SUCCESSFUL MANAGEMENT OF RESPIRATORY FAILURE FOLLOWING SNAKEBITE IN GERIATRIC PATIENT

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ABSTRACT

Introduction: Neurotoxicity manifestations following venomous snakebite may lead to life-threatening conditions such as respiratory muscle paralysis leading to respiratory failure and loss of consciousness. Prompt treatments are required.

Case illustration: A-90-year-old woman presented with loss of consciousness and respiratory failure following snakebite. On general examination, a patient was unconscious (Glasgow Coma Score [GCS] 3) with respiratory rate 4-6 rates per minute and frequent apnea period. Her blood pressure was 267/155 mmHg with sinus tachycardia (150 bpm) and low oxygen saturation (50-65%). Early intubation was performed due to respiratory failure. Rapid neurological improvement was seen after snake antivenom and anticholinesterase administration. She was discharged on the fifth day without any neurotoxic sign.

Discussion: The respiratory failure and loss of consciousness were regarded as acute and severe neurotoxic envenoming. Geriatric patient may have reduced respiratory capacity which may further accelerate the respiratory failure. Neurotoxin acted at the pre- and post-synapse neuromuscular junction. Antivenom is the only definitive therapy in envenoming. Trial of anticholinesterase should always be conducted in neurotoxic envenoming. Early mechanical ventilation support should be given in respiratory failure cases.

Conclusion: Antivenom administration and trial of anticholinesterase should be performed in neurotoxicity envenoming. Mechanical ventilation should not be delayed in present of respiratory failure.

Keywords: venomous snakebite, respiratory failure, neurotoxin, snake antivenom

Abstrak

Pendahuluan: Manifestasi neurotoksik dari gigitan ular berbisa dapat menyebabkan keadaan mengancam nyawa seperti paralisis dari otot pernafasan sehingga terjadi gagal nafas dan penurunan kesadaran. Tatalaksana segera sangat diperlukan.

Ilustrasi kasus: Seorang wanita berusia 90 tahun datang dengan penurunan kesadaran dan gagal nafas setelah gigitan ular. Pada pemeriksaan fisik umum, pasien tidak sadar (GCS 3) dengan frekuensi pernafasan 4-6 kali per menit dan periode apneu berulang.Tekanan darah 267/155 mmHg dengan sinus takikardia (150 kali per menit) dan saturasi oksigen yang rendah (50-65%). Intubasi dini dilaukan atas indikasi gagal nafas. Perbaikan neurologis

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dini didapatkan setelah pemberian serum anti bisa ular dan antikolinesterase. Pasien tersebut dipulangkan pada hari kelima tanpa adanya tanda neurotoksik.

Diskusi: Gagal nadas dan penurunan kesadaran termasuk dalam tanda keracunan neurotoksik akut dan berat. Pasien geriatri secara umum telah memiliki penurunan kapasitas respirasi yang dapat mempercepat kondisi gagal nafas. Neurotoksik bekerja pada celah neuromuskuler pre- dan post-sinaps. Serum anti bisa adalah satu-satunya terapi definitif pada kasus keracunan. Uji coba pemberian antikolinesterase harus selalu dilakukan pada kasus keracunan neurotoksin. Bantuan ventilasi mekanik perlu diberikan secara dini pada kasus gagal nafas.

Kesimpulan: Pemberian anti bisa dan uji coba pemberian antikolenesterase sebainya diberikan pada keracunan neurotoksisitas. Pemberian ventilasi mekanik tidak boleh ditunda pada keadaan gagal nafas,

Kata kunci: ular berbisa, gagal nafas, neurotoksin, serum anti bisa ular

INTRODUCTION

Venomous snakebite can cause variable symptoms accord to the snake species and the amount of venom injected.[1] Neurotoxin from snakebite may result acute neuromuscular paralysis. Paralysis of respiratory muscle which leads to respiratory failure is one of the most fatal neurotoxin effects.[2] Prompt treatments such as timely snakebite antivenom and ventilation support administration are mandatory.[1] In this case report, we present a-90-year-old woman with respiratory failure symptom as the neurotoxicity manifestation following snakebite, she managed to survive the critical phase.

CASE ILLUSTRATION

A-90-year-old woman admitted to Emergency Department with chief complaint of shortness of breath after being bitten on her dorsal left foot by a snake since two hours before hospital admission. She was bitten while walking at grass field near her house. She also complained vomiting, ptosis, and speaking difficulty. She had history of uncontrolled hypertension. She was brought to the community health center and given one vial of snake antivenom (BioSave®). On the way to the hospital, approximately 15 minutes before arrived to the hospital, the dyspnea was worsening and her consciousness was deteriorated.

The snake was caught, photographed, and disposed by local people. The snake was described had approximately two-meter length, black skin with eye-like scale pattern at the back (Figure 1). It has two prominent fangs in the front and can rise from the ground and flatten the neck to form hood. The bite mark had two punctures wound on the skin without any ecchymosis or necrotic changes.

