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The impact of misdiagnosing Bell's palsy as acute stroke

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ABSTRACT

Idiopathic Bell's palsy can lead to a serious and, sometimes permanently, disfiguring and emotionally challenging facial palsy. Early diagnosis and treatment with corticosteroids are important, as they significantly improve recovery rates. Bell's palsy is a benign condition that should be diagnosed and managed in primary care. Patients who self-present to the emergency department should be managed and discharged without needing admission. We reviewed all patients referred urgently to our hospital with facial weakness and discharged with a diagnosis of Bell's palsy, to explore whether clinicians were confident in making this diagnosis at initial assessment and, if not, how often they sought a specialist opinion. Furthermore, we assessed the impact of its over-investigation and mistreatment on healthcare resources and the patients.

KEYWORDS: Acute stroke, Bell's palsy, facial nerve, over-investigation and resources

Introduction

Acute cranial nerve VII (CN VII) palsy has an annual incidence of around 38 per 100,000 people in the UK, usually aged between 30–50 years, and is the most common nerve paralysis in humans.[1,2] Bell's palsy accounts for 60–70% of all unilateral facial nerve palsies.[3] Many patients suffer a transient but disfiguring and emotionally challenging facial palsy. However, some can also be left with permanent aesthetic, functional and emotional deficits as a result. Therefore, its early diagnosis and treatment with corticosteroids is important, as it has shown to significantly improve recovery rates, as well as referral to ear, nose and throat (ENT) specialists where appropriate.[4]

The diagnosis of Bell's palsy can be challenging, especially as there are many differential diagnoses including trauma, neoplasm and infection. This is due to the facial nerve's both complex and convoluted journey from its origin in the pons, through the cranium, until it branches superficially to supply motor function to the 18 muscles of facial expression.[5]

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For many clinicians, acute stroke remains a concerning diagnosis in patients presenting with facial palsy, but there are key characteristics which facilitate differentiation of the two conditions, often without the need for further investigations. Our study aimed to explore whether clinicians could diagnose Bell's palsy in patients presenting with facial palsy at initial assessment and, if not, how often they sought a specialist opinion, as well as to assess the impact of over-investigation and mistreatment of this condition on healthcare resources.

Methods

We carried out a retrospective observational study in a large teaching hospital in Cambridge, UK, over 3 years (October 2014–17). We analysed electronic medical records of all patients discharged with a final diagnosis coded 'Bell's palsy'. Direct admissions to the emergency department (ED) from both primary care and self-referrals were included. Exclusion criteria were those <18 years old, having an existing Bell's palsy diagnosis or being admitted electively for aesthetic surgery. None of the patients included had had any recent head injury or trauma.

We collected admission data including patient age, sex and referral source. For all patients, we documented their clinical course, including the team (medical or ED) and grade of doctor (junior doctor taken as below the level of registrar) that initially clerked them, as well as any escalation through team members and referral to either stroke or neurology. The number of imaging modalities undertaken, as well as treatments given, along with length of hospital stay in days was used to estimate the impact on healthcare resources.

This project was carried out as part of a service evaluation project within the stroke department and it was confirmed that formal ethical approval was not required.

Results

Over the 3-year period, 118 people were discharged with a diagnosis of Bell's palsy. After exclusion criteria, we gathered data on 46 patients with a mean age of 59.0±18.6 years. Table 1 summarises basic demographics, information regarding the admission and inpatient stay.

No patient was discharged directly from ED within the 4-hour target and most patients underwent a complicated pathway through the hospital. The average length of stay (LOS) was 1.75 days; (this number was based on 43 patients as three had a lengthened hospital stay due to complications and issues unrelated to Bell's palsy, including discharge planning). Each admitted patient was assessed by between 2–3 clinicians. Out of all the patients who were eventually given a diagnosis of Bell's

Table 1. Basic demographics of patients discharged with diagnosis of Bell's palsy	
Total screened, n	118
Excluded, n	72
Included, n	46
Age, mean (SD)	59 (18.6)
Male, %	48
Female, %	52
Primary care referrals, n (%)	13 (28.3)
Length of stay = 1 day, n	22
Length of stay = 2 days, n	9
Length of stay = 3 days, n	11
Length of stay = 4 days, n	1
Average Length of stay, days	1.75
Clinicians reviewing each patient, mean	2.85

palsy, 28 (60.8%) patients had been referred for specialist review by stroke or neurology teams (Fig 1).

