

Brachial Plexus Injury ? After Brachial Plexus Block

¹ Bishwarad M Shrestha

² Narendra B Basnyat

A young lady of 27 years suddenly, after surgery for olecranon fracture, developed complete motor paralysis of right hand. The surgery was done under Brachial plexus block. Whether it was due to surgery or anaesthesia, is still a question of debate, as there are numerous other causes also which can cause this type of nerve damage. Fortunately, the patient is improving and review of literature reveals, that such paralysis recovers after intensive physiotherapy in about three months.

CASE REPORT

M K - 27 years old female patient was admitted on May 28, 1990, from Emergency Department of Bir Hospital, with diagnosis of fracture right olecranon and fracture pubic rami. She had fallen from about 15 ft. of height on sustaining electric shock. She was conscious and had no vomiting after falling from height. Open reduction and internal fixation with tension band wiring was done on 15th June 1990. Her hand was covered with posterior slab and bandage. Preanaesthetic nerve function test could not therefore be done before operation in preanaesthetic check up.

A Regional Brachial Block was given. Twenty ml, of 5% Xylocain with adrenalin (Asta) was deposited above the first rib with supraclavicular approach.

A single shot injection without eliciting the paraesthesia, by hitting the first rib, was performed. After 10 min. of brachial plexus block, operation was started, applying the

pneumatic tourniquet. The anaeroid barometer of the tourniquet was not in proper condition, so the pressure sufficient for haemostasis, as thought by the surgeon was applied. Inj Ketamin 50 mg intravenously was added after 45 min of brachial block as the patient complained of little pain at operation site. The duration of operation was 1 hour, while tourniquet application time was 1 hour and 15 min. The course of operation was uneventful.

Two days after operation, anaesthetist was informed that the patient had developed right wrist drop. A thorough clinical examination was performed with the help of neurologist. There was hyperalgesia on deep pressure and pinprick but hypoalgesia on light touch from tip of finger to the middle arm. Finger's joint position sensation was impaired but joint mobility of shoulder was present. Vibration was absent from fingers tip to elbow but present above elbow joint. Radial supinator and biceps reflexes were absent, triceps reflex was present. Touch and pinprick as well as muscle power in deltoid, supraspinatus

1 MBBS, DA, Senior Consultant Anaesthetist, Bir Hospital.

2 MD (Hons), DA, Anaesthetist, Bir Hospital.

infraspinatus and trapezius were intact. An electromyography was also done which showed severe injury of the posterior cord and partial injury of lateral and medial cords. Sensory, touch and temperature sensations were completely intact and there was no disturbances in sympathetic function.

DISCUSSION

When apparent nerve damage follows a surgical procedure carried out under brachial plexus anaesthesia, as evidenced by the appearance of persistent sensory and or motor deficit with or without paraesthesias, the etiology is extremely difficult to assess because of the multitude of factors, that might be involved.

The list below gives the possible causes, that may be operative,

1. Operative Factors

i) Pre-operative Factors: Pre existing neurological disease or damage latent or overt.

ii) Intra-operative Factors:

- a) Surgical procedure and its accompanying trauma.
- b) Damage due to the tourniquet utilized for hemostasis.
- c) Chemical damage due to the local anaesthetic agent itself.
- d) Malpositioning of an anaesthetized extremity on the operating table (traction injury).
- e) Neural damage due to needle trauma and/or intraneural injection.

iii) Post-operative Factors: Trauma that can occur when the anaesthetized extremity is paralysed and insensitive to pain during the subsequent recovery period, while the block is wearing off.

2. Electric Current

3. Tight posterior slab.

4. Fall from height leading to cervical injury

or brachial plexus injury following fracture of first rib.

5. Paralytic Brachial neuritis - which can be initiated after operation.

Some other factors, though not applicable in this case, are-

6. Congenital anomalies such as presence of a cervical rib, anomalous derivation of the plexus (especially a prefixed plexus), hypertrophy of the scalene muscle.
7. Apical tumor - Pan coast syndrome
8. Toxic - specially after heroin injection
9. Radiation - after many years.
10. Plexitis following vaccines - such as TABC, DPT, etc.

A joint clinical meeting was organized in Bir Hospital with participation of Anaesthetists, Surgeons, Orthopaedicians, Neurologists, Physiotherapists and other many interested doctors from Bir, TUTH, Kanti and Tri Chandra Military Hospital to discuss the possible etiology in this case. The discussion was in favour of Tourniquet paralysis.

Tourniquet paralysis syndrome is a localized form of mechanical nerve damage, which affects primarily the larger A-alpha and A-beta fibers, so that the motor, power touch, pressure, vibration and proprioception are affected rather than pain and sympathetic function, which are subserved by the smaller A-delta and C-fibers. There are four definitive diagnostic criteria for the tourniquet paralysis syndrome. (1) There is motor dysfunction with paralysis and hypotonia or atonia, but no appreciable atrophy. (2) There is sensory dissociation wherein the modalities of touch, pressure vibration and position sense are usually absent, while temperature and pain remain intact. (3) Sympathetic function is not affected so that pilomotor and psychogalvanic reflexes are intact and the skin colour, temperature and plethysmographic findings are all normal. And (4) the response to electrical stimulation indicates a conduction block characterised by no response to stimulation of the motor nerve above the level of injury but a good response below it. More or less all the criteria mentioned above were seen

in our patient, but still we could not conclude that it was only tourniquet paralysis syndrome because it needs a very high pressure for a prolonged time for tourniquet paralysis to develop and that there was a conduction block not only above the level of injury but also below it.

