malperfusion syndrome, including paraplegia.

Case report

A 64-year-old male patient was admitted to our Medical Intensive Care Unit (MICU) on 3 May 2025 due to septic shock with multiorgan failure caused by nosocomial bacterial pneumonia. From 5 April 2025 to 26 April 2025, he was hospitalized in Clinical Hospital Dubrava due to Stanford Type A aortic dissection. There, he underwent surgical repair of the aortic arch and ascending thoracic aorta, aortic valve replacement, and endovascular stent-graft implantation extending from the left subclavian artery to the mid-thoracic segment of the aorta. Upon admission, he also presented with heavy paresis of lower limbs, which postoperatively progressed into paraplegia. A neurologist raised suspicion of hypoperfusion or medullary infarction. However, MRI could not be performed due to the presence of metallic foreign bodies. Follow-up brain MSCT revealed a small ischemic lesion in the left superior cerebellar hemisphere, without evidence of new neurological deficits. Due to paraplegia and urinary bladder dysfunction, the patient had an indwelling urinary catheter, and because of long-term supine position, a presacral grade 4 pressure ulcer developed. On 30 April 2025, surgical follow-up was conducted, and a necrectomy was performed.

At the beginning of hospitalization in our MICU, the patient was mechanically ventilated (SIMV mode, FiO2 35 %, Vt 470 mL, RF 24/min, PEEP 7, pressure support 14), unconscious and continually sedated by Sevofluran (AnaConDa), the method of sedative administration by inhalation, which contributes to faster awakening from sedation, significantly reduces negative impact on the liver and kidneys, simplifies titration process and causes less side effects compared to intravenous sedative administration (13). According to the available medical documentation, the patient has a history of post-traumatic stress disorder (PTSD), nicotine dependence, arterial hypertension, right hip total endoprothesis, right inguinal hernia, and surgically treated left inguinal hernia. Since the patient presented with nosocomial sepsis, bilateral pneumonia, and elevated inflammatory parameters, targeted antibiotic therapy was initiated. Sacral decubitus was continually observed and treated by the surgeon, and negative pressure wound therapy (NPWT) was implemented. Peripheral edema, likely resulting from hypoalbuminemia and inactivity, was also noted.

Physiotherapy was initiated on the first day of the patient's admission to the ICU. Initially, the patient presented with complete motor paralysis of the upper and lower extremities, as well as core muscle weakness. Excessive use of accessory respiratory muscles and hypersecretion of bronchial mucus were observed. Respiratory care focused on airway clearance techniques, including postural drainage in combination with chest vibrations, percussion, and aspiration of bronchial secretion. Following improvement in the patient's respiratory status and a reduction in analgo-sedation, the weaning process was initiated and successfully completed on 26 May 2025. The patient continued to breathe via an endotracheal cannula and required only low-flow oxygen therapy.

Maximal inspiratory pressure (MIP), measured using a PowerBreathe device, was 52 cm H_2O, which is about 50 % of the predicted reference value. In addition, diaphragm function was assessed using ultrasound. On the right side (8th–9th rib, intercostal approach), the diaphragm thickness was measured at the end of expiration and found to be 1.7 mm, and at the end of inspiration, 2.4 mm. Diaphragmatic movement (subcostal approach, right side) was 2.17 cm during quiet breathing and 5.23 cm during deep breathing. Peak cough flow was measured using a peak flow meter and was found to be 180 L/min, indicating an increased risk of sputum retention. In response to significant respiratory muscle weakness, the patient underwent a respiratory training program that included inspiratory muscle training (IMT), positive expiratory pressure (PEP) training, and incentive spirometry via endotracheal cannula.

