

A Study of Post-stroke Pain in a Tertiary Care Hospital in Eastern India

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ABSTRACT

Pain is a common symptom of stroke. The exact prevalence is not known due to lack of studies. Acute and chronic pain can occur after stroke. In this study, the prevalence of different types of chronic post-stroke pain and their characteristics are evaluated. Identifying the types of pain that can occur after stroke can help to prevent and manage those cases. A retrospective and prospective study were undertaken among 1060 patients of stroke. In this series, temporomandibular joint dysfunction (TMJD) 14%, neck pain 40% hemiplegic shoulder pain (HSP) 41.9%, brachial plexus injury 1.03%, heterotopic ossification (HO) of elbow 0.6%. Compressive neuropathy due to poor positioning like median nerve entrapment neuropathy 41.2%, ulnar nerve entrapment neuropathy 30.6%, dependent edema of hand and forearm 27.9%, complex regional pain syndrome type I (CRPS type 1) 12.3%, pressure ulcer (PU) 31.03%, deep vein thrombosis (DVT) 2.3%, central post-stroke pain syndrome (CPSP) 11.03%, and spasticity 21.03%.

Keywords: Chronic pain syndromes, Morbidity, Post-stroke pain.

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INTRODUCTION

A stroke is a "brain attack" a sudden interruption of blood flow to the brain that causes brain damage and loss of function. Stroke is the first leading cause of long term disability worldwide. Various chronic pain syndrome occurs after stroke. Early recognition of those pain and managing promptly can prevent further damage and improve quality of life. Pathophysiology of post-stroke chronic pain is diverse. Some of them are nociceptive and some of them are neuropathic. Both central and peripheral sensitization leads to chronicity of pain. Dyspnea,

dysarthria and cognitive impairment are a hindrance for elicitation of proper history and examination findings. Some chronic pain conditions are common and easily recognized like hemiplegic shoulder pain, central post-stroke pain (CPSP), some are common but under-recognized like temporomandibular joint dysfunction (TMJD), compressive neuropathy of upper limbs, and some are rare like brachial plexus injury and heterotopic ossification.

MATERIALS AND METHODS

This study includes the stroke patients attending the neurology department, outdoor (n = 593) or indoor (n = 467) at Calcutta National Medical College, Kolkata from October 2015 to September 2017. Some of the patients treated before attending the neurology department were evaluated retrospectively (n = 318) and the rest (n = 742) from our prospective study. Inclusion criteria were the strength of the affected organ <4/5 as measured by the Medical Research Council Scale. Stroke occurring >3 months before study entry, pain duration >3 months, pain in visual analogue scale (VAS) >4 for the last 7 days. Exclusion criteria were pre-stroke pain, use of >1 non-opioid, or opioid analgesic for pain, use of analgesics for any other acute or chronic pain, hemi-neglect, medical instability, and impaired memory. A thorough history including history of pain in any part of the body, starting from head to toe were taken keeping in mind nociceptive (aching, throbbing) neuropathic (paresthesia, dysesthesia) and mixed character of pain. Pain score was done by VAS. Clinical examinations were done to detect neurological, musculoskeletal and other abnormality. Consultation of Orthopedician, rheumatologist and physical medicine experts were taken when necessary to identify proper causes of chronic pain. Detail sensory examination was done including touch (a wisp of cotton), pain (pin), temperature (hot and cold test tubes), joint position and vibration sense (tuning fork). Cortical sense for 2 point discrimination (2 point discriminator), graphesthesia (pencil), stereognosis. For somatic pain, inspection (color change, swelling), palpation (tenderness) and autonomic changes (swelling, temperature and trophic changes) were noted. Investigations including a blood test, neuroimaging [X-ray, color Doppler, computed tomography (CT) scan and Magnetic Resonance Imaging

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(MRI)], electrophysiology (EMG, NCV) and others were performed to confirm the diagnosis.

OBSERVATIONS

Only those patients who finished the study were taken total number of patient were 1060. Among them 44.06% were male and 55.94% (n = 503) were female. 70% (n = 742) had infarction, 21.9% (n = 233) were below 40 years of age. Hypertension noted among 50.9% (n = 540), hypercholesterolemia 17.3% (n = 184). Tobacco smoking 16.2% (n = 172) and diabetes mellitus in 56.1% (n = 595) mean VAS score 6 ± 2 .