Only 6 patients were diagnosed with Bell's palsy without any neuroimaging. The remaining 40 patients (86.9%) underwent a variety of neuroimaging which included a mixture of computed tomography (CT) of the head, magnetic resonance imaging (MRI) of the head, CT angiography (CTA) or magnetic resonance angiography (MRA) or carotid Doppler ultrasound. A total of 57 scans were performed in these patients, with the most common modality being a non-contrast CT of the head (63.2%). Only two patients underwent CT of the head due to the presence of pre-existing intracranial disease (Fig 2). Out of the 57 scans performed, 54 were reported as normal with the other three scans reporting

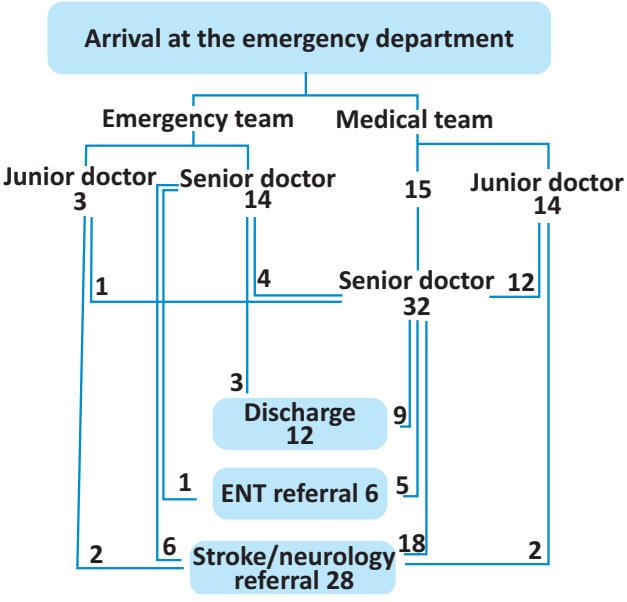

Fig 1. The convoluted pathway of assessments and referrals for patients diagnosed with Bell's palsy.

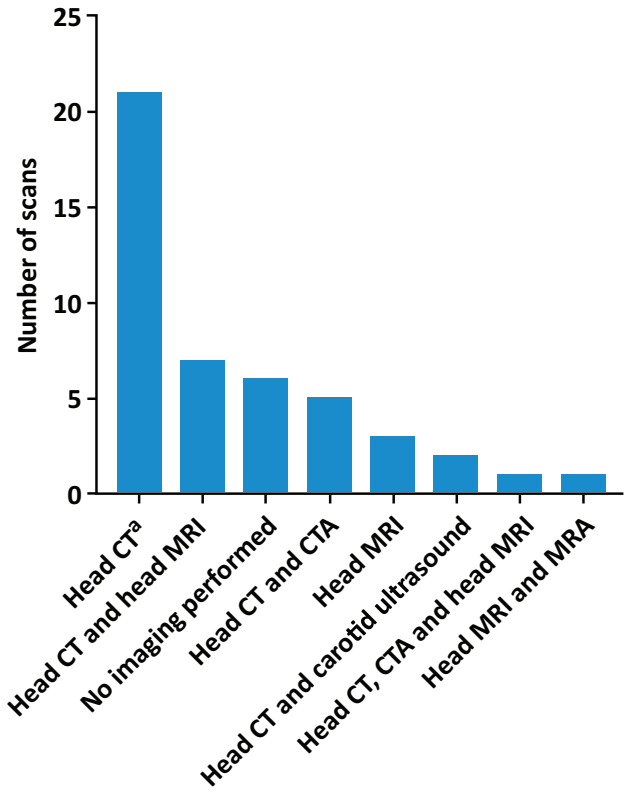

Fig 2. Number of different imaging modalities undertaken in 46 patients with a final diagnosis of Bell's palsy. [a] = two scans performed due to pre-existing disease, one ventriculoperitoneal shunt and one known meningioma; CT = computed tomography; CTA = computed tomography angiography; MRA = magnetic resonance angiography; MRI = magnetic resonance imaging.

unrelated information not influencing the final diagnosis (in one patient, CT of the head showed an old asymptomatic lacunar stroke, and carotid Doppler showed mild asymptomatic carotid disease in two other patients).