The other most possible cause was positional paralysis. If apparent positional injury had occurred, the extent and degree of paralysis should have been noted immediately upon awakening. According to Clausen, sensation is less frequently affected than motor power and in many cases, it may not be involved at all, though he did report some patients who complained of anaesthesia, hypoaesthesia, hyperaesthesia and even paraesthesia. He stated that reflexes may or may not be present depending upon the severity of the injury and that usually but not invariably, tenderness appeared in the supraclavicular space 5-10 days after operation. In several of Clausen's cases Horner's Syndrome was also noted, indicating damage to the lower roots of the plexus with resultant sympathetic dysfunction.

Another potential source of nerve damage following brachial plexus block, perhaps more theoretical than real is chemical damage from the anaesthetic solution. If apparent chemical nerve damage should occur in spite of taking precautions, Wooly and Vandam have indicated that such damage can be differentiated from damage due to pressure (tourniquet or positional damage) in that chemical damage characteristically involves small fibers. Hence toxic damage causes anaesthesia, analgesia, hypoaesthesia, hypoalgesia, hyperalgesia, and spontaneous paraesthesias and in addition, there may be disturbances in sympathetic function.

Though the incidence is extremely rare, nerve damage secondary to brachial plexus block is possible either because of damage produced by the needle itself or because of damage produced by an intraneural injection. If an electromyogram performed immediately after surgery shows denervation activity, obviously this activity must have resulted from

a preexisting lesion or some other concurrent problem, since it takes 18-21 days for such activity to appear after the damage. It is well known that reflex sympathetic dystrophy can follow even the slightest neural or perineural trauma. A rather traumatic brachial block, especially with multiple injections could result in a reflex sympathetic dystrophy with progressive development of pain, vasomotor disturbances and even trophic changes, particularly if the block failed to produce the desired anaesthesia and the accompanying sympathetic blockade. Several of the cases of neurological sequelae reported in the literature after brachial block anaesthesia showed the classic signs and symptoms of 'Causalgia' and in several of these complete recovery was provided by a stellate ganglion block.

Other possible causes were also discussed but we could not conclude in favour of one single etiology.

CONCLUSION

Though tourniquet paralysis seems to be the most likely etiology of paralysis, other factors such as electric shock, fall from the height, brachial plexus block may also be operative in addition.

From our experience in this patient, we would like to conclude that such injuries can be prevented by-

1. Careful positioning of the extremity.
2. Doing careful preoperative neurological evaluation. Anaesthetist should be aware of all the possible factors which are capable of producing nerve damage.
3. Avoiding regional block in case as this when preoperative neurological evaluation is not easy and there are other factors (such as electric shock which may be causing neural injury).

ACKNOWLEDGEMENT

We are thankful to Lt Col Dr BB Swaraj, Dr RB Ghale, Dr GR Bajracharya, Dr BD Joshi, Dr Shova Aryal, Dr Purna Joshi, Dr BD Lekha.

for their active participation in the discussion. We would also like to thank Dr DB Shah and Dr Amina Shrestha for their kind cooperation

in assessing the neurological function of the patient. Lastly we thank Dr Ashok Bajracharya for his cooperation in treating the patient.

REFERENCES

1. Alon P Winnie. *Pexus Anaesthesia*. Vol I. Schultz Medical Information Aps Montegraue, 1983.
2. WD Wylie and HC Churchill - Davidson. *A Practice of Anaesthesia*. Alloydlike Publication. Chicago, 1972.
3. TH Taylor, Edward Major. *Hazards and Complications of Anaesthesia*. Churchill Livingstone. New York, 1987.
4. RS Atkinson et al. *A Synopsis of Anaesthesia*. Wrigt Psg Bristol, London, Boston, 1982.

PREVENTION OF PERINATAL TRANSMISSION OF HEPATITIS B

A Review by Debra Boyer MD

HBsAg+ mothers will transmit their infection to their babies in 1-2% of cases, or more if they are HBeAg+. A chronic carrier state with increased risk of chronic liver disease and hepatocellular carcinoma occurs in nearly all those babies whose infection is acquired perinatally.

A combination of active and passive immunization of all infants born to HBsAg + mothers has been shown to be effective in preventing hepatitis B in neonates. Current recommendations published by the American Academy of Pediatrics Committee on women should be screened for HBsAg routinely in the prenatal period. Children whose mothers are HBsAg + should be given HBIG (0.5ml), IM within 12 hours after birth. In addition, they should receive three doses of HB vaccine, 0.5 ml. The first dose should be given at birth, with subsequent doses given at 1 and 6 months of age. These recommendations are summarized in the following table.

Routine Pediatric Vaccination Schedule & HBV Prophylaxis for Infants of HBsAg-positive Mothers*

Age	Hepatitis B Prevention Schedule	HBV Marker Screening	Routine Pediatric Schedule
Birth	HBIG, ● HB vaccine ○
1 mo.	HB vaccine
2 mo.	DTP, OPV
4 mo.	DTP, OPV
6 mo.	HB vaccine	...	DTP
> 9 mo.	...	HBsAg and anti-HBs tests ▶	...
15 mo.	MMR
18 mo.	DTP, OPV, PRP-D

* Modified from ACIP; Post-exposure prophylaxis of hepatitis B (MMWR 84; 33:285-290)

● HBIG, 0.5 ml IM within 12 hours of birth

○ HB vaccine, 0.5 ml IM within 7 days of birth (prior to hospital discharge).

▶ HBsAg positivity indicates failure of immunoprophylaxis. Anti-HBs positivity indicates successful immunoprophylaxis. If negative for HBsAg and anti-HBs, fourth dose of vaccine should be administered, followed in one month by repeat testing for HBsAg and anti-HBs.

Report of the Committee of Infectious Diseases 1988, American Academy of Pediatrics.