정맥 주입의 혈관외유출로 인한 전골간신경 증후군

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Anterior Interosseous Nerve Syndrome due to Extravasation of Intravenous Infusion

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Anterior interosseous nerve is a motor branch from median nerve. It innervates three muscles, the flexor pollicis longus, the flexor digitorum profundus, and the pronator quadratus muscles in the forearm. Anterior interosseous nerve syndrome refers to a neuropathy affecting this nerve, to develop weakness of innervated muscles. The etiology can be diverse and the pathophysiology is not clearly documented. We report a case of a 67-year-old male patient with diabetes, who presented symptoms of right thumb and index finger weakness, forearm pain, which was in conclusion diagnosed as anterior interosseous nerve syndrome through electrodiagnostic studies. His anterior interosseous nerve syndrome was revealed as due to fluid extravasation. Fluid extravasation can cause various side effects, and peripheral nerve injury is an uncommon side effect. This is a rare case of anterior interosseous nerve syndrome developed from fluid extravasation.

Keywords: Neuropathy, Extravasation

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Introduction

Anterior interosseous nerve (AIN) is a pure motor branch of the median nerve. AIN arises just below the elbow at the forearm level, and courses distally on the volar side of the interosseous membrane [1]. It innervates the flexor pollicis longus (FPL), the flexor digitorum profundus (FDP) to the index finger and the middle finger, and the pronator quadratus (PQ) in the forearm. AIN syndrome is an isolated palsy of these supplied muscles [1].

Extravasation injury is the damage of subcutaneous or perivascular tissues during intravenous infusion, caused by the efflux of

solutions from a vessel into surrounding tissue spaces [2]. Signs and symptoms of extravasation injury include pain, erythema, swelling, tenderness, local blistering, skin darkening, firm induration, ulceration, or full-thickness skin damage and injury can vary from minor to severe damages [2].

This case study represents a patient who developed AIN syndrome after intravenous fluid injection.

Case report

A 67-year-old male with past history of diabetes, visited the

outpatient clinic with complaint of edema and pain of right forearm and weakness of right thumb. He could not flex his right thumb and the distal phalanx of right index finger. One week before the visit, he was carried to a local clinic due to hypoglycemic shock, and had intravenous dextrose fluid injection. After the fluid injection, he developed symptoms and signs of right forearm edema, erythema, heating sensation, and pain at the infusion site. On the physical examination, he still had pain on his forearm and edema without clear margin at the volar side of forearm below elbow. Neither heating sensation nor redness was observed, but general induration at the upper region of forearm could be palpated. He had weakness with motor grade 1 on Medical Research Council (MRC) scale in his interphalangeal flexor of right thumb and distal phalanx flexor of right index finger. Also, he could not make an "OK" sign with his right thumb and index finger.

Damage of anterior interosseous nerve was suspected, and nerve conduction studies (NCS) and needle electromyography (EMG) test were performed to confirm the AIN syndrome. NCS and EMG test was performed 15 days after the onset.

The sensory NCS revealed decreased amplitudes of sensory nerve action potentials in bilateral median and ulnar nerves, and the motor NCS revealed decreased conduction velocities in bilateral ulnar, peroneal and tibial nerves (Table 1).³ The needle EMG revealed abnormal spontaneous activities, increased insertional activities and reduced interference pattern in right flexor digitorum profundus, flexor pollicis longus and pronator quadratus muscles (Table 2). The patient was diagnosed with right anterior interosseous nerve lesion, concurrent with peripheral sensorimotor polyneuropathy of bilateral upper and lower extremities, which seemed to be diabetic polyneuropathy considering the patient's medical history. The results satisfied the simplified criteria for the electrodiagnostic confirmation of distal symmetric polyneuropathy with abnormal values in peroneal motor NCS, and abnormal contralateral median and ulnar sensory NCS and ulnar motor NCS [4]. We could conclude that AIN injury was attributed to intravenous fluid injection.

After confirming the diagnosis, physical therapy of stretching and strengthening exercise was done to recover strength and function. Also, physical modalities of ultrasound, hot pack and interferential current therapy were applied and oral nonsteroidal anti-inflammatory drug was prescribed for pain management. After one month of therapy, the symptoms of forearm edema and pain improved, and the patient showed improvement of muscle strength in interphalangeal joint flexor of thumb and distal phalanx flexor of index finger from MRC scale 1 to 2. He showed spontaneous recovery to some extent without surgical or aggressive interventions.

Discussion

Peripheral nerve damage is not a common complication of extravasation injury. According to a retrospective analysis of 67 adult patients who developed intravenous catheters related complications, hand was a common site for minor and major complications [5]. The most frequently reported complications of extravasation were phlebitis and cellulitis, and two patients developed compressive neuropathies due to infiltration in hand and wrist, which were median nerve palsy and AIN palsy [5]. One of them required nerve decompression and tendon transfer [5].