Even though all the clinicians noted facial weakness during their initial clerking, only 40% of them documented other important non-stroke-like symptoms such as pain and only three patients overall were asked about hyperacusis. None of the assessments documented asking about changes in taste or lacrimation which are key attributes of a facial nerve affected peripherally. Only 6/17 (35.3%) ED doctors and 28/46 (60.9%) medical doctors asked about any progressive nature of the patient's facial palsy symptoms in their initial review, a key differentiating point from acute stroke which must be discerned in every case. The average duration of symptoms at admission for patients in our cohort was nearly 3 days (ranging from 3 hours to 21 days). Even in the 13 primary care referrals, only two general practitioners (GPs) had confidently stated this is Bell's palsy (but still referred to secondary care without starting corticosteroids), with eight querying acute stroke despite a clear history of progressive symptoms elicited later at stroke or neurology review.

From initial clerking, only nine patients received steroids before investigations were complete or further specialist review sought. 31/46 patients received no treatment after initial clerking, with all

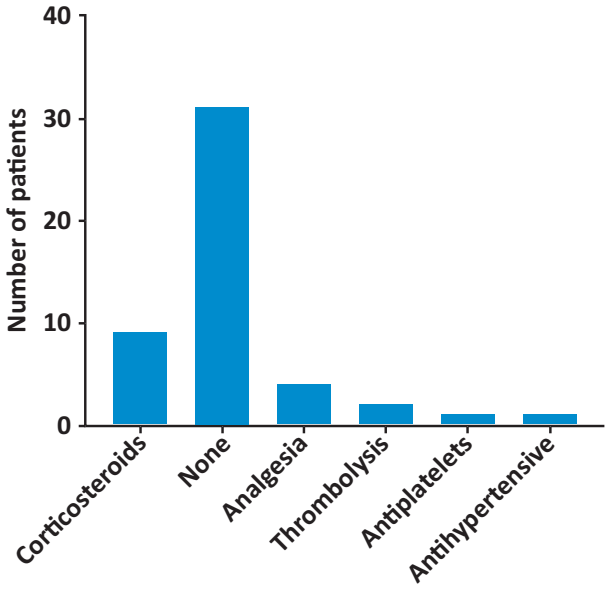

Fig 3. Initial treatment given to patients who were diagnosed with Bell's palsy prior to organisation of specialist review or further imaging.

being referred for further imaging, medical or stroke/neurology specialist opinion prior to treatment. Two patients received intravenous thrombolysis for possible stroke. Ten inappropriate follow-up appointments were arranged, six patients were to be followed up in neurology clinic and four in neurovascular (transient ischemic attack (TIA)) clinic (Fig 3).

Discussion

Bell's palsy can sometimes be a difficult diagnosis to make, especially with acute stroke being high on the list of concerning differential diagnoses in the mind of many clinicians. However, an understanding of both the origin and route of CN VII through the nervous system and its adjacent anatomical structures and a detailed history helps narrow down the differentials and pinpoint a diagnosis (Boxes 1 and 2, Table 2).

