LETTERS TO THE EDITOR

To the Editor

Screening for Adhesive Capsulitis in the Timely Diagnosis of Parkinson's Disease

Frozen shoulder, or adhesive capsulitis, is a condition characterized by pain, stiffness, and a limited range of motion of the glenohumeral joint.[1] The presence of frozen shoulder is common among elderly patients and is often under-recognized as a preceding symptom of Parkinson's disease (PD).[2] Since adhesive capsulitis usually manifests before the classic parkinsonian symptoms, it often creates a diagnostic predicament for family physicians.[2] Previous studies have suggested a strong correlation between the incidence of frozen shoulder and the classic parkisonian symptoms that include, but are not limited to bradykinesia, resting tremor, cogwheel rigidity, and high scores on the Unified Parkinson's Disease Rating Scale (UPDRS).[1] Currently, there are only limited studies that examine the screening and diagnostic trends in elderly PD patients that present with adhesive capsulitis prior to their diagnosis of PD. This oversight of frozen shoulder as an early sign of PD may lead to unnecessary diagnostic procedures, while also delaying the treatment of PD symptoms contributing to a poor quality of life.

Methods

As a part of routine history taking, patients presenting with possible PD initially were asked questions regarding the symptoms of frozen shoulder between the years of 2005 and 2011. This practice continued during subsequent follow-up visits. Patients with suspected frozen shoulder were further questioned about the presence and onset of shoulder pain, the time of presentation of classic parkinsonian symptoms, and the date of first family physician visit concerning the shoulder pain. Patients were also asked to report the overlap period between the classic parkinsonian signs and the discomfort associated with the frozen shoulder during follow-up visits. Additional information on whether clinical presentation of the frozen shoulder was ipsilateral or contralateral to the parkinsonian symptoms was noted. Furthermore, the effectiveness of anti-parkinsonian medications in treating the frozen shoulder was also evaluated.
All patients were diagnosed with stage II of PD according to the modified Hoehn and Yahr (HY) classification system, which consists of stage I to V ranging from least to most severe form of the disease.[3] The population of interviewees consisted of 63% males and 37% females. The mean age of patients selected for the interview was 64.5 years old, with the youngest patient being 37 years-of-age, and the eldest patient being 78 years old. The symptoms of shoulder pain were distributed to left and right shoulder at 40% and 60%, respectively, and no injuries were reported which could have caused a frozen shoulder.

While gathering information, patients were also asked about any investigational or diagnostic procedures, specialist care, or alternative treatment modalities used in the treatment or diagnosis of the frozen shoulder. Investigational procedures that patients had to undergo prior to the diagnosis of PD included EMG, X-ray, Ultrasounds, and MRIs. Specialist referrals were comprised of Orthopedic Surgeons, Physiatrists, and Rheumatologists. Additionally, alternative treatments included physiotherapy, surgery, steroid injections, pain medications and the use of chiropractors. A chart review was conducted to corroborate the information gathered during the patient assessment.

Results

A total of 19 patients out of 320 patients with PD were found to have a history consistent of frozen shoulder prior to their diagnosis of PD. On average, there was a one-month delay before patients sought help from family physicians with regards to the frozen shoulder, and approximately six months between the onset of frozen shoulder and PD. As depicted in Table 1, most patients who had frozen shoulder experienced the symptomology for 6-24 months before being referred to a neurologist, while four patients reported having the symptoms for a period greater than two years.

The majority (63%) of patients who had frozen shoulder reported that symptoms started before the onset of PD with an average overlap of symptoms from anywhere between one to six months. Most patients (95%) with adhesive capsulitis also manifested the initial parkinsonian symptoms on the ipsilateral side of the frozen shoulder. Additionally, all 19 patients reported an improvement in their conditions when administered PD medications. The symptoms of frozen shoulder lead to the early diagnosis of PD in one patient, while the remaining 18 patients underwent various diagnostic tests. Of these 18 patients, 32% of patients had to undergo at least two investigational procedures before eventual diagnosis of PD was made. The most commonly reported diagnostic imaging procedure was X-ray followed by Ultrasonography. Half the patients in this group were referred to other specialists before being referred to a neurologist. When asked about alternative treatments, nine patients reported having been treated with at least one other treatment modality. Physiotherapy was the most common alternative treatment used (68%) at the time. Alternatively, ten patients also underwent invasive procedures such as steroid injections (21%) and surgery (5%) in attempts to alleviate the symptoms of frozen shoulder.

Discussion

Early symptoms of PD can be very subtle and sometimes hard to discern.[4] Patients often have milder symptoms of PD including symptoms of frozen shoulder for years before appropriate medical attention is sought. In this study, frozen shoulder was present as a preceding symptom in 6% of the cohort of PD patients that were assessed. Previous studies demonstrate that symptoms of adhesive capsulitis could precede classic Parkinson's symptoms by 2 to 15 years, and failing to recognize frozen shoulder as an early symptom of PD can cause diagnostic confusion and delay in treatment.[2] Consistent with previous reports, the results of our study also demonstrate that the majority of patients eventually developed the classic parkinsonian symptoms on the same side that exhibited the frozen shoulder.[1] The significance of this finding adds evidence that there is a strong association between frozen shoulder and PD. Despite this association, only one patient in the study was

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Table: Parkinson's disease (PD) patients with frozen shoulder

