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CLINICAL INFORMATION

Cerebellar infarct following orchidopexy under spinal anesthesia



Sunny Goel^a, Gaurav Garg^{b,*}, Manoj Kumar^a, Ruchir Aeron^a

^a King George's Medical University, Lucknow, India

^b King George's Medical University, Department of Urology, Lucknow, India

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KEYWORDS

Ischemia;
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Abstract The report describes a case of peri-operative stroke that presented as diplopia and gait difficulty on 2nd post-operative day after routine orchidopexy under spinal anesthesia in an otherwise healthy young boy. Magnetic resonance imaging of the brain revealed acute infarct in bilateral cerebellar hemispheres, left half of medulla and left thalamus. A diagnosis of acute stroke (infarct) was made and patient was started on oral aspirin 75 mg.day⁻¹, following which his vision started improving after 2 weeks. Possible mechanisms of development of stroke in the peri-operative period are discussed, but, even after extensive investigations, the etiology of infarct may be difficult to determine. Acute infarct after elective non-cardiac, non-neurological surgery is rare; it may not be possible to identify the etiology in all cases. Clinicians must have a high index of suspicion to diagnose such unexpected complications even after routine surgical procedures in order to decrease the morbidity and long term sequelae.

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PALAVRAS-CHAVE

Isquemia;
Acidente vascular cerebral;
Orquidopexia;
Raquiianestesia

Infarto cerebelar após orquidopexia sob raquiianestesia

Resumo O presente relato descreve um caso de acidente vascular cerebral perioperatório que resultou em diplopia e dificuldade de marcha no segundo dia após orquidopexia de rotina sob raquiianestesia em um jovem, em outros aspectos, saudável. Ressonância magnética cerebral revelou infarto agudo em hemisférios cerebelares bilaterais, metade esquerda do bulbo e tálamo esquerdo. Um diagnóstico de acidente vascular cerebral agudo (infarto) foi feito, e o paciente começou a receber tratamento com aspirina oral (75 mg.dia⁻¹), após o qual sua visão começou a melhorar após duas semanas. Possíveis mecanismos de desenvolvimento de acidente vascular cerebral no período perioperatório são discutidos, mas, mesmo após extensas investigações, a etiologia do infarto pode ser difícil de determinar. O infarto agudo após cirurgia eletiva não cardíaca e não neurológica é raro; talvez não seja possível identificar a etiologia

* Corresponding author.

E-mail: gougarg@gmail.com (G. Garg).

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em todos os casos. Os médicos devem ter um elevado grau de suspeita para diagnosticar essas complicações inesperadas, mesmo após procedimentos cirúrgicos de rotina, para diminuir a morbidade e as sequelas a longo prazo.

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Introduction

Peri-operative stroke after non-cardiac, non-neurosurgical procedures is very rare and reported to have an incidence of around 0.05–7%.¹ In the majority of patients, it is reported in the postoperative period after discharge and the etiology is presumed to be mostly thrombotic in nature.¹ This case represents a routine elective surgery with an unexpected complication. The lesson learned is that there should be a high index of suspicion to diagnose such unexpected complications even after routine surgical procedures.