Our study shows that cases presenting to ED who are eventually diagnosed with Bell's palsy undergo reviews by multiple clinicians and different modalities of neuroimaging, with most not receiving the necessary corticosteroid treatment after initial review. Out of the 46 patients in our study, 28 patients (60.8%) were referred to stroke or neurology services for specialist opinion. We believe that this is due to a lack of eliciting key aspects of the medical history that aids in differentiating Bell's palsy from acute stroke. Subsequently, this leads to multiple reviews, delays in appropriate treatment and organisation of unrequired neuroimaging. Bell's palsy is mostly progressive over days, and only 35% of ED doctors and 61% medical doctors asked about this characteristic, which is a crucial differentiating feature from acute stroke. A detailed history in a Bell's palsy case should elicit discomfort or sensory symptoms in the distribution of the CN VII in the hours or days preceding facial palsy, and it is very important to uncover whether the symptoms were progressive in nature. The presence of other symptoms more commonly encountered in Bell's palsy that would not be attributable to stroke, such as hyperacusis, localised pain and changes in taste or lacrimation must also be enquired about. Interestingly, despite the average duration of symptoms being nearly 3 days; stroke or query stroke was the primary initial diagnosis in 54% patients, which were all then changed to Bell's palsy after neuroimaging or specialist review.

Not correctly diagnosing Bell's palsy can have several negative impacts. Firstly, and most importantly, the negative psychological impact on the patient can be considerable. Developing a facial palsy is known to cause psychological distress, and being misdiagnosed as stroke, admission to hospital including to the hyperacute stroke units, and increased burden of unnecessary imaging and treatment can only compound the anxiety felt by patients and their families.[6] This is more important if we consider that majority of these patients are young. On occasion, considerable reassurance is needed to alleviate the stress of being misdiagnosed as a stroke at the front door and some patients may seek second opinions despite normal imaging.

Secondly, delaying the diagnosis by requesting neuroimaging and specialist review led to delays in initiating appropriate treatment that can increase the likelihood of complete recovery, ie corticosteroids. 40/46 patients underwent neuroimaging with no useful extra diagnostic information obtained from these scans and only 9/46 patients received steroids after initial review before any imaging. In addition, unnecessary investigations and treatment exposes the patient to unwanted side effects such as radiation, contrast exposure and drug side effects.

Thirdly, we must consider the avoidable use of hospital resources and clinician time. Bell's palsy is a benign condition that should

Box 1. Neuroanatomy of cranial nerve VII

Understanding the neuroanatomy of cranial nerve VII (CN VII) is key in differentiating upper motor neuron from lower motor neuron facial palsy, as it provides a road map of innervation from its origin, which will help localise the lesion during clinical examination. The facial nuclei originate in the pons and receive corticobulbar motor fibres from the primary motor cortex, travelling through the internal capsule.[5,7] Disruption of these supranuclear pathways, above the level of the facial nucleus, cause a centrally originating contralateral facial palsy of which acute stroke is the most common aetiology.

Intracranially, the motor and sensory roots of CN VII arise in the pons and travel through the internal acoustic meatus along an opening in the petrous temporal bone. They then enter the facial canal where the roots join together forming the facial nerve, which then exits the cranium at the stylomastoid foramen.[7] Intracranial damage to the facial nerve roots prior to their exit, from traumatic damage to the temporal bone, neoplastic disease of the middle ear or mastoid, or infective and inflammatory causes, often leads to compression of the CN VII nerve roots causing loss of lacrimation, taste, salivation and stapedial reflex, along with facial palsy.

The facial nerve then branches into the parotid gland where it splits into its temporal, zygomatic, buccal, marginal mandibular and cervical branches, which are responsible for innervating the muscles of facial expression.[5,8] Compression or damage here often by trauma or neoplasm within the parotid gland or mandible, leads to isolated lower motor neuron facial palsy.

Box 2. Differentiating Bell's palsy from acute stroke

Initial assessment of any patient must involve establishing the onset characteristics and duration of the facial palsy. Here, the timing and progression of Bell's palsy helps distinguish it from an acute cause such as stroke. A key feature is the progressive nature of Bell's palsy, which can be elucidated by detailed history taking.[9] The history must also check for recent trauma, surgery or infection.

Table 2 summarises the common differences between Bell's palsy and acute stroke.

Determining whether the facial nerve paralysis is central or peripheral is therefore key to diagnosis. Central lesions will cause paralysis of the lower face alone, sparing the forehead; however, clinicians must ensure they ask about the duration and nature of symptoms including the presence of associated symptoms such as hyperacusis, posterior auricular pain, taste and lacrimal changes in their history.[10,11] A full cranial nerve examination as well as ocular, otologic and oral examinations must be carried out in all patients presenting with a facial palsy.

