

CASE REPORT

OLGU SUNUMU

BECK SYNDROME IN A YOUNG PATIENT WITH RESPIRATORY ARREST

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ABSTRACT

Anterior spinal artery (ASA) infarcts are also called Beck syndrome. It has a sudden onset and often has radicular or girdle like pain. Flank tetraplegia/paraplegia (within minutes and hours), flask bladder, thermoanesthesia, analgesia, flammable pain are other findings. A small number of ASA infarction cases in which onset with dyspnea or apnea have been reported. With this case report, it is aimed to keep in mind that the ASA infarcts can apply with respiratory arrest which is an atypical clinic. A 21-year-old female patient applied with weakness in the whole body and respiratory distress that developed suddenly. Respiratory arrest developed during her examinations, she was intubated and taken to the intensive care unit. Except for a vague upper respiratory tract infection and polycystic ovary syndrome, there was no medical histories. In her neurological examination, she was tetraplegic, deep tendon reflexes were hypoactive, Babinski sign was bilaterally positive. In addition to the Guillain-Barre and myasthenic crisis, examinations were planned for all differential diagnosis. Meanwhile in the cranial and cervical MR, a T2 hyperintense pathological signal was observed, which started from the bulbous level, followed along the cervical and upper thoracic vertebra. Spinal cord infarcts are extremely rare compared to cerebral infarcts. Infarcts are most frequently observed in the lower thoracic segments and conus medullaris. This young patient who applied with respiratory arrest and having a long segment anterior spinal infarction including bulbous, entire cervical and upper thoracic level, is presented because of its rarity.

Keywords: Anterior spinal artery, Beck syndrome, quadriparesis.

SOLUNUM ARRESTİYLE GETİRİLEN GENÇ BİR HASTADA BECK SENDROMU

ÖZ

Anterior spinal arter (ASA) infarktleri Beck sendromu olarak da isimlendirilmektedir. Ani başlangıçlı olup sıklıkla radiküler veya kuşak tarzı ağrı vardır. Flank tetrapleji/parapleji (dakikalar ve saatler içinde), flask mesane, termoanestezi, analjezi, yanıcı ağrılar gelişebilecek diğer bulgulardır. Dispne veya apne ile başlayan az sayıda ASA enfarktüsü vakası bildirilmiştir. Bu olgu sunumuyla solunum arresti şeklinde atipik bir klinikte getirilen hastada ASA infarktünün da akılda bulundurulması gerektiğini vurgulamak amaçlanmıştır. 21 yaşında bayan hasta tüm vücutta güçsüzlük ve aniden gelişen solunum sıkıntısı ile başvurdu. Tetkikleri sırasında ani solunum arresti gelişmesi üzerine entübe edilerek yoğun bakım ünitesine alındı. Olay öncesinde müphem bir üst solunum yolu enfeksiyonu geçirmesi, polikistik over sendromu dışında öz ve soygeçmişe ait bir özellik yoktu. Nörolojik muayenesinde, tetraplejik, derin tendon refleksleri abolik, taban cildi refleksi bilateral lakayttı. Guillain-Barre ve myastenik krize ek olarak, tüm ayırıcı tanılar için incelemeler planlandı. Bu arada çekilen kranial ve servikal MR'ında bulbos düzeyinden başlayan, servikal ve üst torasik vertebra boyunca izlenen T2 hiperintens patolojik sinyal gözlemlendi. Spinal kord infarktleri serebral infarktlerle karşılaştırıldığında oldukça nadir görülür. İnfarktler en sık alt torasik segmentlerde ve konus medülleriste gözlenir. Solunum arrestiyle getirilen, bulbos, tüm servikal ve üst torakal seviyeyi içine alan oldukça uzun segment anterior spinal infarktüs saptanan bu genç olgu, oldukça nadir görülmesi nedeniyle sunulmuştur.

Anahtar Sözcükler: Anterior spinal arter, Beck sendromu, quadriparezi.

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INTRODUCTION

Anterior spinal artery infarction is the most common type of spinal infarction. It is also known as the "Beck's syndrome." It is frequently observed in the thoracic region (1). Most of the cases experience motor loss of paraparesis below the lesion level, decreased bilateral pain sensation, temporary knife-like stabbing back pain in the region corresponding to the lesion level, and 75% of the cases exhibit autonomic findings (urinary retention, constipation) (2). Loss of motor function is usually in paraparesis, flaccid and areflexic at first and spastic in later stages. Symptoms may vary depending on where the infarct affects. For instance, if it affects only the anterior horn motor neurons, it may mimic ALS; in this case, sensory and sphincter functions are normal (2). In an infarct higher up on the spinal cord, if the lesion reaches above the C3-C5 levels, affected intercostal muscles and diaphragm may cause respiratory distress, even leading to a respiratory arrest (3,4). Although the pathogenesis and clinical findings of spinal cord infarcts are clear, the findings regarding high-level infarcts are less defined (5,6). This case report emphasizes that ASA infarction should also be considered in a young patient with an atypical clinical presentation of respiratory arrest.