Case presentation

A 10-year-old boy was admitted in June 2016 with a diagnosis of bilateral undescended testes in a tertiary care center in North India. He had no vascular risk factors including previous history of blood dyscrasias like sickle cell disease, cardiovascular or metabolic diseases. The child was fairly built (weight, 30 kg; height, 120 cm; BMI, 20.8 kg.m⁻²). The baseline heart rate was 96 min and blood pressure was 104/65 mmHg. His general physical examination was unremarkable and on local examination, he was found to have rudimentary scrotum, bilateral inguinal testis with normal penile length as per age. He underwent bilateral orchidopexy under spinal anesthesia. After sedation with intravenous midazolam 1 mg spinal anesthesia was given using a 25 gauge Whitacre spinal needle at the L3–L4 interspace, through which hyperbaric bupivacaine 12 mg and fentanyl 25 mg was administered intrathecally. The spinal anesthesia level for sensory blockade was reached up to T10 spinal level and for touch was reached up to T6 spinal level. Apart from these, the only other medication that the boy received was injection cefazolin 500 mg intravenously (i.v.) as prophylactic antibiotic. The intraoperative course was uneventful. The patient received 800 mL of intravenous fluid solution (Ringer lactate), and there was minimal blood loss. The effect of spinal anesthesia blockade lasted for around 2.5 h. The vital parameters viz. heart rate, respiratory rate, and blood pressure were maintained throughout the procedure within their prescribed normal limits for his age (documented records revealed that the mean blood pressure never dropped below 65 mmHg while the least systolic blood pressure recording noted was of 90 mmHg). On 2nd post-operative day, he complained of diplopia and difficulty in walking. There was no associated fever, neck rigidity, seizures or vomiting. On examination, his vital parameters were found to be temperature, 37°C, pulse rate,

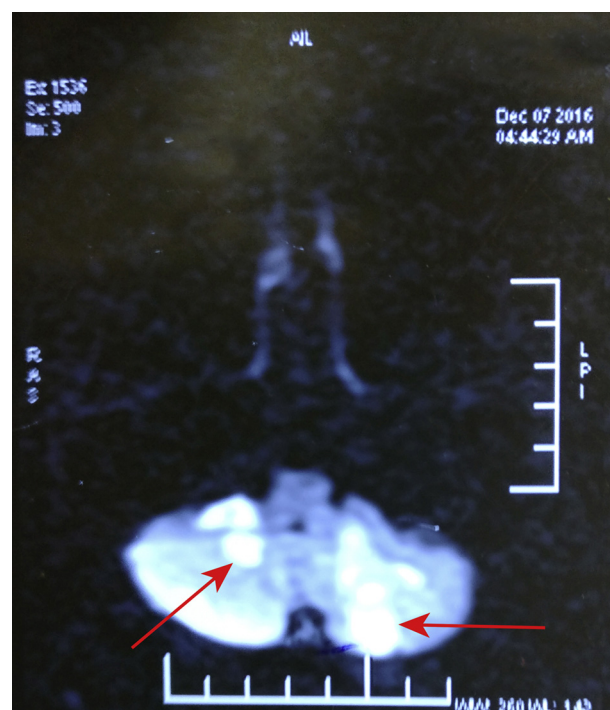


Figure 1 Multiple small, discrete and confluent areas of true diffusion restriction are seen involving bilateral cerebellar hemispheres suggestive of acute infarct (red arrows).

92– (normal range: 80–120 min), blood pressure: 107/78 mmHg (normal systolic pressure: 102–120 mmHg; normal diastolic pressure: 61–80 mmHg), respiratory rate, 16 min (normal respiratory rate: 20–25 min), and Glasgow Coma Scale, 15/15. Ophthalmological examination revealed bilateral normal-sized pupils reactive to light (direct and consensual). There was decreased visual acuity, nystagmus in all four gazes, left-sided convergent latent squint with positive Romberg sign (cerebellar ataxia positive); the fundoscopic examination was normal. The rest of the neurological examination was unremarkable. There were no clinical features suggestive of deep vein thrombosis. The patient was referred to neurology department and was advised an urgent magnetic resonance imaging (MRI) of the brain. MRI (fluid-attenuated inversion recovery T1, weighted and fast-spin echo T2-weighted sequences) revealed multiple small acute infarcts in bilateral cerebellar hemispheres, left half of the medulla and left thalamus (both the AICA and PICA territories) (Fig. 1),