Figure 1: The captured snake

On general examination, patient was unconscious (E1V1M1; Glasgow Coma Score [GCS] 3) with respiratory rate 4-6 rates per minute and frequent apnea period leading to respiratory failure. Her blood pressure was 267/155 mmHg with sinus tachycardia (150 bpm) and low oxygen saturation (50-65%). Cardiovascular, lung and abdominal examinations were unremarkable. Neurological examination revealed generalized hypotonic but motoric power could not be assessed, due to her unconsciousness. Pupils were miosis (1mm/1mm) with negative light pupil reflex. Patient was mechanically ventilated because of poor respiratory efforts. Her laboratory results showed leukocytosis (23,010/uL) and blood glucose 238 mg/dL; otherwise, the coagulation, liver and renal function were normal.

We administered additional one vial of snake antivenom (Biosave®) and neostigmine 0,02 mg/kg intramuscularly (IM) with premedication of atropin sulfate 0,6 mg intravenously (i.v.),

ceftriaxone injection 2 gram per day, diltiazem injection, rapid-acting insulin, and ringer lactate solution for maintenance.

One hour after antivenom and neostigmine administration, patient started showing improvement. She regained consciousness and had improvement from GCS 3 to 6 (E3VettM3). The pupils were found 2 mm/2 mm in diameter with slow light reflex. Neostigmine was continued to be given every 3 hours. No additional antivenom was given.

On the second day, her GCS is 10 (E4VettM6), with reactive light pupils reflex (2mm/2mm). Neostigmine administration was stopped. Ventilator mode was changed from controlled mechanical ventilation (CMV) into adaptive support ventilation (ASV). On the third day, she was extubated and transferred to the ward on the fourth day. The patient was discharged on the fifth day. She went home with a stable vital sign and without a sign of neurotoxicity.

DISCUSSION

The respiratory failure and loss of consciousness in our patient were regarded as acute and severe neurotoxicity manifestation following snakebite.[1,2] Acute neurotoxicity usually occurred within six hours of bite and may manifest as ptosis (85,7%), ophthalmoplegia (75%), limb weakness (26,8%), respiratory failure (17,9%), and neck muscle weakness (7,1%).[3] The manifestation usually started descending from eye muscle then followed by bulbar, respiratory, and extremities muscles.[2] In our case, the patient had rapid progressive of respiratory muscle paralysis which leads to respiratory failure and deterioration of consciousness following one hour after getting bitten. Our very old aged patient may contribute to this condition as the capacity of respiratory and cardiovascular function may have declined.[4] Neurotoxicity manifestations were caused by the blockage of neuromuscular junction in the pre-synapse and post-synapse.[1,2] The blockage in pre-synapse is initiated with motor neuron denervation and degeneration by beta-neurotoxin that consists of phospholipase A2 enzyme.[1] The neurotoxins bind irreversibly and the recovery of damaged neuron depends on the ability of neuron regeneration.[1,2] Other neurotoxin is alpha-neurotoxin that acts as competitive inhibitor in post-synapse and bind acetylcholine receptor in nicotinic type.[1,5]

Neurotoxicity envenoming can be caused by Elapidae family such as *Bungarus spp*. and *Naja spp*. also the Viperidae family.[1] Venomous snakes usually have characteristic of triangular heads, heat sensing pits, elliptical pupil, single row of subcaudal scales and typical bite mark with only one or two fangs punctures.[6] The bite mark on dorsal left foot in this patient has two fangs punctures on the skin which indicate a venomous snake bite mark. In our case, from the snake characteristics described, we suspect of cobra species (*Naja sp*.) bite in this patient.

Antivenom administration is the only effective treatment for toxin neutralization in circulation.[1,6] Early administration should be given because antivenom may not effective after neurotoxin binds the neuron.[1,2] The patient receive antivenom within two hours after the bites. The only antivenom available in Indonesia is Biosave® and only cover for venom that comes from *Naja sputatrix*, *Bungarus fasciatus*, and *Agkistrodon rhodostoma*.[6,7] Our patient showed rapid neurologic improvement following antivenom administration. The initial dosage was two vials (10 ml). Another dose may be administered six hours apart from the initial dose. Antivenom can be given every 24 hours if envenoming symptoms still remain with maximal dose is 80-100 ml.[1,6] In this case, we did not administer additional snake antivenom because there was significant clinical improvement after the initial dose.

According to the World Health Organization Snakebite guideline, a trial of anticholinesterase should always be given in the presence of neurotoxicity. Anticholinesterases are beneficial against the postsynaptic toxins that blockage of neuromuscular junction.[1] Neostigmine dose is 0,02 mg/kg IM for an adult with atropine for premedication.[1] If there is a neurologic improvement then neostigmine can be given every 1-3 hours until maximum dose 10 mg/24 hours.[1,6] In our case, the patient showed neurological improvement after neostigmine and antivenom administration.

Respiratory failure due to neurotoxin is the

most common causes of mortality in snakebite cases.[1,3,6] Early ventilation support is important and should be given whenever sign of respiratory failure and bulbar involvement presence. Mechanical ventilation is usually uncomplicated and temporary in neurotoxic snakebite cases.[8]

CONCLUSION

Neurotoxin from snakebite may result a life threatening condition. Antivenom, which is the definitive therapy for snake envenoming, should be administered timely. Trial of anticholinesterase should always be performed in neurotoxic envenoming cases. Early ventilation support should not be delayed in respiratory failure due to neurotoxic.

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