

SPEECHLESS AFTER GENERAL ANAESTHESIA FOR CAESAREAN SECTION

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Abstract

‘Speechless’ patient after general anaesthesia may be a real horror for the anaesthetist as well as the patient and his relatives. Whatever the cause “functional or organic” the anaesthetist will be under pressure as his patient is not able to talk. Here we report a 40 years old patient who has no history of medical problems and developed aphemia after general anaesthesia for emergency caesarean section with an uneventful intra-operative course. Clinical examinations and investigations failed to reveal any clear cause and the patient returned her ability to talk and discharged home with normal voice.

Key words: Speechless, Aphemia, General Anaesthesia, Caesarean Section.

Introduction

It is a Medico-legal landmine for the anaesthesiologist when the patient is waking up ‘speechless’ unexpectedly after general anaesthesia. Hence, it is imperative to examine the potential causes, in order to guide appropriate clinical management, assuage anxious patient relatives and pacify fellow surgical colleagues. The term aphemia is used for one of these presentations. It means an acute onset of severely impaired fluency of speech (dysarthria/mutism) when writing, reading, normal repetition and comprehension are intact, so this is not a true ‘aphasia’ syndrome^{1,2}. Here we report a case of aphemia or speechlessness in a 40 years old patient who underwent an emergency caesarean section for twin pregnancy.

Case report

A 40 year old patient underwent an emergency caesarean section for twin pregnancy. Patient was anxious but verbalizing well when wheeled into the operating room for surgery. She had no previous general anaesthesia and she denied any history of other medical problems. General anaesthesia was decided for her as she refused spinal anaesthesia. A rapid sequence general anaesthetic was administered with propofol-suxamethonium and maintained with sevoflurane and cisatracurium. Fentanyl was given after delivery of baby. Intra-operative vitals were maintained stable. Recovery was uneventful initially and postoperative analgesia “morphine” was given. In postanesthesia care unit the patient stayed free of pain and able to communicate with the nurses. About thirty minutes later, the patient was shifted to the ward then she suddenly became agitated

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and “speechless” with no respiratory stridor or distress. We immediately tried to reassure the patient and her family.

Laboratory values (hematocrit, glucose, urea, Ca, Mg, K, and blood gases analysis) were obtained and were within normal range in the early postoperative period. Neurology consultation established a provisional diagnosis of aphemia. She was found to be writing out her problems. The loss of speech was not associated with any other sensorimotor or autonomic neurological deficit. MRI-brain showed only old non specific white matter changes. Electroencephalography did not show any specific changes. Otolaryngology examination could not establish any cause in the larynx or vocal cords for her problem. Psychiatry review assessed her as being conscious, euthymic, with no social stressors and no similar past problems or family history.

On the second post-operative day, patient was found to be cheerful, awake and resumed spontaneous speech; initially starting out with soft, low volume voice and then progressing on to normal speech. After two days, clinical re-evaluation by otolaryngologist, neurologist and psychiatrist did not reveal a clear cause for her presentation. The patient was scheduled for psychiatric outpatient follow up after discharge. Further investigations were called off by the admitting team, partly because of no residual speech deficit and otherwise normal vital signs and behavior. However on the subsequent day, she had fresh complaint of blurring of vision. Ophthalmology evaluation diagnosed keratoconus, with normal intra-orbital pressure but with refractory changes. This finding was attributed to post-partum hormonal changes and scheduled for review after one month. Patient had wound hematoma which was treated conservatively. Patient was discharged home nine days after surgery.

Discussion

Speechless during the peri-partum period is not an uncommon entity; however it is more often reported as aphasia rather than aphemia. The main cause ascribed being Cerebral Venous Sinus Thrombosis (CVST). CVST incidence increases in relation to the oral contraceptive pill, pregnancy and puerperium. CVST generally carries a favourable prognosis. Usually the

clinical course of this disease is progressive but tends to become stable or begin to subside within 5 to 10 days. Obstetric patients seem to do well also, and if occurring during peripartum, there is no evidence that future pregnancies should be avoided. This diagnosis was excluded in our case as MRI did not show any suggestive features of CVST and the patient improved dramatically within 24 hours³.

Lesser known but equally dramatic and short-lived neurological deficits occur due to transient vasospasm, termed Reversible Cerebral Vasoconstriction Syndromes (RCVS) or Cerebral Angiopathy Syndromes. It is a reversible multifocal spasm of the cerebral arteries which may be misdiagnosed as primary cerebral vasculitis and aneurysmal subarachnoid hemorrhage. RCVS is usually self-limited over a period of days to weeks. RCVS is characterized by recurrent thunderclap headache which is not present in our patient.⁽⁴⁾

Postpartum angiopathy (Call-Fleming syndrome) is a rare, reversible cerebral vasoconstriction syndrome affecting women around the time of pregnancy. It can develop within 1 to 3 weeks of delivery. Although headache is the main feature in this type of angiopathy, but others like cortical blindness, seizure, hemiplegia, dysarthria, aphasia, numbness, and ataxia have been reported, with a case-report of a 17-year-old woman. Spontaneous resolution of symptoms occurs with excellent prognosis. A number of migraine-like syndromes also fall in this category of cerebral angiopathy⁵⁻⁶.

Mutisim was reported as a rare side effect of propofol anaesthesia. The reported case was for patient received a Total Intravenous Anaesthesia with propofol for orthopaedic procedure. After recovery from anaesthesia the patient could respond to verbal commands for eye opening and nodding, but she could not speak for eleven days. The inability to speak was associated with brain oedema in CT scan. While in our patient no oedema was seen in brain MRI and propofol used only during induction of anaesthesia⁷.

If organic brain lesions are excluded, the most common cause would be functional disorder, most often clubbed under the umbrella term “Puerperal Psychosis”. A family history or past relevant pointers to Psychotic/Neurotic behavior add weightage to

this diagnosis. Aphemia and aphonia have been described as one of the pseudo-neurologic symptoms in the Complex of Somatization Disorder. Symptoms are inconsistent but the deficit is not intentionally produced or simulated, as happens with malingering. Successful treatment is best achieved through behavior modification. Any attempt to confront the patient usually creates a sense of humiliation and causes the patient to abandon treatment from that caregiver. In some patients, anti-depressants are helpful¹.

Diagnosis for such cases has to be established through a multi-pronged approach due to the possibility of organic brain lesions and if present, to treat them vigorously before ascribing it to functional disorder. The medico-legal fallout is a major professional challenge to the healthcare providers. Common suspicion being effect of anaesthetic drugs, local injury to vocal cords due to airway instrumentation, possible rupture of AVMs or

berry aneurysm, and severe protracted intra-operative hypotension leading to ischemic insult to brain tissue. The onus of providing proof and absolving oneself, still unfairly lies with the anesthesia professional. We should keep in mind the possibility of malingering by patients, quite often to generate attention.

Conclusion

Very often, little time is available to either elicit a detailed history or to perform a credible detailed clinical examination when the urgency of the surgery is paramount, as was the case in this instance. Finding the cause of neuropsychological presentation after general anesthesia might be a real challenge for the anaesthesiologist and the primary admitting team. A multi-pronged approach may be required for diagnosis and further management.

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