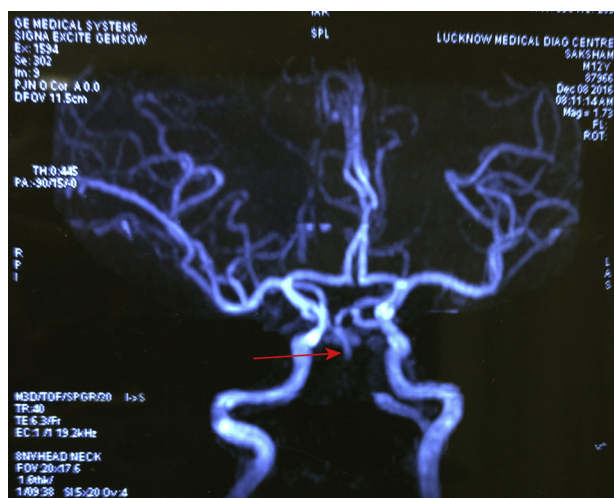


Figure 2 3D-TOF MR angiography image showing non-visualization of flow-related enhancement of bilateral vertebral and proximal portion of basilar artery (distal portion of basilar artery is marked by red arrow).

MR angiography showed non-visualization of bilateral vertebral and proximal portion of basilar artery (Fig. 2) and reformation of the bilateral posterior cerebral artery by bilateral posterior communicating arteries (Fig. 3). There was no evidence of congenital arterial anomalies. MRI of the orbits was unremarkable. Echocardiography revealed patent foramen ovale (left to right shunt). MRI of the lumbosacral spine was normal. CT aortography was also normal. The prothrombin time; activated partial thromboplastin time and serum levels of protein C, protein S, and antithrombin III were normal. D-dimer and ESR were also normal. Other blood studies including general blood picture, hemoglobin electrophoresis, APLA profile, PNH panel, ANA and serum homocysteine were also normal. A possibility of peri-operative acute ischemic stroke was kept. The patient was started oral aspirin 75 mg.day⁻¹,



Figure 3 3D-TOF MR Angiography image showing bilateral posterior cerebral artery (yellow arrowheads) reformed by bilateral posterior communicating arteries (red arrowheads).

following which his vision started improving after 2 weeks. The patient was discharged after 2 weeks on tab Aspirin 75 mg.day⁻¹ for 3 months. After 6 months, the patient had improved with respect to vision and gait.

Discussion

Peri-operative stroke is a serious condition and can have a distressing short- and long-term sequelae unless timely treatment is done. Peri-operative stroke is defined as brain infarction of ischemic or hemorrhagic etiology that occurs during surgery or within a time period of 30 days post-surgery.² There are various risk factors associated with the development of peri-operative stroke such as certain high-risk surgical procedures, co-existing vascular disease, intra-operative hypotension, blood dyscrasias like sickle cell disease.¹ It is uncommon after non-cardiac, non-neurological and minor surgical procedures. However, to the best of our knowledge, there are no cases of peri-operative stroke reported in children so far. Lee et al. reported a case of cerebellar infarction in a 48-year-old man caused by epidural abscess after epidural neuroplasty.³ The patient developed fever, headache and confusion with focal neurological signs of stiff neck 3 days after the procedure. A brain MRI revealed the presence of a left cerebellar infarction.³ In the author's case, the possible mechanism for development of stroke may be due to cerebral vasculopathy secondary to bacterial meningitis that may have developed secondary to epidural abscess. There is another case report by Al-Asmi et al. in which the authors have described a case of a 28-year-old woman who developed extensive spinal cord infarction after treatment for liver hematoma and was given post-operative epidural analgesia.⁴ She had no vascular risk factors for stroke. Eight hours after the initiation of epidural analgesia, she developed both sensory and motor weakness in her lower limbs along with bowel and bladder dysfunction. MRI of the spine done in this patient revealed diffuse hyperintensity of the distal spinal cord involving the central gray matter on the left side.⁴ Possible mechanisms leading to the development of neurological deficits after spinal or epidural anesthesia may also include cord compression secondary to epidural hematoma, injury to epidural vessels supplying the cord, contamination, and iatrogenic meningitis.⁵⁻⁷ Overell et al. did a meta-analysis of case-control studies on inter-atrial septum abnormalities and stroke and proposed that patent foramen ovale can result in cryptogenic stroke in individuals less than 55 years of age adults secondary to paradoxical embolism via patent foramen ovale.⁸ The mechanisms underlying peri-operative stroke during spinal surgery are less clearly defined. Mione et al. described the case of a 55-year-old male patient who presented with bilateral occipital watershed ischemic strokes following lumbar laminectomy surgery performed for spinal stenosis.⁹ The authors proposed that intra-operative hypotension on a variation in anatomy of circle of Willis (fetal origin of both posterior cerebral arteries with hypoplasia of proximal segments) may decrease the cerebral blood flow, leading to stroke.⁹ Zheng et al. reported clinical data and imaging characteristics of 178 patients with cerebral infarction.¹⁰ The authors reported that hemodynamic abnormalities caused by vascular lesions may decrease cerebral perfusion