During the entire intervention period, facilitation of normal movement patterns was continuously applied, resulting in active arm movement and gradual return of core stability. Intentional closure of the ET cannula was performed intermittently as a training exercise, enabling the patient to speak, cough, and breathe spontaneously. Successful decannulation and closure of the tracheostomy stoma were achieved on 23 June 2025. The patient continues to receive low-flow oxygen therapy (1 L/min). As the general condition improved, physiotherapy was progressively

intensified, and high-protein parenteral nutrition was initiated. By 3 July 2025, the patient regained the ability to eat independently. As a result of albumin replacement therapy combined with increased physical activity, peripheral edema demonstrated notable regression. During therapeutic exercise, muscle contractions are evident in the upper limbs and upper trunk, while contractions in the lower trunk remain minimal. The patient had decreased light touch sensation from T8, preserved pinprick sensation from T8 to T12–L1, and complete sensory loss below that level. Due to prolonged decubitus treatment, prone positioning was applied intermittently. To manage ongoing fecal maceration of decubitus wound, an enterostomy was performed. A psychologist was also consulted to assess the patient's emotional status and psychological well-being. Our long-term goals included strengthening the respiratory muscles to improve productive coughing, improving postural control to achieve independent sitting, and restoring the ability to perform selected activities of daily living after discharge. Those goals were achieved and after a 108-day stay in our MICU, the patient was discharged home. He was prescribed physical therapy, home nursing care, a wheelchair, and an anti-decubitus mattress.

Discussion

In this case report, we highlighted the importance of a multidisciplinary approach to treating a patient with Type A aortic dissection complicated by paraplegia. Physiotherapy plays a crucial role in preserving functional ability, preventing complications associated with prolonged bed rest, and improving the patient's quality of life.

Despite an exponentially increasing postoperative mortality rate, rising by approximately 1–2 % with each passing hour and reaching nearly 50 % within the first 48 hours (14), our patient survived urgent, extensive endovascular aortic stenting. Unfortunately, spinal cord ischemia still developed, even though preventive measures were applied. A review of the literature identified only a limited number of cases reporting paraplegia as a complication, with no available studies describing the rehabilitation process.

Kawanishi Y et al. reported three cases in their article. The first involved a 51-year-old man with acute type A aortic dissection who underwent total arch replacement. Two hours after extubation, weakness in his lower extremities progressed to paraplegia. Cerebrospinal fluid drainage was initiated immediately and continued over the next three days, combined with corticosteroid, free radical scavenger, and mannitol treatment, resulting in complete recovery from paraplegia.

The second patient was a 54-year-old man who underwent valve repair surgery and had a type A aortic dissection diagnosed intraoperatively. The operation was complicated by cardiac tamponade, which caused a drop in systolic blood pressure to 50 mm Hg. He developed weakness in his lower extremities but fully recovered due to cerebrospinal fluid drainage and the administration of corticosteroids and mannitol.

The third patient was a 57-year-old man with acute type A aortic dissection who underwent total arch replacement complicated by cardiac tamponade, resulting in a decrease in systolic blood pressure to 40 mm Hg. Upon regaining consciousness, he presented with paraplegia. Computed tomography revealed thrombosis of the false lumen in the descending thoracic aorta, and magnetic resonance imaging showed spinal cord infarction spanning from T6 to L2. In this case, cerebrospinal fluid drainage was not performed due to delayed diagnosis, and he was subsequently diagnosed with spinal cord infarction and permanent neurological deficit (15).

A review of the literature revealed no cases involving women, except one report describing a 74-year-old woman with a presentation very similar to ours. Despite all medical interventions, the outcome in that case was ultimately fatal (16).

As mentioned earlier, there are no published studies detailing the rehabilitation process in such cases. Despite this lack of literature, our rehabilitation approach was based on established principles of neurological, respiratory, and cardiac rehabilitation.

Conclusion

To our knowledge, this is the first case report describing a physiotherapy approach in a patient with type A aortic dissection complicated by paraplegia. This case emphasizes the importance of early rehabilitation including mobilization and respiratory physiotherapy in preventing secondary complications associated with aortic dissection. Rehabilitation should be focused not only on physical recovery but also on providing psychological support to address the emotional and cognitive challenges that these patients may face. Furthermore, the report demonstrates that, despite severe neurological impairment, meaningful recovery is possible with a dedicated, multidisciplinary therapy approach. In the absence of existing reports on rehabilitation in these rare cases, documenting and sharing this experience may support the development of standardized, evidence-based physiotherapy guidelines.

