Management of dysphagia

Dysphagia, or swallowing difficulties, is a prevalent disorder associated with certain neurological, obstructive and muscular conditions; stroke is the most recognised neurological cause. Swallowing difficulties are known to adversely affect all aspects of quality of life. Early identification and management is essential. Prognosis is variable and is improved with multidisciplinary management.[3]

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Dysphagia occurs commonly in primary and secondary care but epidemiological studies of it are rare.[1] A recent study reported the lifetime prevalence of swallowing disorders as 38%. Most participants described a sudden onset with chronic problems persisting more than four weeks.[2] Dysphagia adversely affects all aspects of a person's quality of life. We need to promote a multidisciplinary approach to its management.

Classification

There are three broad categories of swallowing difficulties: neurological, obstructive and muscular. Neurological causes include stroke, cerebral palsy, Parkinson's disease, multiple sclerosis and motor neurone disease. Stroke is the most common cause,[4] but we need to consider differential diagnoses.

Obstructive causes include cancer of the mouth and oesophagus, cleft lip and palate, radiotherapy, gastro-oesophageal reflux disease and infections such as herpes simplex virus and less commonly tuberculosis. Muscular/neuromuscular causes include autoimmune aetiologies, commonly scleroderma and polymyositis and more rarely myasthenia gravis. Achalasia, scleroderma and diffuse oesophageal spasm are the most common causes of neuromuscular motility disorders.[5] We present a case of motor neurone disease (Box 1) and myasthenia gravis (Box 2) to highlight presentation characteristics secondary to a neurological and neuromuscular aetiology, respectively.

Presentation

Presenting symptoms vary but patients usually report coughing, choking or the abnormal sensation of food sticking in the back of the throat or upper chest when they initiate a swallow. Neurological causes of dysphagia are more likely to present with the former symptoms and obstructive dysphagia with the latter.

Other symptoms associated with all three types of dysphagia include difficulty or painful chewing or swallowing, dry mouth (xerostomia), soft voice, unexplained weight loss, regurgitation of food and recurrent chest infections.[6]

The acuteness or otherwise of the symptoms can help make a differential diagnosis. For example, neuromuscular dysphagia gradually progresses whereas mechanical obstruction progresses rapidly.

It is important to review medication to minimise adverse effects; for example, nonsteroidal anti-inflammatory drugs may be contributing to the problem as they can cause direct mucosal injury to the oesophagus. Alcohol and smoking consumption should also be noted as important risk factors for malignancy and obstructive lesions.

Investigations

The focus of physical examination may be based on the patient's history. Neurological examination may reveal signs suggestive of a new stroke, specific cranial nerve palsies and fatiguability associated

with myasthenia gravis, or mixed upper and lower motor neurone signs with prominent fasciculations for motor neurone disease. A cachexic state may also be noted; this is a worrying sign as it can reflect either obstructive dysphagia secondary to malignancy or a chronic presentation of neurological dysphagia. Organomegaly, particularly hepatomegaly, may indicate liver metastases and in the context of dysphagia may well originate from a gastrointestinal source. A bedside swallow assessment is crucial as part of the initial investigation. This requires a patient to drink a small volume of water whilst sitting upright, following which checks are made for delayed swallow, the presence of drooling, coughing and dysphonia.

Simple blood tests, possibly from bleeding ulcers or tumours, can help identify anaemia and can also detect autoimmune cases. However, specific dysphagia studies are usually required. Direct laryngoscopy is useful in evaluating oropharyngeal dysphagias. Barium studies are usually the first-line investigation for identifying obstructive lesions; however, endoscopy provides the optimum assessment of the oesophagus in suspected obstructive cases. Manometry, useful in reflux disease, uses electronic pressure probes to measure motor function and responses to swallowing, and pH monitoring, via a nasogastric tube, records pH levels within the oesophagus. EMG (electromyography) assesses muscular responses to electrical stimulation.

A decremental response indicating muscle fatiguability is characteristic of myasthenia gravis. Reduced muscle action potential and reduced nerve conduction velocity in the presence of spontaneous muscle fasciculation is consistent with anterior horn cell disease. Video-fluoroscopy has a role in assessing neurological cases of dysphagia and specialist input from a consultant neurologist may further elucidate the cause.