Temporomandibular joint (TMJ) dysfunction occurred in 13.9% (n = 148). Neck pain in 40% (n = 424) shoulder pain 41.9% (n = 445) (Table 1). On imaging by MRI of shoulder joint various causes noted were rotator cuff tendinopathy 17.9% (n = 80). Shoulder subluxation 22.4% (n = 100), Deltoid tendinopathy 9.4% (n = 42) rotator cuff tear 11.9% (53) biceps tear 13% (58%) deltoid tear 7.8% (35), labral ligamentous complex abnormalities 6.9% (n = 31), others 10.3% (n = 46) (Table 2).

Nearly 1.03% (n = 11) had brachial plexus injury. Heterotopic ossification of elbow were noted in 0.6% (n = 7), compressive neuropathy due to poor positioning involved median nerve 41.2% (n = 437) and ulnar nerve 30.6% (n = 325).

Dependent edema of hand and forearm were elicited in 27.9% (n = 296). CRPS type I developed in 12.3% (n = 130), pressure ulcer in 31.03% (n = 329). Deep vein thrombosis 2.3% hemorrhagic stroke, 0.56% ischemic stroke. CPSP most popular form of post-stroke pain occurred in 11.03% (n=117). Spasticity developed in 21.03% (n=223). It was determined by the modified Ashworth scale.

Table 1: Pain syndrome

Causes	Number	Percentage (%)
Shoulder pain	445	41.9
Median nerve compression neuropathy	437	41.2
Neck pain	424	40
Pressure ulcer	329	31.03
Ulnar nerve compressive neuropathy	325	30.6
Dependent edema of hand and forearm	296	27.9
Spasticity	223	21.03
TMJ dysfunction	148	13.9
CRPS type 1	130	12.3
Post-stroke pain syndrome	117	11.03
Deep vein thrombosis	25	2.3
Brachial plexus injury	11	1.03
Heterotopic ossification of elbow	7	0.6

DISCUSSION

Temporomandibular joint dysfunction (TMD, TMJD) is an umbrella term covering pain and dysfunction of the muscle of mastication and temporomandibular joints. TMJD reported to be strongly associated with a decrease in chewing function which is contributing to dysphagia, malnutrition and bad oral health in post-stroke hemiparesis. In this study TMJD prevalence is 13.9%. Other study prevalence of TMJD is between 5 to 12%.¹ Neck pain occurs from bottom of the head to top of shoulders. It can spread to the upper back or arms. Pain comes from an unusual position of joints resulting from muscle tightness, spasticity or stiffness. For the entire adult population, the mean prevalence is 29.80%.² There is hardly any study depicting the prevalence of neck pain in stroke. In this study, it is 40% higher than that of the population in the community. Hemiplegic shoulder pain causes considerable distress and reduced activity and can markedly hinder rehabilitation. The aetiologies are multifactorial and ideal management is prevention. Prevalence of shoulder pain in stroke population is 40%.³ Prevalence of shoulder pain among adults of age 30 to 70 years is 22.9%.⁴ Shoulder pain starts within days to weeks of stroke onset and incidence is slightly higher in the male. Our study shows it to be 41.9%. All patient (n = 445) of hemiplegic shoulder pain undergone MRI (1.5T) by T₁ and T₂ weighted multi-planer and multi-sequence magnetic imaging. All scan were reviewed by one radiologist.

Full-thickness muscle tear was detected by full thickness gap in the entire width of the muscle both coronal and sagittal image. Partial thickness tear detected by an abnormal signal in the bursal and articular surface in T₂. Tendinopathies were diagnosed by thickening and abnormal signal in proton density not in heavy T₂. Labral ligamentous complex abnormalities were diagnosed by a tear of anterior part of the posterior aspect of the labrum.

There are few studies performing of MRI of the shoulder joint in hemiplegic shoulder pain. Full thick-

Table 2: MRI of shoulder

Abnormalities	Number	Percentage (%)
Rotator cuff tendinopathy	80	17.9
Shoulder subluxation	100	22.4
Deltoid tendinopathy	42	9.4
Rotator cuff tear	53	11.9
Biceps tear	58	13
Deltoid tear	35	7.8
Labral ligamentous complex abnormalities	31	6.9
Others	46	10.3

ness rotator cuff tear was reported to be 20.7% in the general population.⁵ In another study tendon and ligament lesions were 57.1% and rotator cuff tear 38.1% in hemiplegic shoulder pain cases.⁶

Brachial plexus injury can occur due to shoulder subluxation or improper handling of upper extremities, and they can be confirmed by electrophysiological evidence of upper trunk involvement. Kaplan et al. presented 5 cases of hemiplegic brachial plexus injury.⁷

Heterotopic ossification is the abnormal growth of bone in the non-skeletal tissues including muscle, tendons or other soft tissues of which the etiology is usually either traumatic or neurogenic. Neurogenic HO is a known but uncommon complication that occurs after cerebral or spinal insult. The occurrence of HO in the post-stroke patient was reported to be 0.5–1.2%.⁸ Prolong immobilization and subsequent manipulations of joints with force to preserve ROM are the key causative factors.⁹ In this study, heterotopic ossification of the elbow was observed in 0.6% (n = 7) of the hemiplegic elbow.

