Background
This case study explores Kate’s experiences in relation to the development, diagnosis and management of food allergy and anaphylaxis at the age of 45. Kate’s name has been changed and no mention of any organisation has been made so as to maintain Kate’s anonymity and her confidentiality. This case study is based in primary care and illustrates the complexities of living with allergy and anaphylaxis and the impact it has on Kate’s family life, work life and social life. Kate’s family history of allergy will be explored as will the onset of her symptoms, assessment, diagnosis and management of anaphylaxis. Kate is married and has no children. She works full-time as a nurse in an older person’s residential unit.

Food allergy occurs when the immune system recognises a food protein as an ‘invader’ and then produces a myriad of symptoms affecting multiple organs in the body. A study in 2003 by Avery et al showed that children with a peanut allergy had a poorer quality of life than children with type 1 diabetes (Avery et al, 2003). It also showed that the biggest concern they had, was fear of dying if they made an error with their food (Avery et al, 2003). There has been an increase in the prevalence of anaphylaxis in the UK by 700% since 1990 (Gupta et al, 2007). A study by Stewart & Evan (1996) found that patients who were referred to A&E had a delay in transfer; only 1/3 of patients were administered adrenaline; were given no management plan; and no recommendation as to further investigations. The study concluded that there was a need for more awareness and management of anaphylaxis (Stewart & Ewan, 1996).
Food-triggered anaphylaxis is unlikely to resolve and more than likely will be long-term.

Personal history
Kate was diagnosed with keratoconus at the age of 25, an auto-immune eye condition present in families with allergy. She also experiences contact dermatitis to washing powder, make-up and shampoos. Her other past history includes appendectomy, multi-nodular goitre, fracture sternum and fractured elbow.

Family history
Kate has 2 brothers and 4 sisters. 5 out of her 6 siblings all have diagnoses of various allergy disorders and atopy in the form of eczema, asthma, allergic rhinitis, and keratoconus. Many of Kate’s nieces and nephews also various allergy diagnoses. Kate’s mother has asthma and is allergic to aspirin and her father has vasculitis and is allergic to penicillin and NSAIDs. Family history is a strong predictor of atopy. Hanson et al (1991) showed that there is a genetic influence for atopic disease with further studies by Jenkins et al (1993) and Bock et al (2001) showing how atopy extends to other organs and its severity.

Figure 1 illustrates Kate’s family history in relation to allergy and anaphylaxis.

Presentation
The initial presentation occurred one hour after eating a salmon bagel when Kate experienced sudden angioedema with closure of her left eye, itching in her left ear, itching in her throat and the back of her mouth and she became wheezy. She describes her chest symptoms as her “lungs filling up with fluid”. Difficulty in breathing and stridor then followed. As Kate is a nurse, she immediately recognised the symptoms of anaphylaxis and took some chlorphenamine (which she obtained from her sister). This eased the breathing difficulties somewhat but it did take 2 hours for her breathing difficulties to fully subside. She continued with normal diet. The next event occurred 3 weeks later when she ate a tuna melt sandwich and within 10 minutes, her “lungs filled with fluid” and she became wheezy and experienced cough. Again, this was relieved by chlorphenamine within 1 hour. This time there was no ocular involvement. The third event took place 2 weeks later following consumption of a tuna sandwich within 10 minutes. Kate then organised a private referral to an immunologist and allergist.
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The incidence and prevalence of anaphylaxis continues to rise and health professionals will need to be skilled in its diagnosis and management.

IgE-mediated food allergy affects multiple systems. In the GI system, oral allergy syndrome (OAS) and gastrointestinal anaphylaxis result. In the cutaneous system, urticaria, angioedema, morbilliform rashes and red flushes can occur. In the respiratory system, acute rhinconjunctivitis and acute asthma occurs. Finally, generalised symptoms such as anaphylaxis and food-dependent exercise-induced anaphylaxis (FEDA) results. FEDA occurs when exercise is undertaken after consuming a food allergen.

Assessment
On review by the immunologist/allergist, Kate had specific IgE tests carried out to various foods and allergens. The following foods were reported as very positive – crab, hazelnut, almond, soya bean, wheat with peanut and rye reported as positive and sesame seed reported as weakly positive. Other allergens which were tested included Timothy grass, cat, dog and mite (farina and pterony) which were reported as very positive. Skin prick testing and prick to prick testing was not carried out. Specific IgE testing is more appropriate than skin prick testing in Kate’s situation as there is an increased risk of anaphylaxis with skin prick testing as Kate has experienced anaphylaxis previously.

A detailed medical history with an allergy focused history was also undertaken as part of Kate’s assessment.

Diagnosis
A diagnosis of anaphylaxis and food allergy was made by the immunologist/allergist based on Kate’s blood tests and history. The immunologist/allergist suggested that the initial event was a scomboid reaction which occurs when fish that is gone off is consumed. This fish is high in histamine and triggers a reaction. Scomboid poisoning is common and is often missed as it resembles food allergy. As Kate’s SpIgE to wheat was also strongly positive, it is difficult to determine what the allergic triggers for Kate are – whether it is wheat or fish or both? She was also told about her other triggers of grass and cat which she was previously aware of as these would have triggered symptoms in the past. Oral allergy syndrome (OAS) is also part of Kate’s diagnosis as she experiences itching in her mouth and throat when she consumes wheat. Foods which cause anaphylaxis include tree nuts, fish, cow’s milk and eggs. Shellfish, some vegetables, some fruits e.g. kiwi can also cause anaphylaxis but are less common. The rise in prevalence in peanut allergy is well documented in comparison to other food allergies (Grundy et al, 2003). A study by Colver et al (2001) showed that fatal reactions to food are rare. This study carried out in Ireland and the UK showed that 229 cases of severe food allergy were identified between 1998 and 2000, 3 were fatal and 6 were near fatal (Colver et al, 2005).

