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Brachial Plexitis: A Rare and Often Misdiagnosed Postoperative Complication

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Abstract. Surgeons should be aware of the brachial plexitis syndrome in order to properly make an early diagnosis and educate the patient and his family on the etiology of this syndrome. It is characterized by the acute onset of shoulder pain, weakness, and paralysis in patients following a variety of surgical procedures.

Key words: Brachial plexitis—Bilateral gynecomastia—Subcutaneous mastectomy

Brachial plexitis is a well-defined syndrome characterized by the acute onset of shoulder pain, weakness, and paralysis in patients following a variety of surgical procedures [2,3]. While this syndrome is well documented in the neurological literature, it has received little attention in the surgical literature. The purpose of this report is to familiarize the plastic surgeon with the clinical presentation and the natural history of this syndrome.

Case Report

A 15-year-old, healthy, left-handed, white male college student on an athletic scholarship underwent an uncomplicated subcutaneous mastectomy for bilateral gynecomastia. Two days following this procedure he complained of the acute onset of right shoulder pain. Physical examination demonstrated severe weakness of the right shoulder and inability of the patient to raise his shoulder more than 45 degrees. The right shoulder showed A-C knotching (Figs. 1, 2, 3). The patient was initially misdiagnosed by a neurosurgeon to have a cervical spine injury presumably from hyperextension of the neck during intubation. Two days later a negative computed tomography (CT) of the cervical spine lead to the second misdiagnosis of brachial plexus injury by an orthopedist. Three days went by until the proper final diagnosis of brachial plexitis was made by a neurologist. Had the proper diagnosis been made 7 days earlier, with an explanation of the etiology and prognosis of the disease to all concerned parties, an otherwise unbearable situation would have been slightly eased.

The patient had decreased sensation to pinprick over the lateral aspect of the right upper arm. The wrist and finger strength and sensation were normal. Routine serum laboratory tests were normal. Cervical spine films and magnetic resonance imaging (MRI) were unremarkable. An electromyogram (EMG) and NCV showed moderate muscle and nerve injury. He responded to analgesics and physical therapy; 2 months following the injury the patient was able to regain full range of motion in the right upper extremity (Figs. 4, 5); however, he did have some residual weakness.

History

Spillane in 1943 was the first to recognize brachial plexitis as a distinct clinical entity. Since then however, the syndrome has been given a variety of names including acute brachial neuritis, paralytic brachial neuritis, shoulder–girdle neuritis, neuralgic amyotrophy, and Parsonage-Turner syndrome [1–6,9]. Parsonage in 1948 published an extensive analysis of the syndrome in 136 military personnel from the World War II command which included patients from the United Kingdom, India, Burma, and other parts of the world [2]. Today this syndrome is widely recognized in the civilian population by neurologists.

Incidence and Etiology

The incidence of brachial plexitis is estimated to be approximately 1.64 in 100,000 with an age range from 3

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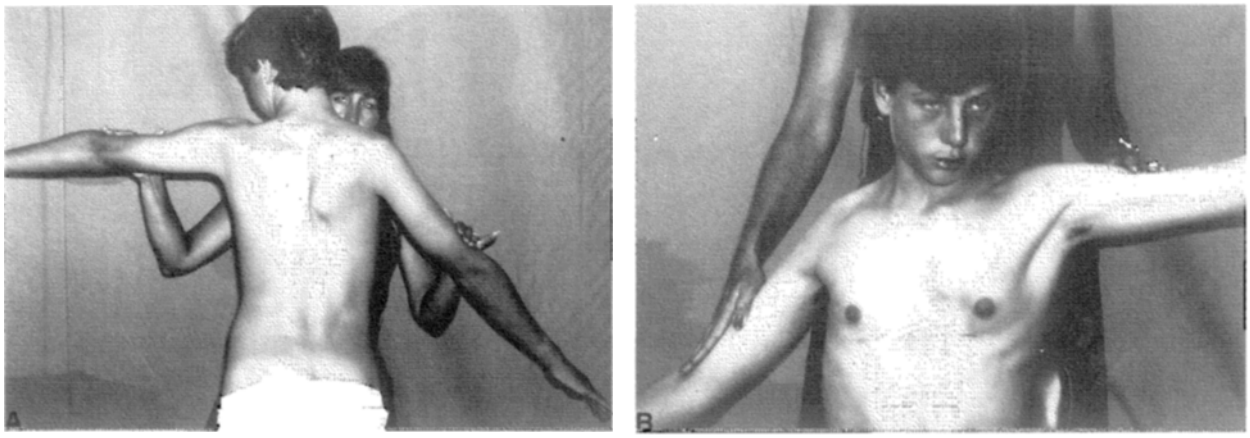

