



Contents lists available at SciVerse ScienceDirect

International Journal of Surgery Case Reports

journal homepage: www.elsevier.com/locate/ijscr

Generalized epileptic seizure in an adolescent idiopathic scoliosis (AIS) patient with syringomyelia after deformity correction surgery

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ARTICLE INFO

Article history:

Received 11 April 2013

Accepted 2 May 2013

Available online 23 May 2013

Keywords:

Adolescent idiopathic scoliosis

Epilepsy

Vertebral posterior instrumentation

ABSTRACT

INTRODUCTION: Adolescent idiopathic scoliosis and epilepsy are pathologies rarely seen together. In this study we report an AIS case we operated in which epilepsy was seen post operatively. We want to emphasize the items one should pay attention in such cases.

PRESENTATION OF CASE: In a 14-year-old girl with AIS and concomitant syringomyelia and spondylolisthesis, posterior deformity correction and fusion were performed. After stabilization the patient was discharged on the 10th day of discharge epileptic seizure appeared.

DISCUSSION: In scoliosis surgery, the mechanic stress and bleeding caused by the operation itself can cause neurological problems due to primary nervous system injury. The operation and bleeding during and after the operation, pulmonary and cardiac functional instability, metabolic imbalance can be the causes of epileptic seizures.

CONCLUSION: Epilepsy seen after a major surgery like scoliosis surgery, can be either as a result of central nervous system originated vascular and hypoxic problems or metabolic. In our case we concluded that massive hemorrhage must have induced epilepsy. In neurologic consultations the case was considered as an incidental epileptic picture.

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1. Introduction

Scoliosis is frequently idiopathic, in patients with additional neurological problems like syringomyelia complication rate is higher.¹

Syringomyelia is a cystic cavitation of the spinal cord accompanying 80% of the scoliosis cases. Usually cysts localized at the dorsal aspect of the central canal are supposed to squeeze medial nuclear groups thus effecting anterior horn cells and it is reported that at these levels trunkal muscular balance is lost, influencing scoliosis.^{2,3}

Epilepsy affects approximately 1% of the whole population and though different etiological factors are suggested, certain etiological cause cannot be obtained in most of the cases. Genetic factors are thought to play a role in the etiology of 40% of epilepsy cases which cannot be explained and which are idiopathic.^{4–6} In epileptic seizures the most common seizures are partial epileptic seizures.⁷ In epilepsy etiology, cellular metabolic mechanisms also share a part and blood Ca²⁺ levels should be closely monitored. Central nervous system space occupying lesions also should be kept in mind in

etiology. After massive hemorrhages, cerebral ischemia can trigger convulsions at the early post-operative period.^{8,9}

In our literature survey there is no reported case of epilepsy after scoliosis surgery yet known to our knowledge. In our study we report a case of rare complication of a case of with syringomyelia after corrective scoliosis surgery of an AIS patient.

2. Presentation of case

A 14-year-old female patient was admitted with a complaint of back pain, low-back pain and curvature of her trunk. Her physical examination and radiological observation revealed thoracolumbar scoliosis. Also at L5–S1 level Grade I spondylolisthesis, at C4–5 level noncompressive central disk protrusions, thoracic syringomyelia was noted.

After overall investigation and planning corrective surgery for scoliosis was performed under general anesthesia with posterior incision between Thorakal 2 and Lumbar 3 vertebrae by dissection of posterior elements and muscles. To avoid injury to the syrinx without using an osteotome, with the help of burr motors articular resections were performed. Spondylolisthesis screws were inserted at the concave side of the T5 and L1 vertebrae. T2–4 vertebrae were fixed with pedicular screws. At the convex side of the vertebrae pedicle screws were applied and instrumentation was concluded. After the rod adopted to the spondylolisthesis screws of the concave side were pulled, the fixation ended with the rod applied to

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Fig. 1. 14-Year-old female standing orthorontgenogram AP graphy. Lenke Tip3A.



Fig. 3. 14-Year-old female standing orthorontgenogram post-operative AP graphy.



Fig. 2. 14-Year-old female standing orthorontgenogram LAT graphy. Lenke Tip3A.