CASE REPORT

A 21-year-old female patient, who presented to the emergency service due to respiratory distress following sudden whole body weakness, developed respiratory arrest during the examinations and was intubated and referred to the intensive care unit. Except for the diagnosis of polycystic ovary syndrome and a suspected upper respiratory tract infection one week before the event, there were no significant indications in her medical history and family history. Her relatives reported no drug/substance use. The patient was switched to tracheostomy due to the continued respiratory problem despite the reduced need for sedation. The patient's neurological examination was performed under tracheostomy; eyes were spontaneously open, cooperation was limited, verbal output was hypophonic due to tracheostomy, muscle strength was 1/5 in all four extremities, deep tendon reflexes were abolic, and

plantar reflex was bilaterally absent. The senses of touch, pain - heat, and vibration could not be evaluated due to limited patient cooperation. The patient did not have any significant pathological findings in the laboratory tests. In the lumbar puncture, the CSF glucose was 75 mg/dl (simultaneous blood glucose was 100 mg/dl), and protein was 29 mg/dl. No cells were observed except for 10 leukocytes. Due to rapid progression, the patient could not give a clear response in the 2nd cycle of plasmapheresis treatment for autoimmune diseases (planned as 5 cycles in total), and the simultaneous electromyography revealed acute-subacute neurogenic involvement symptoms, including acute denervation findings and apparent bilateral upper extremity involvement. The repetitive nerve stimulation test results were normal. Cranial and cervical MR examinations also revealed increased T2 hyperintense pathological signal consistent with ASA infarction starting from the bulbous level, followed along the entire cervical vertebra, and continuing throughout the upper thoracic vertebra, and fine calibration in the distal right vertebral artery (Figure 1 a, b, 2, 3).

All vasculitis markers were negative in the etiologic examination for increased signal consistent with anterior spinal artery infarction. The patient had no history of trauma or surgical intervention, and the tests for infectious processes such as syphilis and HIV were also normal. No pathology was observed in genetic diagnosis tests such as the thrombophilia panel. Anti-aquaporin 4 antibody was negative in immunofluorescence examination. No etiologically significant pathology was detected in the cranial and cervical MR angiography examinations, except that the bilateral vertebral arteries were in fine calibration.

Her pre-discharge examination was conscious and had limited cooperation, difficulty speaking, and hypophonia; her four extremity muscle strength was 3/5. She had hypoesthesia in 4 extremities, including neck, trunk, and mandible. Vibration and position sense in 4 extremities were normal as far as could be evaluated. After long-term physical therapy, extremity spasticity developed and only person-assisted mobilization was possible. All necessary consent and signature of the patient were obtained before discharge for this publication.