pressure, and may lead to accumulation of microemboli in the vascular periphery, leading to infarction in watershed area of supplying cerebral vessels.¹⁰ The authors proposed that patients with cortical watershed infarctions have high prevalence of stenosis in major blood vessels, and an examination of extracranial vessels especially the extracranial segment of the vertebral artery must be performed so that an early intervention and further progression of stroke can be prevented.¹⁰ However in our patient there was no evidence of any cerebral vessels anatomic variation and neither any episode of documented intra-operative hypotension was present. We can only hypothesize that cerebellar stroke may have been caused due to combination of factors like patent foramen ovale, co-existing vascular disease (non-visualization of bilateral vertebral and proximal portion of basilar artery) combined with un-noticed blood pressure oscillations during spinal anesthesia. Despite extensive workup, the exact etiology of stroke could not be determined in the present case.

Conclusion

Acute infarct after elective non-cardiac, non-neurological surgery is rare; it may not be possible to identify the etiology in all cases. Clinicians must have a high index of suspicion to diagnose and treat these unexpected complications in order to decrease the morbidity and long-term sequelae. Subtle findings like asymptomatic patent foramen ovale (L to R shunt) can result in cryptogenic stroke secondary to paradoxical embolism.

Conflicts of interest

The authors declare no conflicts of interest.

Acknowledgments

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References

1. Ng JL, Chan MT, Gelb AW. Perioperative stroke in noncardiac, nonneurosurgical surgery. *Anesthesiology*. 2011;115:879–90.
2. Sacco RL, Kasner SE, Broderick JP, et al. An updated definition of stroke for the 21st century. *Stroke*. 2013;44:2064–89.
3. Lee HY, Wang HS, Kim SW, et al. Cerebellar infarction following epidural abscess after epidural neuroplasty. *Korean J Spine*. 2015;12:26–8.
4. Al-Asmi A, John R, Nandhagopal R, et al. Spinal cord infarction following abdominal surgery and postoperative epidural analgesia. *Sultan Qaboos Univ Med J*. 2010;10:396.
5. Kane RE. Neurologic deficits following epidural or spinal anesthesia. *Anesth Analg*. 1981;60:150–61.
6. Brull R, McCartney CJ, Chan VW, et al. Neurological complications after regional anesthesia: contemporary estimates of risk. *Anesth Analg*. 2007;104:965–74.
7. Bromage PR, Benumof JL. Paraplegia following intracord injection during attempted epidural anesthesia under general anesthesia. *Reg Anesth Pain Med*. 1998;23:104–7.
8. Overell JR, Bone I, Lees KR. Interatrial septal abnormalities and stroke: a meta-analysis of case-control studies. *Neurology*. 2000;55:1172–9.
9. Mione G, Pische G, Wolff V, et al. Perioperative bioccipital watershed strokes in bilateral fetal posterior cerebral arteries during spinal surgery. *World Neurosurg*. 2016;85:e17- e21.
10. Zheng M, Sun A, Sun Q, et al. Clinical and imaging analysis of a cerebellar watershed infarction. *Chinese Med*. 2015;6:6:54.