Literature

1. Nienaber CA, Clough RE, Sakalihasan N, Suzuki T, Evangelista A, Mehta RH, et al. Aortic dissection. Nature Reviews Disease Primers. 2016;2:16053. doi:10.1038/nrdp.2016.53
2. American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. 2022 ACC/AHA guideline for the diagnosis and management of aortic disease: a report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines [Internet]. Circulation. 2022;145(21):e403-e407 [cited 2025 Jul 2]. Available from: https://doi.org/10.1161/CIR.0000000000001070
3. Huckaby LV, Trimarchi S, Nienaber CA, Evangelista A, Mehta RH, et al. Sex-based aortic dissection outcomes from the International Registry of Acute Aortic Dissection. The Annals of Thoracic Surgery. 2021;113(2):498-505. doi:10.1016/j.athoracsur.2021.03.100
4. Yuan X, Mitsis A, Nienaber CA. Current understanding of aortic dissection. Life. 2022;12(10):1606. doi:10.3390/life12101606
5. Salameh MJ, Ratchford EV. Aortic dissection. Vascular Medicine. 2016;21(3):276-280. doi:10.1177/1358863X16632898
6. Steuer J, Eriksson MO, Nyman R, Björck M, Wanhainen A. Early and long-term outcome after thoracic endovascular aortic repair (TEVAR) for acute complicated type B aortic dissection. European Journal of Vascular and Endovascular Surgery. 2011;41(3):318-323. doi:10.1016/j.ejvs.2010.11.024
7. Wortmann M, Peters AS, Erhart P, Körfer D, Böckler D, Dihlmann S. Inflammasomes in the pathophysiology of aortic disease. Cells. 2021;10(9):2433. doi:10.3390/cells10092433. PMID:34572082
8. Silaschi M, Byrne J, Wendler O. Aortic dissection: medical, interventional and surgical management. Heart. 2017;103(1):78-87. doi:10.1136/heartjnl-2015-308284
9. Kam CT, Soliman M, Okafor N, Rait J. Extensive aortic dissection (Stanford type A) presenting with confusion in a patient: a case report. Radiology Case Reports. 2022;17(9):3031-3034. doi:10.1016/j.radcr.2022.06.020
10. Levy D, Sharma S, Grigorova Y, Farci F, Le JK. Aortic dissection [Internet]. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2024 [cited 2025 Jul 2]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK441963/#article-20603.s8
11. Ishika P, Howard DPJ. Aortic dissection complicated by paraplegia [Internet]. Journal of the Nuffield Department of Surgical Sciences. 2020;1(1) [cited 2025 Jul 2]. Available from: https://doi.org/10.37707/jnds.v1i1.49
12. Luehr M. Treatment of acute type A aortic dissection with the Ascyrus Medical Dissection Stent in a consecutive series of 57 cases. European Journal of Cardio-Thoracic Surgery. 2023;63(3):ezac581. doi:10.1093/ejcts/ezac581
13. Aseri ZA, Alansari MA, Al-Shami SA, Alaskar B, Aljumaiah D, Elhazmi A. The advantages of inhalational sedation using an anesthetic-conserving device versus intravenous sedatives in an intensive care unit setting: a systematic review. The Annals of Thoracic Medicine. 2023;18(4):182-189. doi:10.4103/atm.atm_89_23.
14. Salem M, Rylski B, Bavaria JE, et al. Risk factors for mortality in acute aortic dissection type A: a centre experience over 15 years. The Thoracic and Cardiovascular Surgeon. 2021;69(4):322-328. doi:10.1055/s-0040-1710002
15. Kawanishi Y, Matsumura Y, Koyama T, et al. Three cases of newly developed paraplegia after repairing type A acute aortic dissection. The Annals of Thoracic Surgery. 2007;84(5):1738-1740. doi:10.1016/j.athoracsur.2007.05.076
16. Chiang JK, Tsai KW, Lin CW, Shen TC, Hu SC, Chen CY. Acute paraplegia as the presentation of aortic dissection: a case report. Tzu Chi Medical Journal. 2005;17(5):369–371. doi:10.6440/TZUCMJ.200510.0369