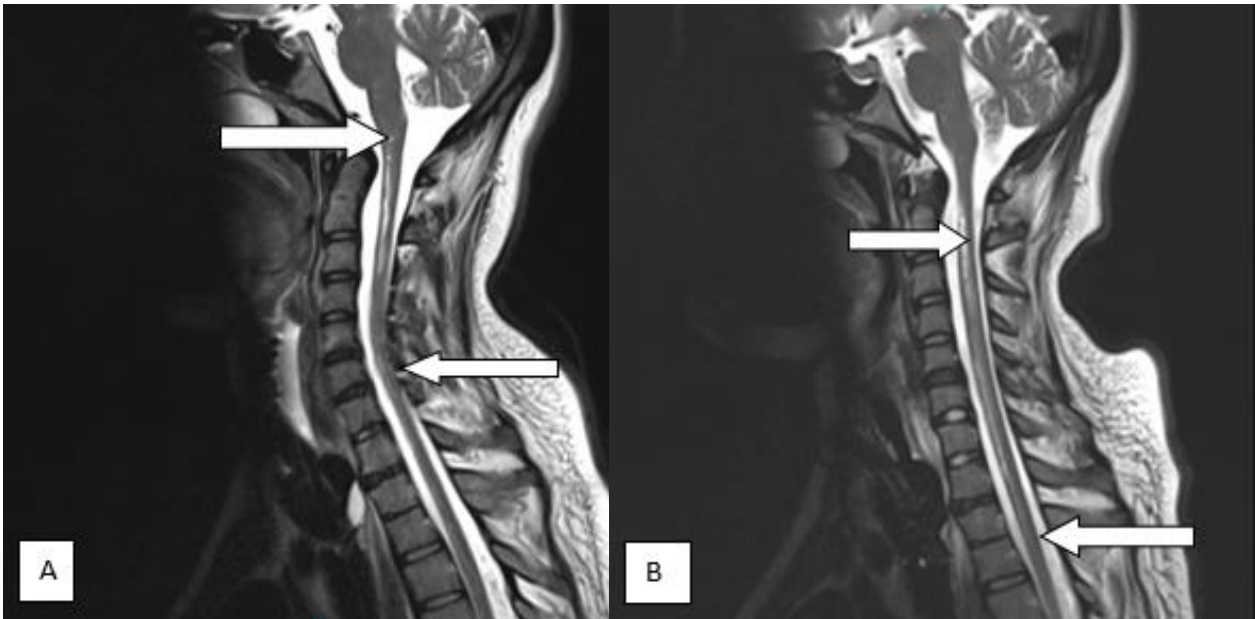


Figure 1a, b. Cranial and cervical MRI: Arrows show increased T2 hyperintense pathological signal consistent with ASA infarction starting from the bulbus level and continuing along the C1-T1 vertebra.

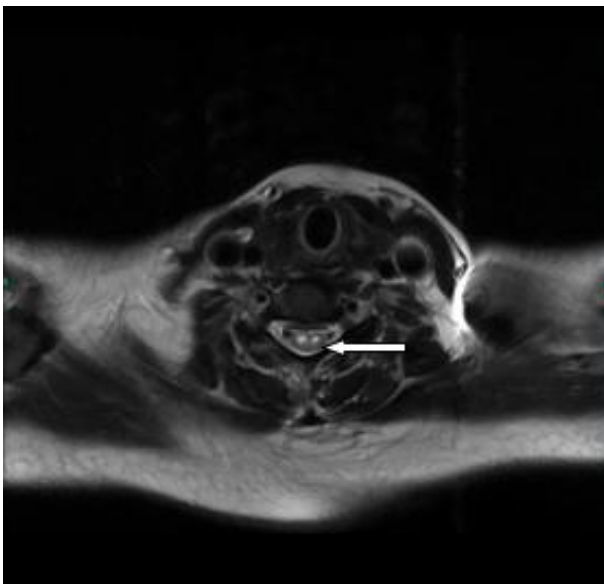
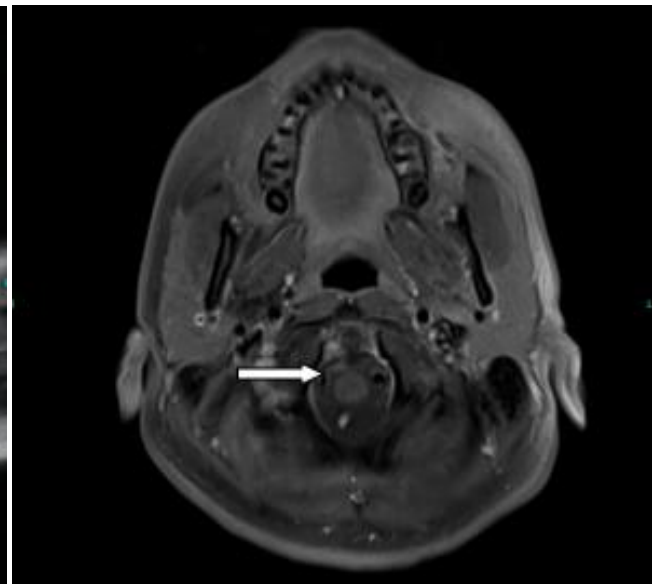


Figure 2. The arrow points to the typical 'owl eye' appearance consistent with an infarct in the center of distribution of the anterior spinal artery on spinal transverse sections, anterior to the gray matter.



Resim 3. The arrow shows the fine calibration of the right vertebral artery in the T1 axial section.

DISCUSSION AND CONCLUSION

Spinal cord infarcts are extremely rare compared to cerebral infarcts. The most important reason for this is believed to be the rich anastomotic structure in the vascular anatomy of the spinal cord (2). The fact that spinal infarcts are

primarily observed in the lower thoracic level and the conus medullaris and that the anterior spinal artery infarction is presented in different clinics according to the lesion level makes it difficult to consider in the differential diagnosis.

In our case, the history of upper respiratory

tract infection, respiratory arrest, together with the clinical picture of areflexic quadriparesis, initially suggested autoimmune diseases such as Guillain Barré syndrome and myasthenic crisis, and rapid progression required plasmapheresis treatment. Posterior spinal artery syndrome was not considered primarily because of the examination findings, and plasmapheresis treatment applied for possible longitudinal myelitis did not provide clinical improvement. Anterior spinal artery infarction, which occurred in the same period, started from the bulbus level and affected the entire cervical and upper thoracic level, causing respiratory arrest in our case, making the diagnosis difficult.

This young case, brought with respiratory arrest and had a very long segment anterior spinal infarction including the bulbus, entire cervical and upper thoracic levels, has been presented due to its rare incidence.

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Ethics

Informed Consent: The authors declared that informed consent form was signed by the patient.

Copyright Transfer Form: Copyright Transfer Form was signed by the authors.

Peer-review: Internally peer-reviewed.

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