Fig. 1. (A) Five days S.P. Severe weakness with winging of the scapula affecting right arm and shoulder. (B) Five days S.P.

months to 75 years of age [3,10]. There is a male predominance. Most cases occur unilaterally but bilateral cases have been reported [12]. No definite etiology has been identified but a number of precipitating events have been implicated in the onset of symptoms. The etiology may include bacterial, parasitic, and viral infection [1–3]. Surgery or trauma to areas remote from the shoulder girdle appears to be the precipitating event in all cases [1–3,11]. Immunizations including tetanus toxoid, tetanus antitoxin, DPT, smallpox, and swine flu [1–3,6,7]. Others think the disorder is a manifestation of a systemic allergic hypersensitivity or auto-immune reaction [3,6,7]. Surgery from the shoulder girdle have most often been associated with the onset of brachial plexitis [1–3,11].

Operations that have been associated with the syndrome include hernia repairs, appendectomies, gastrectomies, meniscectomies, and mastoidectomies as well as other surgical procedures [1–3,11]. This is the first case of brachial plexitis to be presented in the plastic and reconstructive literature. Development of the symptoms in these patients is not thought to be related to a traction injury of the brachial plexus or pressure palsies secondary to positioning, but due to a systemic reaction triggered by the stress of surgery or trauma [1–3,11]. The onset of symptoms usually occurs within approximately 5–7 days following the surgery which emphasizes that mechanical injury is an unlikely etiology [3,4,9,11].

Typically a few days to 2 weeks pass after the precipitating event before the onset of the syndrome [1–4,9,11]. Pain in one or both of the shoulders and the scapula is the prominent characteristic of this disorder. Occasionally pain is not associated with an otherwise [2–4] typical case. The pain is described as constant, intense, sharp, stabbing, or aching and is more pronounced at night [1–4,11]. The pain is usually localized to the shoulder but often radiates into the neck and down the arm and forearm. The wrist and hand are spared [3,12]. Abduction of the shoulder as well as flexion and extension of the elbow usually aggravates the pain [2,3,12]. The patient is most comfortable with the shoulder adducted and elbow flexed [2,3,12] The pain, unlike cervical root pain, is not affected by coughing, sneezing, or neck movements. There are no constitutional symptoms associated with the syndrome [3]. Sever pain persists for a few hours to 3 weeks but can last much longer. It is frequently replaced by a milder ache which may last for over a year. As the pain begins to subside, weakness is noted 1–7 days after onset of symptoms but can occur as late as up to 14 days. The severity of weakness varies but is most commonly found in the distribution of the upper plexus in the shoulder–girdle muscles [1,2–4,11,12]. The paralysis involves the lower motor neurons and is characterized by muscle flaccidity and rapid wasting without a true motor, radicular, or nerve trunk pattern. There is no relationship between the location of the initial pain and the subsequent paralysis [2–4]. Muscle weakness generally follows multiple peripheral nerves, by one or more nerve roots, or by a combination of one or more peripheral nerves and the nerve root [3,4,8,9]. The axillary nerve is the most commonly involved followed by the suprascapular nerve. When two peripheral nerves are involved, it is these two nerves that are most frequently seen in combination [3,4,8,9]. When roots are affected, the most commonly involved and they are seen in combination are the C5,6,7 [8]. Deltoid muscle paresis is the most frequent followed by the supraspinatus, infraspinatus, and serratus anterior [1–4,11]. Moderate to severe atrophy is seen in the majority of patients. Sensory loss ranges from 30%–70% and commonly involves a small area [1–4,11] of hypoesthesia overlying the deltoid muscle followed frequently by hopoesthesia of the radial surface of the forearm [3,8]. These changes, however, do not parallel the motor changes [1–4,8,9,11]. Proprioception is spared. Deep tendon reflexes vary according to the severity of the initial symptoms [4] but become hypoactive or absent occasionally in 25%–30% of such patients [4]. Hypoactivity of the biceps and triceps reflexes are the most common [4].