Box 1: A case of dysphagia of neurological aetiology

An 89-year-old Asian woman was referred to a secondary care medical clinic with a five-month history of difficult and painful swallowing and significant weight loss. Her past medical history included tuberculosis, asthma and gastritis. She was taking ranitidine and using a salbutamol inhaler. She lived with her extended family, had never smoked and did not drink alcohol. Clinically she was emaciated, frail and had hepatomegaly. Tumour markers, chest and abdominal imaging, barium swallow and the opinion of ear, nose and throat specialists were all negative for malignancy and tuberculosis. One month later she had developed choking and breathlessness. She failed a bedside swallow test, had both upper limb and tongue fasciculation and brisk reflexes. Motor neurone disease was therefore suspected. An electromyogram showed reduced muscle action potentials, reduced nerve conduction velocities and spontaneous muscle fasciculation, while sensory nerve conduction was normal. These features were consistent with anterior horn cell disease. The patient was referred to a local tertiary motor neurone disease service. Unfortunately, she rapidly developed type 2 respiratory failure, markedly deteriorated and died.

Management

The management of neurological dysphagia requires a multidisciplinary approach. Here, we focus on stroke. Much of this relates to, and overlaps with, the management of other neurological/muscular dysphagia cases.

Stroke

The speech and language therapist (SALT) is a key member of the stroke multidisciplinary team. In accordance with national stroke guidelines, the SALT will assess a swallow within 24 hours of admission, and advise on food modifications and language skills. In our hospital, we have a nutrition team with specialist nurses who daily assess patients with suspected poor swallow and initiate naso–gastric (NG) feeding, or, if the swallow is unlikely to return, liaise with the gastroenterology team regarding more permanent feeding such as percutaneous endoscopic gastrostomy (PEG). Many senior nurses are being trained to screen for swallowing difficulties.[7]

Unsafe swallow

An unsafe swallow may or may not improve. The SALT may give guidance to enable patients to "relearn" how to swallow. Dietary changes may be necessary and include softer foods and thickening of fluids, or feeding via an NG or PEG tube. NG tubes last 10–28 days. PEG tubes are designed to last for six months.

There are complications associated with PEG placement. These include tube displacement, skin infection, tube blockages

and leakages, and less common complications, such as serious internal infection and bleeding.

Medication management

It is important to think carefully about medication management. Pharmacists may help with consideration of liquid, or alternative routes of administration. An occupational therapist may advise on feeding implements and adapted cutlery, and a dietician may advise on nutritional intake. Nursing staff have an important role in the physical action of taking in food. The National Institute for Health and Clinical Excellence (NICE) policy on nutrition support in adults is particularly useful.[8]

Other neurological causes

In management of non-stroke neurological causes, specific medications may help. Botulinum toxin can treat muscular dysfunction, such as achalasia. For obstructive causes, surgical procedures, or dilatation and stenting may help.

Prognosis

The prognosis for recovery from dysphagia varies from excellent to poor depending on its severity, aetiology and compliance with treatment.

Dysphagia affects up to 80% of stroke patients.[9] However, previous research has suggested that 37% suffer for less than eight days, and up to 86% may be able to swallow normally within 14 days.[10]

Primary prevention is where we as clinicians must focus our efforts. We need to educate our patients regarding risk factors for cancers, such as smoking and excess alcohol, and we need to promote healthy lifestyles to reduce risk factors for cerebrovascular disease.

I have no conflict of interest

Box 2: A case of dysphagia of neuromuscular aetiology

An 80-year-old woman presented to the acute medical ward with a three-day history of difficulty swallowing and inability to tolerate food or fluid, which she felt was sticking in her throat. She was also experiencing problems being understood and felt her speech was softer. She described increased sputum, and frequent coughing and choking fits over the past year. Her past medical history included hypertension and chronic renal impairment. She took diltiazem, irbesartan and omeprazole. She was widowed, had never smoked and did not drink alcohol. She regularly played golf up to a few days prior to admission. Clinically she had expressive dysarthria and an unsafe bedside swallow with pooling of secretions. She had no long tract signs. The differential diagnosis underlying her dysphagia was stroke, or another structural or neuromuscular cause. ENT investigation showed weakness of the left palate and secretions of the pyriform fossa. The impression was of a stroke affecting the 9th and 10th cranial nerves. However, her head CT scan showed no acute infarction, haemorrhage or space-occupying lesion. She clinically deteriorated and developed progressive bulbar and facial weakness with incomprehensible speech. An electromyogram was performed. Repetitive nerve stimulation showed significant decremental responses supportive of a diagnosis of myasthenia gravis. She was commenced on pyridostigmine and prednisolone, and transferred to the local tertiary neurology centre. She was later admitted to the intensive care unit, underwent a tracheostomy and received a course of immunoglobulins. She later stabilised and was transferred back to ward-level care.

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