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CASE STUDY

Paravertebral block as a sole technique for the anaesthetic management of a patient with myalgic encephalomyelitis undergoing breast cancer surgery

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Myalgic encephalomyelitis (ME) is a multifaceted organic disease which, owing to its non-specific multiple symptoms that include incapacitating fatigue, deeply affects the quality of life of diseased patients. It carries a perceived risk of an adverse reaction to drugs, including anaesthetics. However, there is very little information in the medical literature on the anaesthetic management and outcomes of patients with this condition. According to current scientific literature, there is no causal relationship between ME relapse and anaesthesia, surgery or both. We present the anaesthetic management of a ME patient who underwent breast cancer surgery.

Keywords: breast cancer surgery, chronic fatigue syndrome, myalgic encephalomyelitis, paravertebral block

Introduction

The term "myalgic encephalomyelitis" (ME), was first included by the World Health Organization (WHO) in its international classification of diseases in 1969. Other name labels, such as post-viral fatigue syndrome, idiopathic environmental intolerance syndrome (IEI), multiple chemical sensitivity syndrome and chronic fatigue syndrome (CFS), have been historically used to refer to ME, possibly because of inadequate knowledge of the disease aetiopathogenesis. Furthermore, the disease has been described as a "twentieth century disease" or "contested illness", leading to conflict and mistrust between sufferers and healthcare professionals.[1] In 2007, the WHO acknowledged ME as a disease of the nervous system, under the code G93.3. Recent research and clinical experience with these patients has resulted in the identification of unique and distinctive characteristic patterns of symptom clusters of ME, as set out in the international consensus criteria, published by an international group of clinicians, researchers, a teaching faculty and patient advocates in 2011.[2] This document provides a clear understanding of the complexity of this disease. It also offers clarity on diagnosis and clinical treatment criteria, based on current knowledge.

It is now scientifically proven that ME is a multifaceted organic disease with a sudden onset after a viral infection in a previously fit and healthy individual, and involves significant dysregulation of the central nervous, cardiovascular and immune systems, cellular metabolism and ion transport mechanisms.[2,3] Characteristically, it presents with incapacitating fatigue not improved by rest, and multiple other neurological, cognitive, immune, metabolic, cardiovascular, respiratory, gastrointestinal and/or genitourinary symptoms.[2] The incidence of ME is estimated to be 0.5 % in the Western world,[4] and it affects females twice as commonly as males, with a peak in the most productive part of the adult life, therefore representing a significant burden to both the diseased patient and society. When a ME patient is exposed to intervening stress, such as a surgical intervention, the effect on the recurrence and aggravation of symptoms can be unpredictable, and postoperative recovery may be prolonged. We present the anaesthetic management of a ME patient who underwent breast cancer surgery.

Case report

A 61-year-old woman, diagnosed with ME in the early 1990s, maintained normal essential physiology since, except for occasional intermittent episodes of significantly reduced energy levels. Her medical history was otherwise insignificant. Calcium supplements were her only regular medication. Previous general anaesthesia for a minor gynaecological procedure 20 years previously resulted in a severe ME relapse, with substantial energy loss, impaired vision, tinnitus, balance impairment and headaches which lasted for two years thereafter.

On this occasion, she was diagnosed with high-grade ductal carcinoma in situ, under the auspices of the breast screening programme, and scheduled for a wide local excision with sentinel lymph node biopsy. She was referred to the anaesthesia assessment clinic for assessment and planning of imminent anaesthesia management. The preoperative physical examination, laboratory tests and investigations were all within normal limits.

From the outset, the patient refused the option of general anaesthesia and/or sedation for the scheduled procedure. Therefore, we discussed the option of a paravertebral blockade (PVB) and/or deep tissue infiltration with a local anaesthetic with her. Regional technique failure rates and complications, as well as the possibility of conversion to general anaesthesia, were thoroughly discussed with the patient.

On the day of surgery, after intravenous cannulation and monitoring, a right-sided paravertebral block was performed at T3 level in a left lateral position, using a landmark technique. After skin infiltration with lignocaine 1% of an area 2.5 cm lateral to the spinous process of T3 vertebra, an 18-G Tuohy needle was inserted perpendicular to all planes until the transverse process of T4 vertebra was met at a depth of 3.7 cm. The needle was then walked off the upper border of the T4 transverse process, and advanced a further 0.3 cm until a subtle loss of resistance to normal saline 0.9% was felt. A test dose of bupivacaine 0.375% 3 ml was injected, after which a catheter was inserted 5 cm in the paravertebral space. Further boluses of bupivacaine 0.375% were injected in 5 ml increments, up to

a total of 20 ml. A right-sided sensory blockade was identified from T1-T8 at both the midclavicular and midaxillary lines, after which surgery proceeded.

Intraoperatively, the patient remained haemodynamically stable and the surgery advanced uneventfully. She received further deep tissue infiltration with lignocaine 1% 20 ml for some minor discomfort in the axillary area. Since the patient refused benzodiazepines, nonsteroidal anti-inflammatory drugs and opioid analgesics, an increased dose of paracetamol 2 g was administered intravenously. Although unconventional, such a loading dose has been suggested to improve immediate postoperative analgesia without increased toxicity in healthy adults. The paravertebral catheter was removed at the end of surgery as per our institutional protocol.

Postoperatively, the patient was comfortable, and one more oral dose of paracetamol 1 g was required overnight for minor pain. She recovered well and was discharged home the next day without any complications. Follow-up at two weeks after discharge showed that she had recovered well, without ME relapse.