Pharmacological interventions
The initial acute management of anaphylaxis includes the administration of adrenaline, chlorphenamine, hydrocortisone and anti-histamines (Resuscitation Council UK, 2008). At her initial appointment, Kate was prescribed a Jext auto-injector adrenaline pen, levocetirizine 5mgs, prednisolone 5mgs, chlorphenamine and a salbutamol inhaler. Following her second appointment, Kate was prescribed bilastine 20mgs in addition to her initial treatment.

Adrenaline is the mainstay of the treatment of anaphylaxis and should be administered to all patients who experience respiratory difficulties. A delay in treatment has been associated with higher mortality rates. In adults, the dose is adrenaline 1 in 1000 500mcg delivered by subcutaneous injection (Resuscitation Council UK, 2008). This can be repeated every 5 minutes if there is no response. There is minimal risk with adrenaline but caution should be taken in people with cardiovascular disease, uncontrolled BP and arrythmias. After the administration of adrenaline, chlorphenamine should be administered. Chlorphenamine helps protect against histamine-related vasodilatation.

Hydrocortisone is given to reduce the cascade of inflammatory mediators and takes 4 – 6 hours to have effect. Hydrocortisone should be continued for 48 hours because of the risk of a bi-phasic reaction. As Kate experiences respiratory symptoms, Salbutamol is given to reduce bronchoconstriction by acting on the B2 receptors in the airways. Oxygen should also be administered in the acute anaphylaxis situation.

At her second visit to the allergy service, Kate admitted she wasn’t taking her medications as prescribed due to headaches. She was prescribed bilastine 20mgs in the morning. However, this has to be taken 1 hour before food or 2 hours after food and Kate found this difficult to implement into her daily life. She was reviewed again 3-4 months later when the bilastine was discontinued.

The long-term management of anaphylaxis doesn’t include pharmacological management but does include the accurate identification of the allergen(s), patient education and regular follow-up by a specialist service. Patients are advised and educated in the acute management of anaphylaxis and should be given a management plan.

Non-pharmacological management
Since her diagnosis, Kate has eliminated nuts and fish from her diet. She also has eliminated wheat but finds this more difficult as wheat is hidden in many foods. She hasn’t eaten fish and wheat together since her initial reactions but has eaten wheat as it is difficult to avoid. Kate has been referred to a diettian who will assist her dietary management. It is recommended that patients with a documented diagnosis of anaphylaxis, the food trigger should be totally avoided. Food-triggered anaphylaxis is unlikely to resolve and more than likely will be long-term. In Kate’s situation, as her anaphylaxis events have been life-threatening as there was respiratory involvement, this is likely to be the case unless Kate opts for immunotherapy at a later stage. As Kate works 12 hour shifts in the nursing home, she eats the majority of her meals there. She is now considering bringing her meals in to work as it is proving more and more difficult to avoid wheat, fish and nuts.

Cat and dog allergens were also another Kate’s triggers. Kate
has no pets at home. However, pet allergens are also found in homes without pets and in public buildings and on transport as the allergens are transported on people’s clothes (Almqvist et al, 2003). Again, avoidance of these and dust mites are virtually impossible. Kate was given advice on limiting dust mite in the home. Information was also given to Kate about the avoidance of grass pollens and the times of year that these are most likely to be problematic.

Personalised written plans can assist patients in managing their condition. They have been found to reduce the number of reactions and when the reaction does occur, they are less severe as the management plan is followed (Ewan & Clark, 2005, Numatov et al 2008).

Kate should be encouraged to wear a medic-alert bracelet which are available from www.medicalert.org.uk. Kate carries her Jext pen, salbutamol inhaler, prednisolone and chlorphenamine with her at all times.

Education of Kate, her family, her friends and her work colleagues is essential in the management of her anaphylaxis. Kate has been educated and given written instructions on the use of her pen. As Kate is a nurse, she feels competent in identifying and managing her anaphylaxis. Kate has educated her family, friends and her work colleagues on what to do in an anaphylaxis event and all have been shown how to use her pen. Kate has been encouraged to seek medical assistance after an event.

In relation to her social life, Kate describes eating out as a “nightmare” and as a result is limited. Since starting her medication, she has a few episodes which usually happen when she is exercising. This is known as food-dependant exercise-induced anaphylaxis (FEDA).

Anyone who experiences an allergic reaction with respiratory or cardiovascular compromise should be considered for immunotherapy. Currently, immunotherapy is not used for treating individual food allergies. However, research is ongoing and is promising (Clark et al, 2009, Skripak et al 2008). Kate will discuss this her consultant at future consultations.

Conclusion
The incidence and prevalence of anaphylaxis continues to rise and health professionals will need to be skilled in its diagnosis and management. Pumphrey (2004) states that potentially fatal reactions can be prevented by appropriate management after recovery, accurate identification of the cause, effective avoidance of the allergen(s), and effective training in self-treatment with an adrenaline auto-injector. This case study has explored Kate’s story following her diagnosis with anaphylaxis and food allergy. It illustrates the complexities of assessment, diagnosis, management and living with anaphylaxis. It also illustrates the importance of exploring the patient’s family history and the impact that anaphylaxis and allergy can have on daily lives at home, in the workplace and socially.

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