Recovery Of Intra Parenchym Lesion Hemorrhagic Stroke
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Abstract
Recovering from a stroke can be a lengthy process that requires patience, hard work, and commitment. It may take years to recover. Recovery time after a stroke is different for everyone—it can take weeks, months, or even years. Some people recover fully, but others have long-term or lifelong disabilities. Strokes can cause significant impairment in language, cognition, motor, and sensory skills. This is why it’s considered to be a leading cause of serious long-term disability. The hemorrhage at the left cortex and subcortex of temporal lobe gives manifestation to contralateral hemiparesis. In acute setting, we should optimize the cerebral blood oxygen perfusion, so the penumbra area can survive. After surviving the acute stage, we have to assest functional prognosis in activities and optimized it because patient still has the contralateral extremities to help with activities.

Keywords: Functional ability, Hemorrhagic stroke, Rehabilitation,

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Foreword
Strokes can be broadly classified as hemorrhagic or nonhemorrhagic. Intracerebral hemorrhage (ICH), classified as either primary or secondary, occurs in ≈10% to 15% of all strokes and is associated with a higher risk of fatality compared with cerebral infarction. Intracerebral hemorrhage (ICH) is a highly morbid disease process due to the mass effect and secondary injury that occurs upon the surrounding brain. The majority of recovery after ICH occurs early, within the first few months post-stroke.

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It is generally believed that hemorrhagic stroke survivors have better neurological and functional prognoses than nonhemorrhagic stroke survivors.

Case Presentation
ANAMNESIS (heteroanamnesis from wife and medical record) on October 6th 2017
Chief complaint:
Weakness on right extremities
History of present illness:
At night on October 3rd 2017 Mr.K suddenly felt weakness on his right arm and leg, 15 minutes before he lost his consciousness. He was brought to Fatmawati Emergency Room, his blood pressure was 200/120mmHg, respiration rate and oxygen saturation were in normal limit, his blood kalium was critically low and was corrected on the next day.

On October 4th 2017 he had head CT scan with result intra parechymal hemorrhage of left temporal lobe with perifocal oedem (estimated volume 17,19 cc), left maxila and left ethmoidal sinusitis. He also had chest X-rays with result cardiomegaly with lung oedem suspected. He was then hospitalized in High Care Unit.

He regained his consciousness on october 4th 2017. He opened his eyes but he mostly slept during day and did not notice his wife

He was then diagnosed as cerebrovascular disease stroke hemorrhagic and consulted to PMR departement on October 5th 2017. He was fully awake, his speech was poorly understood. His blood pressure was 204/114mmhg, normal
saturation and respiration rate with oxygen 3 liter/minute on nasal canule. There is minimal slime on both of his lung. He was asked to bedrest, positioning head up 30 degree, joint stifness prevention and slem retention prevention.

He has been suffering from hypertension since 2 years ago and does not take his medicine (captopril) regulary. He smokes cigarette since 3 years ago, one pack/day.

History of past illness:
Sinusitis went to one-day surgery on September 26th 2017, he got paracetamol, mefenamic acid, rhinofed as post operation medication.

History of family illness:
There is history of hypertension on the family and his uncle suffered stroke.

History of functional ability:
Normal

Psychosocialeconomy status:
Patient was the breadwinner of his family. He has two daughter, the oldest already work, but the youngest still on university in bandung. His wife sometime earn a living by making and selling cake. His income is 9 million rupiah/month and his wife can earn 1-2 million rupiah/month.

He worked as Human Resource Development (HRD), he used to manage employee recruitment and their career. He went to work by riding motorcycle. He uses BPJS for medical insurance.

III. PHYSICAL EXAMINATION on October 6th 2017

General physical examination
- Consciousness : comos mentis
- Vital sign:
  - BP : 200/103 mmHg
  - HR : 98 beat per minute (regular)
  - RR : 26 times per minutes
  - SaO₂ : 98% with nasal canule 3 L/m
- Head:
  - No anemic conjunctiva
- Chest: Symmetrical shape and movement of both hemithorax
  - Heart : regular heart sound, no murmur.
  - Lung : vesicular breath sound right = left, slime minimal on lower and posterior aspect of the lung. No rhonchi
- Abdomen : flat & supple
- Neurologic examination:
  - Communication :
    - Receptive : good
    - Expressio : poor (slurred speech)
  - Swallowing: not evaluated. Patient on NGT already.
  - Cranial nerves:
    - Parese right N.VII central
    - Parese right N XII
  - Motoric examination : right hemiparetic, non functional; right upper extremity is weaker than right lower extremity.
  - Sensory examination : not evaluated
  - Elimination function :
    - Micturion : on DC
    - Defecation : cannot be evaluated because patient has not defecated yet
  - NIHSS: 13

IV. SUPPORTING MEDICAL EXAMINATION
- CT Scan (October 4th 2017)
Perdarahan intraparenkim lobus temporal kiri dengan perifokal edema dengan estimasi volume 17,19 cc. Sinusitis maksilaris kiri dan ethmoidalis kiri.
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Thorax X-Ray (October 4th 2017)
Kardiomegali dengan suspect tanda bendungan paru.