Puhaindran, et al. introduced a case of AIN syndrome in a 42-year old male, after peripherally inserted central catheter insertion into a brachial vein for the administration of intravenous antibiotics [6]. He had the catheter for a long course of intravenous antibiotics, and eventually developed pain of burning nature and edema in his forearm. The EMG confirmed the AIN palsy which was attributed to the line insertion and infusion [6]. It has been estimated that between 10% and 30% of patients receiving intravenous therapy may experience AIN palsy as complication [7]. In our case, the patient may have been injected intravenously with median cubital vein and AIN palsy have developed due to extravasation of the fluid.

Even though AIN is a pure motor nerve, and no sensory loss occurs, pain may be present in the forearm. Patients with AIN syndrome are unable to make an "OK" sign with thumb and index finger, due to paralysis of FPL and the radial FDP.¹ This was also consistent with our subject. The most definite confirm of the diagnosis and disease severity is evaluation by electrodiagnostic studies. No optimal treatment of AIN syndrome has been established, but once the diagnosis is made, avoidance of aggravating activities, rest, and anti-inflammatory medication has been recommended. Most of the patients with AIN syndrome have improvement in symptom without any surgical intervention. Non-surgical conservative management for at least 12 weeks are still recommended. This was also true for our patient. Physiotherapy specifically towards pain, and soft tissue massage, stretch and strengthening exercises may be used, and it brought improvement of pain and motor scale in our patient.

The AIN palsy comprises less than 1% of upper extremity nerve palsies [6]. Etiology of AIN palsy can be categorized into traumatic and non-traumatic/spontaneous causes, and common factors of the non-traumatic factors include neuralgic amyotrophy, isolated neuritis such as brachial plexus neuritis, and entrapment neuropathy, since the nerve is susceptible to compression by soft tissue, vascular or bony structures [1]. Collins and Weber considered entrapment as by far the most common cause of AIN

Table 1. Nerve Conduction Study

Sensory	Stimulation site	Recording site	Latency (ms)	Amplitude(μV)	
Rt. Median	Wrist	3rd finger	3.1	7.9*	
Lt. Median	Wrist	3rd finger	3.1	6.9*	
Rt. Ulnar	Wrist	5th finger	2.3	5.2*	
Lt. Ulnar	Wrist	5th finger	2.3	4.6*	
Rt. Peroneal	Ankle	Lateral ankle	2.4	4.5	
Lt. Peroneal	Ankle	Lateral ankle	3	4.7	
Rt. Sural	Calf	Lateral ankle	3.2	6.3	
Lt. Sural	Calf	Lateral ankle	2.3	6.7	
Motor	Stimulation site	Recording site	Latency (ms)	Amplitude (mV)	Velocity (m/s)
Rt. Median	Wrist	APB	3.8	10.8	
	Elbow	APB	7.9	10.5	53.7
Lt. Median	Wrist	APB	3.8	10.9	
	Elbow	APB	8.0	9.4	52.4
Rt. Ulnar	Wrist	ADM	3.0	6.0	
	Below elbow	ADM	7.2	5.7	47.6*
Lt. Ulnar	Wrist	ADM	3.0	5.8	
	Below elbow	ADM	7.3	5.9	46.5*
Rt. Peroneal	Ankle	EDB	4.7	5.3	
	Below fibular head	EDB	12.5	4.7	38.5*
Lt. Peroneal	Ankle	EDB	4.7	5.0	
	Below fibular head	EDB	12.4	4.8	38.9*
Rt. Tibial	Ankle	AH	3.7	8.5	
	Knee	AH	13.3	6.8	34.4*
Lt. Tibial	Ankle	AH	3.7	11.7	
	Knee	AH	13.2	8	34.7*

Rt.: right, Lt.: left, APB: abductor pollicis brevis, ADM: abductor digiti minimi, EDB: extensor digitorum brevis, AH: abductor hallucis.

*Abnormal value.