Diagnosis and Laboratory Studies

Brachial plexitis must be differentiated from other causes of shoulder pain which include poliomyelitis, cervical spine pathology, brachial plexus trauma, rotator cuff injury, bursitis, and peripheral neuropathies. Routine labo-

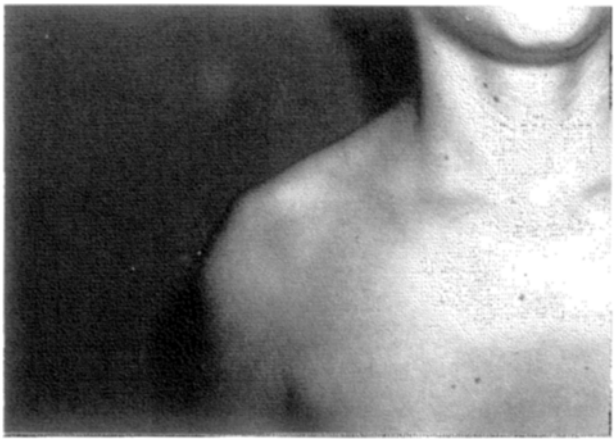

Fig. 2. Right side AC knotching.

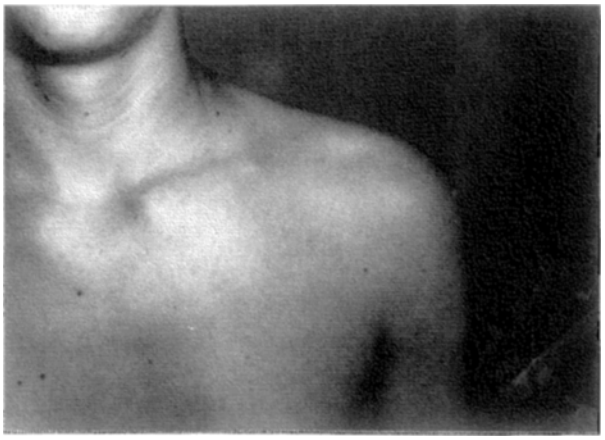

Fig. 3. Left side normal AC junction.

ratory studies including erythrocyte sedimentation rate, hemoglobin level, hematocrit, white blood cell count and serum electrolyte concentrations, urinalysis and liver function test and CSF are invariably normal in patients with brachial plexitis [3,4]. Viral and immunologic studies reveal no abnormalities. Plain radiographs of the cervical spine are normal. Likewise MRI and myelograms show no specific abnormalities. Electromyography results vary depending on the timing of the injury and the severity of neural damage [3,4,8,9]. Needle electrical examination shows fibrillation potentials and positive waves suggesting denervation of the affected muscles after approximately 3 weeks [8,9,11]. At 3 months giant polyphasic potentials are demonstrated indicating early recovery of the denervated areas. EMG recovery depends on several factors including, age of the patient, severity of the lesion, the length of nerve involved, and systemic diseases like diabetes and ethanol abuse [8,9,12]. Bilateral EMG studies in patients with unilateral symptoms may show EMG abnormalities in the contralateral, asymptomatic extremity. Nerve conduction studies indicate that axonal degeneration in the brachial plexus and/or on multiple nerves originating from the plexus. Median and ulnar nerve conduction times are normal [8,9].

Analgesics and physical therapy, including passive and active range of motion, are the treatment of choice in these patients [3]. Corticosteroids have not been proven to be beneficial. Prognosis in patients with brachial plexitis is usually good with full recovery anticipated. The time of recovery to normal function is variable ranging from several months to several years depending on the severity of the initial symptoms and the degree of muscle atrophy [3].

Conclusion

Surgeons must be aware of this syndrome in order to make an early diagnosis and, through patient education, to absolve the surgical team of any wrongdoing. Early diagnosis and supportive reassurance to the patient and family members also lessens the emotional agony that accompanies any postoperative complication with symptoms of such magnitude.

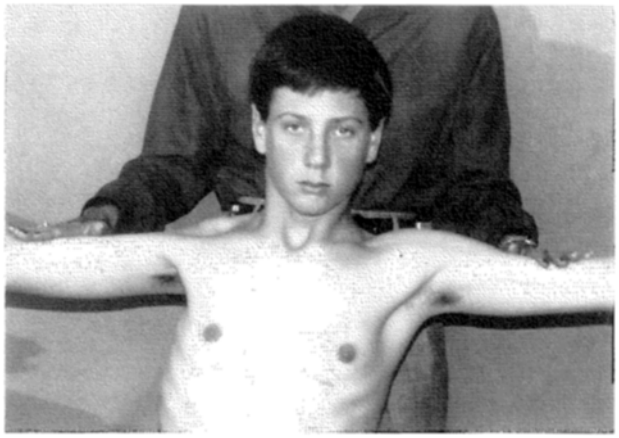

Fig. 4. Six months recovery—70%.

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