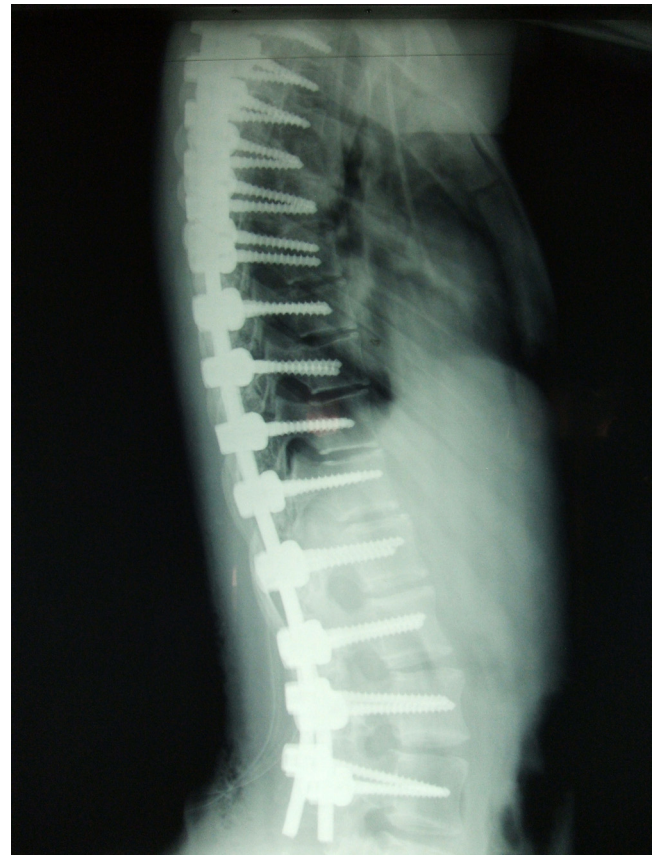


Fig. 4. 14-Year-old female standing orthorontgenogram post-operative LAT graphy.

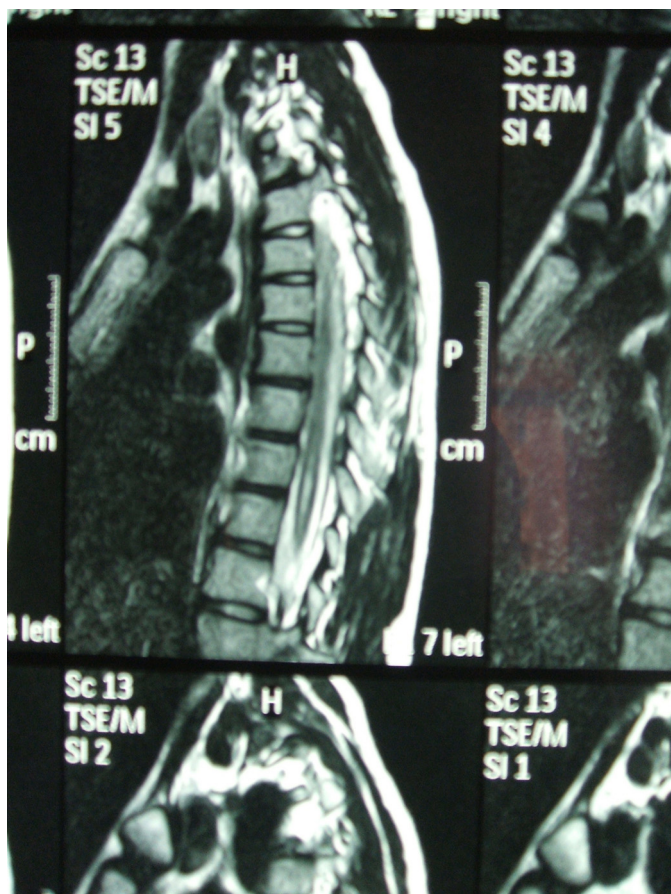


Fig. 5. MRI scan of the 14-year-old female with syringomyelia.

the convex side. After segmental derotation is performed in each vertebral segment, convex side was compressed at the apical region and concave side was distracted to conclude reduction. During decortication procedures, burr motor was used trying not to injure syringomyelia. Allograft was applied, drain was left at the surgical wound and tissues were anatomically closed. Pre-operative blood loss was 2700 ml. After the operation 6 units of erythrocyte suspension, 2 units of fresh frozen plasma were administered. She was closely monitored at the intensive care unit for 12 h and sent to the clinic after stabilization. Antibiotic prophylaxis and wound healing took 3 days' follow-up, and then the patient was mobilized. She was discharged after clinical, radiological and laboratory data were normal (Figs. 1–5).

On her admittance to the emergency unit with convulsive spasms and cyanosis on the 10th post-operative day her cranial and cervical MRI imaging was noted as normal. Her examination and laboratory findings were normal; after 2 months' follow-up epileptic seizure repeated and EEG was performed. Generalized epileptiform irregularity was detected.

Early and late laboratory findings after seizure were compared between blood Ca^{2+} levels and other blood mineral-electrolyte tests were normal.^{10,11} The transfusion amount of more than 2000 ml is accepted as massive transfusion. In our case pre-operative 4 units, post-operative 2 units of erythrocyte suspension and 2 units of fresh frozen plasma transfusion were administered.

In our case we could not verify any metabolic or central nervous system pathology to result with epileptic seizures in our work-up.

3. Discussion

In scoliosis surgery, the mechanic stress and bleeding caused by the operation itself can cause neurological problems due to primary nervous system injury. Besides we sometimes have to face more complicated problems like epileptic seizures, operation and bleeding during and after the operation, pulmonary and cardiac functional instability, metabolic imbalance can be the causes of epileptic seizures.¹² In early post-operative period change in Ca vs Mg levels, infection, subarachnoid hemorrhage can also cause epilepsy.^{10,13,14} During AIS surgery 2000–2500 ml bleeding in average is accepted as normal. This bleeding can cause hemorrhagic shock. The hypoxia due to bleeding can damage cellular functioning in the brain causing convulsive seizures. Massive blood transfusion can cause hypocalcemia which can trigger convulsions.^{14–19}

In our case we report that hypoxia depending on massive hemorrhage can be the cause of epileptic seizure.