Compressive neuropathy due to poor positioning of the paretic limb may cause morbidity after stroke. Nerve conduction studies confirm the diagnosis. In one study it has been shown in the paretic limb 37.5% suffered from median nerve entrapment neuropathy at the wrist and another 37.5% from ulnar nerve neuropathy at the elbow, whereas in non-paretic side it was 25% and 18.7%, respectively.¹⁰ This study showed 41.2 and 30.6% on the paretic side whereas on the non-paretic side it was 28.9 and 10% respectively. This type of variation can occur as populations and rehabilitation differs from region to region.

Swelling and edema of hand developed due to increased venous congestion related to prolonged dependency and loss of muscle pumping function in the paretic limb. Swelling can be measured by hand volumeter or measuring tape and edema by palpation. We have used the later methods. In this study, dependent edema of hand and forearm were elicited in 27.9% (n = 296). In one study edema of the hand were observed in 33% of patients.¹¹

Complex regional pain syndrome I (CRPS type I) is a chronic pain condition characterized by prolonged and excessive pain and changes in skin color, temperature and/or swelling in the affected arm, leg, hand or foot. In post-stroke CRPS usually involves shoulder and wrist with the elbow relatively spared. It has also named as a shoulder-hand syndrome. CRPS I can be diagnosed by the Budapest clinical diagnostic criteria.¹² In our study CRPS type I were elicited in 12.3% (n = 130) by the Budapest criteria. In our study CRPS type I was noted to be 15% by IASP criteria.³ This disparity can be explained by the facts that IASP criteria is more sensitive but later developed. Budapest criteria are more specific.

Pressure ulcers also called pressure sores, bedsores and decubitus ulcers are of tissue damage that occurs in people who cannot turn themselves like, stroke, acutely ill or elderly immobile persons. Categories of PU can be classified as epidermis (grade I), partial thickness dermis (grade II) partial thickness subcutaneous tissue (grade III), muscle and bone (grade IV). In this study prevalence of PU were observed to be 31.09% (n = 329) which is slightly higher than that of 28%.¹³

Deep vein thrombosis occurs when blood clots forms in any one of the deep vein of the body usually in legs. Stroke is a risk factor for developing DVT. In one study it was noted that the incidence of DVT in hemorrhagic stroke 1.9% and ischemic stroke 0.5%.¹⁴ This study showed the incidence of DVT were 2.3% (n = 25) in hemorrhagic stroke and 0.56% (n = 6) in ischemic stroke. Although the incidence is slightly higher, but the ratio remains the same.

Central post-stroke pain, formerly known as thalamic pain syndrome of Dejerine and Roussy, is a central neuropathic pain occurring in patients affected by stroke. This disease affects an estimated 8% of all stroke patients and possibly more.¹⁵ CPSP usually starts within 4 to 8 weeks of stroke but can appear within 1 year. Usually, 60% of them are thalamic in origin. In this study, CPSP was observed in 11.03% (n = 117) of stroke patient which is similar to the above and other studies.

Post-stroke spasticity is a common complication associated with other signs and symptoms of the upper motor neuron syndrome. It contributes to impairment and disabilities. Clinical measures of spasticity were done by the modified Ashworth scale. Prevalence of spasticity after stroke was noted to be of 27% by modified Ashworth scale.¹⁶ Our study showed it to be 21.03% (n = 223).

CONCLUSION

Chronic pain syndromes are common, even following mild to moderately severe stroke. Chronic pain is associated with greater decline in physical and cognitive health making it an important medical complication of stroke. It is a source of anxiety, depression, and a hindrance to physical rehabilitation. Early identification of the type of pain and their proper management can reduce the morbidity. Consultations of different medical specialties like orthopedics, rheumatologist, psychiatrist, internist, and radiologist are often required to pinpoint the diagnosis. Both nociceptive and neuropathic pains are recognized as a complication of stroke. Their occurrence according to descending order of frequency is shoulder pain, median nerve compression neuropathy, neck pain, pressure ulcer, ulnar nerve compressive neuropathy, dependent edema of hand and forearm, spasticity, TMJ dysfunction, CRPS

type 1, post-stroke pain syndrome and brachial plexus injury.

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