Table 2. Electromyography Study

Muscle	IA	Fib	PSW	IP
Paravertebral Rt. C5-T1	Normal	-	-	
Paravertebral Lt. C5-T1	Normal	-	-	
Rt. Deltoid	Normal	-	-	Complete
Rt. Biceps	Normal	-	-	Complete
Rt. Triceps	Normal	-	-	Complete
Rt. FCR	Normal	-	-	Complete
Rt. APB	Normal	-	-	Complete
Rt. 1st DI	Normal	-	-	Complete
Rt. FDP	Increased	+	+	Single
Rt. FPL	Increased	+	+	Single
Rt. PQ	Increased	+	+	Single to partial
Lt. Deltoid	Normal	-	-	Complete
Lt. Biceps	Normal	-	-	Complete
Lt. Triceps	Normal	-	-	Complete
Lt. FCR	Normal	-	-	Complete
Lt. APB	Normal	-	-	Complete
Lt. PQ	Normal	-	-	Complete
Lt. 1st DI	Normal	-	-	Complete

IA: insertional activity, Fib: fibrillation, PSW: positive sharp wave, IP: interference pattern, Rt.: right, Lt.: left, FCR: flexor carpi radialis, APB: abductor pollicis brevis, DI: dorsal interosseous, FDP: flexor digitorum profundus, FPL: flexor pollicis longus, PQ: pronator quadratus.

palsy [8]. Neuropathies such as brachial neuritis mimic the clinical manifestations of an AIN neuropathy, so differential diagnosis and confirmation is needed.

Extravasation is not a known common cause for AIN palsy. All fluids can cause tissue damage, but certain substances may cause damage with greater risks. Soft tissue damage due to extravasation depends on the type of the agent, which can be classified as irritants or vesicants. For some known potential causes of severe tissue necrosis, there are parenteral alimentation fluids, antibiotics, calcium, potassium, and sodium bicarbonate solutions [2]. Any hyperosmolar solutions with osmolarity greater than plasma, including parenteral nutrition solutions can cause tissue damage by osmotic pressure [7].

There are some identified risk factors for extravasations. Patients at risk of extravasation injuries, include those with small, fragile or thrombosed veins increasing the risk of leakage, with chronic diseases, those who are unable to communicate due to confused state or language issues, those on anticoagulants, or those who are obese which makes detection of misplacement difficult, or have undergone multiple intravenous cannulations or venepunctures [9]. Patients with fragile veins include conditions such as cancer, whose veins are more fragile, mobile and difficult to cannulate, peripheral vascular diseases, or patients with diabetes [9]. Diabetic patients with peripheral neuropathy may not be aware of pain or discomfort of an infusate leakage which will as a result, increase risk of extravasation injuries [2,7]. Other patient risk factors involve the elderly because of fragile skin and veins, or neonates and infants with small veins difficult to cannulate [7].

The etiology of tissue damage resulting from extravasation can be categorized into following pathophysiological mechanisms ; vasoconstriction and ischemic necrosis, direct toxicity to the tissue, osmotic damage, extrinsic mechanical compression by large solution volumes, or superimposed infection [7]. The severity of the result varies from minor symptoms to serious sequelae such as necrosis, tissue loss or contractures or deformities of joints, and we introduced a case of peripheral neuropathy. The past history of diabetes and injection of hyperosmolar solution would have increased the risk of extravasation in our subject. Our patient may have been injected dextrose fluids over 20% for hypoglycemia. There are some reports of extravasation injuries after dextrose infusion and in many cases dextrose exceeded 10% [10]. In our case, hyperosmolar dextrose solution and increased pressure of the interstitial space have led to nerve injury. The leakage of hyperosmolar dextrose leads to fluid shifting from intracellular to extracellular tissue spaces, and thus drawing fluid from the neighboring cells and blood cells [10]. This increased

osmotic pressure can compress and damage blood vessels, nerves, and muscle tissues [10]. Furthermore, increased fluid and pressure can also lead to compartment syndrome, damaging the surrounding muscles and nerves.

The patient developed anterior interosseous nerve injury after dextrose administration through the median cubital vein. Anatomically, the anterior interosseous nerve accompanies the anterior interosseous artery along the interosseous membrane and supplies the deep muscles on the anterior forearm. The median cubital vein generally runs just below the cubital region, connecting the basilic and cephalic vein. The anterior interosseous nerve lies deeper than the median cubital vein, but it can be assumed that the pressure via extravasation caused injury to the nerve in this case.

The AIN injury in our case was confirmed by the electrodiagnostic study. However, since the study was performed 15 days after the onset, it had limitations in detecting abnormalities. If follow up electromyography was performed more than three weeks after the injury, further recovery information such as large motor units or polyphasic potentials would have been revealed. Also, electromyography of pronator teres muscle would have been helpful to localize and to rule out more proximal lesions of the median nerve, such as pronator teres syndrome. Another limitation to our case is that no imaging studies were performed. Forearm imaging evaluations such as magnetic resonance imaging, ultrasonography, or computed tomography would have been helpful to confirm nerve compression or soft tissue injury.

This is a case of AIN syndrome which occurred after intravenous fluid injection in a diabetic patient due to extravasation. The peripheral neuropathy could have been caused by direct compression from the fluid or inflammatory reactions. To prevent such neurological injury, special caution is needed when fluid is infused through intravenous line, especially on fragile sites, in high risk patients or with substances with higher risks.

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