In AIS surgery to restrict hemorrhage, primarily hypotensive anesthesia, secondarily gentle dissection and decortication of the muscles, control of blood loss, rapid blood transfusion are necessary to avoid probable neurological problems.

In our case hemorrhage amount and electrolyte levels were carefully monitored and adequate timing and transfusion rates are administered to avoid hypoxic injury.

4. Conclusion

In literature after scoliosis surgery this is the only reported case of epilepsy yet known to our knowledge. We report this case to share our experience with epilepsy problem in an AIS case and draw attention to the importance of patient history, monitoring, and follow-up. We think that in AIS operations, knowledge about hypoxic injury and problems must be added to the pre-operative patient information and consent form.

In this case we could not determine any findings to explain the cause of epilepsy and concluded that the epileptic seizure is miscellaneous.

Conflict of interest statement

None.

Funding

None.

Ethical approval

Written informed consent was obtained from the patient and her family for publication of this case report and accompanying images. A copy of the written consent is available for review by Editor in Chief of this journal on request.

Author contributions

Gultekin Sıtkı CECEN: design the study, writing; Deniz Gulabi: writing, review; Ismail Oltulu: data collection; Tolga Onay: data collection.

References

- Weiss HR, Goodall D. Rate of complications in scoliosis surgery. *Scoliosis* 2008;5(3):9, <http://dx.doi.org/10.1186/1748-7161-3/9>.
- Williams B. Orthopaedic features in the presentation of syringomyelia. *Journal of Bone and Joint Surgery* 1979;61B:314.

3. Huebert HT, MacKinnon WB. Syringomyelia and scoliosis. *Journal of Bone and Joint Surgery* 1969;**51B**:338.
4. Browne TR, Holmes GL. Handbook of epilepsy. Epilepsy: definitions and background. 3rd edition, USA; 2004:6–7.
5. Guberman A, Bruni J. Essentials of clinical epilepsy. Epidemiology. 2nd edition, USA; 1999.
6. Cockerell CO, Shorvon SD. Epilepsy current concepts. Epidemiology. London; 1996.
7. Walter N, Bradley G, Robert B, Daroff, Gerald M, Fenichel C, et al. Eurology in clinical practice. *Epilepsies* 2000;**71**:1745.
8. Janicki J, Alman B. Scoliosis: review of diagnosis and treatment. *Paediatric Child Health* 2007;**12**(November (9)):771–6.
9. Stawicki TM, Zhou K, Yochem J, Chen L, Jin Y. TRPM channels modulate epileptic-like convulsions via systemic ion homeostasis. *Current Biology* 2011;**21**(10):883–8.
10. Marino D, Vatti G, Rufa A, Malandrini A, Rocchi R, Bracco S, et al. Transient periodic lateralised epileptiform discharges (PLEDs) following internal carotid artery stenting. *Epileptic Disorders* 2012;**14**(1):85–9.
11. Drummond JC, Petrovitch CT. The massively bleeding patient. *Anesthesiology Clinics of North America* 2001;**19**(4):633–49.
12. Kruskall MS, Mintz PD, Bergin JJ, Johnston MF, Klein HG, Miller JD, et al. Transfusion therapy in emergency medicine. *Annals of Emergency Medicine* 1988;**17**(4):327–35.
13. Xing J, Yan X, Estevez A, Strange K. Highly Ca²⁺-selective TRPM channels regulate IP3-dependent oscillatory Ca²⁺ signaling in the *C. elegans* intestine. *Journal of General Physiology* 2008;**131**(3):245–55.
14. Ho KM, Leonard AD. Concentration-dependent effect of hypocalcaemia on mortality of patients with critical bleeding requiring massive transfusion: a cohort study. *Anaesthesia and Intensive Care* 2011;**39**(1):46–54.
15. Ho KM, Leonard A. Risk factors and outcome associated with hypomagnesemia in massive transfusion. *Transfusion* 2011;**51**(February (2)):270–6.
16. Wilson RF, Binkley LE, Sabo Jr FM, Wilson JA, Munkarah MM, Dulchavsky SA, et al. Electrolyte and acid–base changes with massive blood transfusions. *American Surgeon* 1992;**58**(9):535–44.
17. Rutledge R, Sheldon GF, Collins ML. Massive transfusion. *Critical Care Clinics* 1986;**2**(October (4)):791–805.
18. Sihler KC, Napolitano LM. Complications of massive transfusion. *Chest* 2010;**137**(January (1)):209–20, <http://dx.doi.org/10.1378/chest.09-0252>.
19. Lier H, Krep H, Schroeder S, Stuber FJ. Preconditions of hemostasis in trauma: a review. The influence of acidosis, hypocalcemia, anemia, and hypothermia on functional hemostasis in trauma. *Trauma* 2008;**65**(October (4)):951–60.