Neuroimaging should really be reserved for those patients with other associated physical findings suggestive of a central lesion (such as paralysis of other cranial nerves, associated limb weakness or ataxia) or those whose symptoms have not resolved despite appropriate treatment. Certainly, in cases where there is a history of trauma, computed tomography will adequately demonstrate any disruptions to the temporal bone and structures surrounding cranial nerve VII.

Treatment of Bell's palsy in the acute stage includes early initiation of steroids and eye protection on the effected side to avoid exposure keratitis (in some cases of complete inability to close the eye, urgent ophthalmology assessment is required).[12] Referral to ear, nose and throat specialists can be considered for those who do not respond to or have persistent symptoms despite treatment.[10]

Table 2. Typical presentation of Bell's palsy compared to acute stroke

	Bell's palsy	Acute stroke
Age, years	30–50	Usually >60
Symptom time course	Progressive; over hours or days	Sudden; over seconds
Unilateral facial paralysis	Yes	Yes
Upper face	Always affected	Usually not affected
Lower face	Always affected	Affected
Ability to close eye on symptomatic side	Not likely	Likely
Ear or TMJ area pain	Likely	Not likely
Hyperacusis	Likely	Not likely
Decreased lacrimation, salivation or change in taste	Likely	Not likely
Pupils affected	Not likely	Sometimes
Arm or leg weakness	Not likely	Likely
Speech or vision affected	Not likely	Likely

TMJ = temporomandibular joint.

be diagnosed and managed in primary care, and patients who self-present to the ED, should be managed and discharged by the ED without the need for admission. Our hospital received 13 referrals from GPs, two despite diagnosis of Bell's palsy in the referral letter. The average LOS of our patients was nearly 2 hospital bed-days, including review by between two to three different clinical teams. During this time, 40 patients underwent neuroimaging. Furthermore, 10 patients were referred to TIA or neurology clinic for further outpatient assessment, despite a Bell's palsy diagnosis being made and corticosteroid treatment being initiated prior to discharge.

While it is difficult to directly attribute or measure overuse of resources in this context, if our cohort of patients had been diagnosed and managed by the GP or had been discharged from the ED with appropriate treatment, rather than being admitted and over-investigated, the health economy would have saved over at least £42,600 (this figure includes £32,200 on bed days, £5,640 on the total cost of imaging, approximately £1,800 on unjustified treatments, and £3,000 on unnecessary follow-up appointments). This is not accounting for the time multiple clinical and specialist teams spent reviewing a Bell's palsy patient.

Clearly, the diagnosis is not clear each time and there are scenarios which warrant exploration of a broader differential diagnosis. For example, in our cohort, two patients were referred to the stroke team and actually underwent thrombolysis for a suspected stroke. However, in both cases, the facial symptoms were accompanied by unrelated limb weaknesses or pain which were not properly discerned through careful history taking. The problem is in all the other cases where acute stroke is being explored, the true clinical condition remains untreated and the patient justifiably worried.

Conclusion

Our study suggests that there is a lack of confidence in diagnosing Bell's palsy in both primary and secondary care. With acute stroke being high on the list of differential diagnoses, it is clear that many clinicians want to exclude this first, as they do not want to misdiagnose stroke as Bell's palsy. What is therefore paramount is education of all doctors at all grades on basic neuroanatomy as well as the clinical presentation of Bell's palsy compared to stroke which will assist them when assessing a patient with facial palsy.

Our results show that when the clinical course of Bell's palsy is not discerned through careful history taking and examination, clinicians often rely on specialist neurology or stroke services for further assessment. There is also an over-reliance on neuroimaging, which should be reserved for ruling out trauma, or when centrally caused symptoms are present. Misdiagnosis of Bell's palsy as a stroke leads to incorrect treatment or a delay in initiation of appropriate treatment, unnecessary admissions,

inappropriate use of healthcare resources, and most importantly, negative psychological impact on patients and their families. ■

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