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CASE REPORTS

FAINTING ATTACKS ON THE DENTAL CHAIR: "FUNCTIONAL" OR RAS?

H. Nageswaran[1*] and T. Malathie Peiris[2]

Foundation Year 2, Luton and Dunstable Foundation Trust, Lewsey Road, Luton, UK, LU4 ODZ[1]
Consultant Anaesthetist, Luton and Dunstable Foundation Trust, Lewsey Road, Luton, UK, LU4 ODZ[2]
**Corresponding author E-mail:h.nageswaran@googlemail.com*

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FH is a 57-year-old female teacher who presented to us in 2000 for tooth extraction in our dental outpatient clinic. Upon injection of local anaesthetic she became unrousable by voice or pain, recovering after a brief period of time with no residual effect. Similar events took place at subsequent visits, sometimes with full recovery in minutes and at other times taking a few hours. Her attacks became familiar to the staff and she requested the same surgeon and anaesthetist to be present for her appointments. At her last visit, she became unconscious almost immediately following injection of local anaesthetic and was intensely drowsy for 5 minutes. She was given oxygen and taken to recovery where her observations were BP of 151/86, Pulse 67 and O_2 saturation of 97%. Over the next hour, she had at least 3 further episodes of fainting and drowsiness.

Following this, FH was investigated for Lignocaine allergy. She was first given a placebo of 0.3ml normal saline subcutaneously and monitored for thirty minutes. She had no reaction. However, upon the second dose of 0.6ml normal saline injection, she immediately developed itchy eyes, became very cold and shaky with episodes of vacancy and diminished responsiveness. Her mast cell tryptase levels taken at this time were not elevated. It was concluded that she suffered a non-allergic psychosomatic reaction.

Closer investigation of her medical history revealed that her first recorded episode of "unconsciousness" during a dental procedure occurred at the age of 24. Several further episodes were noted between the ages of 36 to 40, on one occasion requiring admission to A+E. Further treatments were carried out in a hospital environment following recommendations by a consultant anaesthetist. Her blackouts were not confined to the dentist's chair. She suffered episodes following strenuous exercise, whilst teaching, at church and once at rest. During the attacks in our hospital, her whole body becomes hypotonic and sometimes required triple manoeuvre to maintain her airway. On waking from these attacks, her speech tends to be slurry. Her symptoms have no definite ictal features and she has never bitten her tongue or been incontinent, but clawing of the right hand and mild twitching of the fingers have been noted in her records. She has been referred to several neurologists regarding these attacks but no central or peripheral neurological abnormalities have ever been found on examination and a CT head, EEG and 24-hour ECGs have all been normal.

Discussion

Reflex anoxic seizures (RAS) describe a condition whereby a triggering event (hence "reflex") causes a vagally mediated severe bradycardia or cardiac arrest resulting in an episode of unconsciousness, often with tonic posturing and/or myoclonic jerks (hence "anoxic

seizures"). It is most common amongst infants and young children aged 6 months to 2 years of age [1], but can occur and even present in adolescents [2]. While it is recognised that the disorder can persist into adult life [3], evidence is not widely found in the literature, perhaps due to the wide variety of terminology used to describe this syndrome [2] and the frequent misdiagnosis as epilepsy when seizures are present [2,4,5]. However, numerous case histories can be found posted on the Syncope Trust and Reflexic Anoxic Seizures (STARS) website, an internet support group aiming to increase awareness of the condition [6].

The most common triggering event is a sudden pain or fright, such as a blow to the head [3], venepuncture [7], febrile illness [8] and excessively hot or cold baths [3]. Despite the definition, many episodes occur without a noticed precipitating factor [3]. Periods of asystole can occur where the subject appears pale and deathly white with stiffening of the body lasting upto 30 seconds [1]. The episode is self-resolving and there is full recovery [9]. The condition is benign and no lasting cerebral ischaemia results [3].

The diagnosis is a clinical one based on an appropriate history, a normal EEG to exclude epilepsy and a normal ECG to exclude cardiac abnormalities such as long QT-syndrome and pre-excitation. Although ocular compression to stimulate the oculo-cardiac reflex (under ECG and EEG monitoring) can aid diagnosis, it is not a requirement and may be distressing to the patient.

The treatment for RAS is mainly reassurance for the patient and family. In cases where episodes are frequent, regular atropine has been used prophylactically [9,10]. Rarely, in severe forms of the condition, cardiac pacing has also been used. [11-13].

With the subject we describe here, there were no generalised seizures witnessed, although clawing of the right hand and twitching of the fingers were present on a few occasions. The majority of her episodes have been in response to an injection at a dental surgery but they have occurred elsewhere and occasionally with no triggering event recalled. There is no record of asystolic periods during her moments of unconsciousness but bradycardia certainly is a feature. It is unfortunate, but not surprising, that she underwent many years of investigations and neurology clinic appointments only to be labelled as suffering from a "functional" disorder. Although FH recalls fainting episodes during her childhood, they were never investigated until her twenties and this may have been a factor in RAS not being considered a possible diagnosis.

FH continues to have dental treatments at the hospital but with an understanding consultant present. With the provision of a diagnosis and reassurance that her condition in benign, she has benefited greatly, suffering fewer and less severe reactions. During an informal talk with one of the consultant anaesthetists at our hospital, she revealed that she had a vague but frightening memory of a dentist restraining her in the chair by pressing his knee against her chest whilst others held her down during a procedure. She was unable to provide further details. It was suggested by the consultant that she seek psychological support to explore this further but FH refused.

We highlight this case for the reason that there may be others suffering with similar symptoms to FH in whom specialist investigations have yielded no diagnosis and whose symptoms have been given a stigmatising label of "functional". While RAS remains a diagnosis of exclusion, it benefits the patients by acknowledging their symptoms as a recognised disorder and relieves the physician of the inclination to make repeated referrals.

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