

Holmes-Adie Syndrome in the Setting of Induced Hypothermia is Associated with Delayed Recovery from General Anesthesia

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Abstract

Holmes Adie syndrome is a relatively rare condition that results in a tonic pupil which exhibits no response to light, relatively preserved accommodation reflex and focal or generalized are flexi. The condition was first described by Gordon Holmes & William John Adie [1,2].

Keywords: Holmes-Adie Syndrome; Anesthesia; Hypothermia; Minimally Invasive Aortic Valve Replacement

Case Report

Here we describe a case of a 67 year old man with Adies Syndrome and delayed recovery from general anesthesia. Consent was obtained from the patient to submit this report for publication. The patient had a minimally invasive aortic valve replacement which was performed in the setting of induced hypothermia (32°C) for approximately 90 minutes. He received midazolam, sufentanyl and the paralytic agent, cisatracurium. After completion of the procedure, he was re warmed to physiologic temperature and brought back to the recovery room. When the patient did not arouse, his pupils were examined and anisocoria was discovered. Stroke was suspected at the time and we were called in to assess the patient. On examination, the patient was not responsive to verbal or painful stimulation. His pupils were asymmetric with the left pupil measuring approximately 5mm and not reactive to light and the right pupil approximately 2mm and sluggishly reactive to light. His knee and ankle jerks were unelicitable. No imaging was ordered at the time as we felt that he was most likely a slow metabolizer of the anesthetic agents and we also felt that the anisocoria represented Adies tonic pupil. When the family was interviewed, they noted that they were aware of the anisocoria and that it was long-standing for 3 years. After approximately 4h post-operatively, the patient began to slowly arouse. He became fully responsive to verbal commands and was back to his baseline with no neurological deficits apart from the pupillary findings which were now sluggishly reactive to accommodation and the absent knee and ankle jerks.

Conclusion

Several case reports have described anisocoria exacerbated by anesthetic agents in the setting of Adie's syndrome [3,4]. The authors in one such report postulate that the exacerbation of anisocoria by anesthetic agents may be due to parasympathetic predominance in the setting of anesthesia [4]. Interestingly, another case report described Adie's syndrome in association with a cranial neuropathy and coined the term "Adie's plus syndrome". The authors speculated that perhaps Adie's syndrome involves more widespread disturbances than previously thought [5]. Our report seems to support this notion as well, as this case was associated with a prolonged and delayed recovery from anesthesia. We postulated that hypothermia may have slowed down the metabolic rate and therefore the rate of anesthetic elimination in our patient. However, in our discussions with the anesthesiologist, he confirmed that other patients undergoing a similar procedure did not exhibit a similar reaction. We feel that perhaps Adie's syndrome may involve previously unrecognized altered central neural networks and that in the setting of anesthesia, paralytics and induced hypothermia manifests as markedly delayed recovery from anesthesia.

References

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