Laboratory
- Blood: (October 4th 2017)
  - Routine blood exam: within normal limit, except for Hb: ↓ 10.3 g/dl; eritrosit ↓ 3.78 juta/ul.
  - Liver function: Ureum ↑ 49 mg/dl (20-40 mg/dl), kreatinin 3.1 mg/dl (0.6-1.5 mg/dl)

- Blood: (October 5th 2017)
  - Routine blood exam: LED ↑
  - Liver function: Normal except uric acid 9.7 (<7)
  - Kidney function: Normal
  - Random Blood Glucose: Normal
  - Electrolyte: corrected

- Medication
  - Amlodipine 10 mg
  - Valsartan 80 mg
  - Clonidine 0.075
  - Ambroxol 1 tab
  - Nebu Nacl 3x
  - Nacl 0.9% + KCL 25 meq
  - Manitol 250cc
  - Manitol 125 cc
  - Omeprazole 40 g
  - Citicolin 500mg
  - Paracetamol 1 gr
  - Ceftriaxone 2 gr

Medical Diagnosis
- Clinical diagnosis: Hemorrhagic Stroke acute phase, day 3, with unstable hemodynamic
- Etiological diagnosis: Intraparenchymal hemorrhage of left temporal lobe with peripheral edema.
- Location diagnosis: Left temporal lobe (putamen, globus pallidum, internal
Rehabilitation diagnosis

**Impairment:** Right hemiparesis, dysarthria

**Disability:** Communication, airway clearance, activity, mobility

**Handicap:** Vokasional

**Discussion**

Patient is a male, 58 years old with uncontrolled hypertension. This patient had ruptured the left middle cerebral artery that supplies cortex and subcortex of left temporal lobe. The rupture caused hemorrhage at left putamen, globus pallidus and internal capsule, and along with its perifocal edema, the space-occupying lesion compressed adjacent structures that caused dysfunction of each structures. Intracerebral hemorrhage accounts for approximately 10% of all cases of stroke and the most common cause is hypertension. Sudden hemorrhage into brain parenchyma is related to both acute elevations in blood pressure and chronic hypertension. It is thought that hemorrhage occurs through rupture of microaneurysms (Charcot-Bouchard aneurysms) that develop in these vessels in hypertensive patients. They are not true aneurysms of the vessel wall but are pockets of extravasated blood or “pseudoaneurysms”, a sign of previous microscopic ruptures within the vascular wall. Lesion is very likely to damage the motoric pathway from primary cortex homunculus to basal ganglia, mosty affect all area (face, upper extremity and lower extremities).

Patient can not talk fluently, he speaks in low volume and slurred speech. His articulation does not improve until six days from onset. He was diagnosed dysarthria because his speech was mumbling and had limited range of movement of tongue, lip, and jaw active movement, although could still move in all directions. Her communication prognosis is good, he will be able to communicate non verbal.

The hemorrhage at the left cortex and subcortex of temporal lobe gives manifestation to contralateral hemiparesis. Based on the CT scan, the hemiparesis may be caused by the disruption of projection fibers from cerebral cortex to internal capsula, putamen and pallidum caused by hemorrhage. The patient has more weakness on upper extremity because the hemorrhage, that is from middle cerebral artery that supplies more lateral aspect of cerebral hemisphere, causes more disruption on higher projection fibers before it reaches basal ganglia level. Therefore, the upper extremity homunculus is more disrupted. His sensory function is still normal.

Patient feeding is not evaluated yet due to acute condition of the patient, he uses NGT and does not permit to feed orally. He will start feeding independently after stable hemodynamic.

In acute setting, we should optimize the cerebral blood oxygen perfusion, so the penumbra area can survive. Althoug it still minimal, but this patient had sputum retention in both lungs that hinders airway clearance and it would reduce cerebral oxygenation. He already got mucolytic, NaCl nebulization and chest therapy to help mobilize the sputum. Good lung ventilation will improve blood gas exchange in lung and then distributed to cerebrum. This patient also has anemia which could hinder oxygen distribution to cerebrum. After 4days of chest therapy, his lung improve, there were no sputum on both lung.

**Conclusion**

In acute setting, we should optimize the cerebral blood oxygen perfusion, so the penumbra area can survive.

After surviving the acute stage, patient has good functional prognosis in activities because he still has the contralateral extremities to help with activities. He still need to improve his compliance to control his chronic hypertension to prevent recurrent stroke. He might be able to walk independently to ambulate. The trunk strength should not be a problem because got bilateral innervations. If he want go back to his old job, communication rehabilitation become essesntial and would be the main focus to improve his articulation.
Bibliography