Discussion

There is little published medical literature on the anaesthetic management and outcome of ME patients. Yet, anecdotal available evidence and information on a patient advocacy website[5] suggests that ME patients may experience episodes of acute exacerbation of the disease after anaesthesia, which may be long term or even permanent. Alleged postoperative symptoms may include intense fatigue with an inability to perform daily activities for several years after surgery. Such a relapse was experienced by our patient after a minor gynaecological procedure under general anaesthesia which had been performed 20 years prior to the current surgical intervention. Because of such patient reports, and historical mistrust between sufferers and healthcare professionals, it is not surprising that patients present for surgery with misconceptions, and sometimes a checklist of medications and actions which are assumed to be accepted practice in the anaesthetic management of ME patients.[5]

A few other case reports describe anaesthesia in ME patients. Meenakshi and Kumar reported the anaesthetic management of two ME female patients who underwent gynaecological procedures under spinal, followed by general anaesthesia, for incomplete sensory blockade.[6] Both patients experienced an uneventful recovery and were discharged home without any further complications. In another case report, a patient with "sick house" or "sick building" syndrome (a ME-like syndrome, characterised by non-specific complaints associated with indoor environmental chemical pollution), developed non-anaphylactoid bronchospasm on two occasions after propofol, and sudden intermittent severe headaches after surgery.[7] On a third and fourth occasion, propofol was avoided, and no further bronchospasm occurred.

However, the best available scientific evidence for possible interaction between anaesthesia and ME derives from a retrospective observational study and literature review by Fisher and Rose, published in the *British Journal of Anaesthesia* in 2008.[8] Of twenty-seven patients with CFS and IEI, referred to an anaesthetic clinic over a 20-year period, 23 had reported a history of problems relating to previous local anaesthesia, general anaesthesia or both. The historical anaesthetic records of 11 such patients demonstrated minor complications during the immediate perioperative period, such as intra- and postoperative hypotension responding to intravenous fluids, hypertension, hypoglycaemia and slightly delayed awakening. Several patients complained of delayed symptoms, such as panic attacks, uncontrollable shaking, protracted nausea, muscular pain, tiredness and weakness; all self-limiting and not requiring further medical intervention. One patient reported untoward non-specific effects after both an epidural and general anaesthesia, whereas some patients reacted to local anaesthetics with vasovagal symptoms, swelling, hallucinations and fatigue. The authors proceeded with skin testing for general anaesthetic drugs, local anaesthetics or both. The results of these were non-specific, i.e. vasovagal reactions, a skin rash on the opposite arm, and an emotional breakdown in one patient, and bore no scientifically sound relationship to the tested anaesthetic drugs. Finally, the authors followed-up these patients through subsequent anaesthetics, where both local and general anaesthetics were used. Only one patient of the initial 27 experienced complications including narcolepsy, neurocognitive impairment, neuromotor disturbances and seizures. None of them required further medical referral. Further to a subsequent major surgical intervention, the same patient experienced nausea only. At the same time, Fisher and Rose[8] also performed a comprehensive literature review of all published data over a 57-year period (1950-2007). They concluded that there was insufficient evidence to support a causal relationship between the reported non-specific responses and anaesthesia, surgery or a combination of both. There was no evidence that the anaesthetic risk was higher when a particular anaesthetic technique and/or drug was preferred over another, nor that ME patients were at higher anaesthetic risk than non-diseased patients. Therefore, the anaesthetic management of such patients should not be modified. However, the anaesthetist should carefully consider the patient's history and demands, regardless of whether or not they sound unreasonable, and, in general, create an atmosphere of mutual trust, while administering a "safe technique" by avoiding drugs to which the patient says that he or she has experienced an adverse reaction.

As per Fisher and Rose's sound observations and recommendations, we carefully assessed the extent of the background disease, adverse events history, and results of the clinical examination and investigations in our patient. Unfortunately, we were not able to retrieve historical anaesthetic notes to further help us to plan the anaesthetic management. We discussed different options with both the patient and surgeons, and weighted various risks and benefits. Given the patient's strong preference for regional anaesthesia without sedation, we opted for a PVB only. Since its first description as a suitable alternative to general anaesthesia in women undergoing breast surgery,[9] single-injection PVB alone, or combined with general anaesthesia, has been shown to provide better postoperative analgesia, less postoperative nausea and vomiting and better alertness scores, with little adverse effects, than other analgesic strategies for breast surgery.[9,10] The risk of technique failure, incomplete analgesia and the need to convert to general anaesthesia were discussed in detail.

In conclusion, after carefully weighing the available evidence in connection with anaesthesia-related complications, and the risks and benefits of various anaesthetic techniques, as well as the patient's demands, we were able to provide successful regional anaesthesia for breast cancer surgery, and avoid general anaesthesia and/or sedation in a patient with ME who experienced severe disease relapse after a previous general anaesthetic.

Despite recent progress that has been made in the diagnosis and primary treatment of ME, it is likely that ME patients will continue to pose considerable challenges to anaesthetists owing to the unpredictability of its non-specific symptoms. The current evidence, albeit limited, suggests that there is no correlation between a particular anaesthesia drug or technique and recovery from anaesthesia. Yet, patients will continue to look for reassurance that their perioperative course will be uneventful, and without consequence to their quality of life. Therefore, we suggest that careful preoperative assessment, a sound doctor-patient relationship, and a selective approach to anaesthesia, specific to the patient's condition and type of surgery, will ensure safe perioperative management of this group of patients.

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