Age	Max = 78	Min= 37
	Number of cases (n=320)	Percentage
Total Frozen Shoulder Cases	19	6%
Females	7	36%
Males	12	63%
PD eventually initiating on the same side as Frozen Shoulder		
	18	95%
Onset of Frozen Shoulder relative to diagnosis of PD		
Before	12	63%
Same time	4	21%
After	3	16%
Duration of Frozen Shoulder		
<6 months	1	5%
6-12 Months	6	32%
12-24 Months	6	32%
>24 Months	4	21%
Duration of Frozen Shoulder overlap with PD symptomology		
1-6 Months	8	42%
7-12 Months	3	16%
13-18 Months	2	11%
> 18 Months	3	16%
Number of procedures undergone by patient prior to eventual diagnosis of PD		
0	3	16%
1	4	21%
2	6	32%
3	4	21%
>3	1	5%
Number of Specialists Seen		
0	5	26%
1	9	47%
2	4	21%
>2	0	0
Response of Frozen Shoulder to PD medication		
Positive Response	19	100%
Negative Response	0	0%

timely diagnosed with PD based on the clinical presentation of adhesive capsulitis.

A majority of the patients, however, were subjected to various diagnostic or therapeutic procedures between the period of their first visit to the physician complaining of shoulder pain and the eventual diagnosis of PD. For instance, patients were exposed to unnecessary and potentially harmful X-ray radiation in attempts to find the underlying etiology of the frozen shoulder. Other patients underwent invasive orthopedic procedures such as arthroplasties and steroid injections, for which the efficacy is not very well established.[5] It was not until the patient presented with the classical symptoms of PD such as rigidity, overt tremor, and bradykinesia, that physicians were able to appropriately refer these patients to a neurologist.[4]

It has been previously reported that the pain in adhesive capsulitis could be effectively treated and alleviated with L-dopa/carbidopa.[5] Consistent with this, all patients who reported having symptoms of frozen shoulder showed improvement after administering PD medications. Therefore, using frozen shoulder as an early screening tool, physicians can avoid the cost and risks of alternative procedures, while concurrently improving the patient's quality of life. This is particularly essential for proper diagnosis since most patients whose frozen shoulder was caused by underlying Parkinsonism also exhibited early symptoms of PD. These early symptoms include the absence of arm swing, lack of dexterity, motor slowness, drooling, and hyposmia that often go unrecognized in clinical practice.[3]

Due to the time lag present between the initial onset of frozen shoulder and the first assessment by a neurologist, recall bias maybe a limitation of the study. Efforts were made to minimize recall bias by a retrospective chart review. In addition, the absence of a control group may also further limit the internal validity of this study. However, it is important to note that the prevalence of frozen shoulder in a healthy population is scarce, and thus makes it difficult to attain an adequate control group for comparison.[2]

Though the identification of subtle PD symptoms at an early stage sometimes proves difficult and variable among patients,

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increasing the awareness of physicians and patients of the early signs of PD can improve the current under-recognition and misdiagnosis of PD.[5] Since patients are often not aware that adhesive capsulitis can be an early presentation of PD, they will not voluntarily discuss it with their physician and thus, delay the appropriate treatment of their condition. As such, physicians should take the initiative to screen for early PD symptoms when no other causes of frozen shoulder can be elucidated.

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To the Editor

Parinaud's Syndrome Due to Migraine

A 28-year-old Caucasian patient has been treated for two years by his general practitioner (GP) for history of migraine. He has throbbing headache associated with nausea, vomiting, photophobia and phonphobia. He presented with new onset history of two months duration of recurrent, severe and unilateral headache associated with double vision and similar migraine elements he had in the past. When he was examined in the Eye department, he was found to have a head tilt to left to compensate for double vision. His visual acuity was recorded at 6/5 bilaterally, the fundi were normal and there was no ptosis. He has sluggish pupillary reaction to light and accommodation examination provoked convergence nystagmus. He has impaired upward eyes movement. The Hess chart suggested weakness of the left superior rectus and right medial rectus. He was eventually referred to our neurology department for the problem of diplopia. His neurological examination confirmed eye signs and a diagnosis of Parinaud's syndrome were made. His routine vasculitis, autoimmune screen and lactic acid blood tests were all normal apart from a slightly high triglyceride level of 1.8 mmol/L.A. His brain imaging including CT and MRI scans were normal. In the light of clinical presentation and normal investigations which excluded other causes, in our opinion, the most likely cause of his Parinaud's syndrome was migraine.

Discussion

Parinaud's syndrome is diagnosed when a lesion in the superior tectal area causes a combination of supra-nuclear upgaze palsy, retraction convergence nystagmus and light- near dissociation. Recognised causes of Parinaud's syndrome include tumour, haemorrhage, infarction, vascular malformation, demyelination and viral infection. In the absence of all other explanations migraine was thought to be most reasonable cause. Permanent ocular paralysis is very rare but it was described in some patients who experienced recurrent episodes of diplopia and headache. The disorder described could be classified under the rubric ophthalmoplegic migraine. The mechanism of ophthalmologic migraine is not clear. It has been suggested that oedema in the wall of the internal carotid artery results in compression of the oculomotor and trigeminal nerves. This theory has not been supported by angiogram findings.[1] In a recent case report, enhancement and thickening of the cisternal portion of the oculomotor nerve associated with ophthalmoplegic migraine has been described.[2] Neither theory explains occasional development abducens and trochlear palsy. Currently ophthalmologic migraine is considered to be a type of recurrent demyelinating cranial neuropathy.[3]

Conclusion

Migraine is associated with transient neurological deficit such as parathesia, hemiplegia and oculomotor palsy. In rare cases, these deficits become permanent and require further investigations, as in our patient, who developed permanent Parinaud's syndrome.[4,5]

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