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Management of Acute Hypertensive Emergencies on CKD

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ABSTRACT

Research Objectives to determine the factors that influence the success of drug-sensitive pulmonary TB patients at the Rimba Jaya Public Health Center, Merauke Regency. Research Methods: This type of research is analytic observational with a cross sectional design. Results: most of the respondents aged between 20-35 years amounted to 29 people (58%). The gender of the majority of women amounted to 28 people (56%). Most of the education is SMA with 28 people (56%). Most of the occupations are college students, students and not working totaling 19 people (38%). The knowledge of the respondents is good amounting to 32 people (64%). Most of the economic status is moderate amounting to 28 people (56%). Most of the access to treatment is close, amounting to 48 people (96%). Most of the PMO types are families with 46 people (92%). Most of the PMO roles were good, amounting to 44 people (88%). Most of the drug sensitive patients were drug sensitive at 6 months totaling 45 people (90%). There are 3 factors that influence the success of Pulmonary TB Treatment at the Rimba Jaya Health Center, namely education, economic status and access to treatment. Meanwhile, those that do not affect the success of drug-sensitive pulmonary TB treatment are age, gender, occupation, type of PMO, role of PMO, knowledge.

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INTRODUCTION

Chronic Kidney Disease / CKD is kidney damage that occurs for more than 3 months, based on pathological abnormalities or signs of kidney damage such as proteinuria. If there are no signs of kidney damage, the diagnosis of chronic kidney disease is made if the glomerular filtration rate is less than 60 ml/min/1.73m² (Becker et al. , 2012; Vaidya, & Aeddula, 2021; Boyd et al, 2008) .

In patients with CKD, staging is determined by the value of the glomerular filtration rate, i.e. a higher stage indicates a lower value of the glomerular filtration rate. The classification divides chronic CKD into five stages (Guo et al, 2014; Jonsson et al, 2020) . Stage 1 is kidney damage with normal kidney function, stage 2 kidney damage with mild decline in kidney function, stage 3 kidney damage with moderate decline in kidney function, stage 4 kidney damage with severe decline in kidney function, and stage 5 is kidney failure (Becker et al. , 2012) .

In grades 1 and 2, a decrease in GFR alone can be used as a basis for diagnosis, because GFR is usually normal or only slightly abnormal (Kraut, & Kurtz, 2005; Webster et al, 2017). In some cases, the presence of one or more of the following markers of kidney damage can help confirm the diagnosis: Albuminuria (albumin excretion >30 mg/24 hours or albumin:creatinine ratio >30 mg/g (>3 mg/mmol), abnormal urine sediment, electrolyte disturbances due to renal tubular disorders, histological abnormalities, structural abnormalities detected on imaging, and a history of renal transplantation from the same cause. Patients with stage 1-3 are generally asymptomatic. Clinical symptoms are not seen until entering stage 4-5 (GFR < 30 mL/min/1.73 m^2) where metabolic/endocrine disturbances, water and electrolyte balance disorders are clinical manifestations. Signs of metabolic acidosis at stage 5 include protein-energy malnutrition , loss of lean body mass and muscle weakness . Signs of altered kidney function overcome salt and water levels at stage 5 include peripheral edema, pulmonary edema, and hypertension.Anemia in chronic kidney disease is associated with Fatigue, reduced ability to function, impaired cognitive function and immunity, reduced quality of life, early onset of heart failure or progression to more severe heart failure, and increased cardiovascular mortality (Becker et al. , 2012; Soi, & Yee, 2017) .

Another manifestation is the development of uremia in end-stage renal disease, often occurs in patients with inadequate dialysis, including pericarditis (can be a complication of cardiac tamponade, can cause death if not detected); encephalopathy (can lead to coma and death); peripheral neuropathy; restless leg syndrome ; gastrointestinal symptoms (Anorexia, nausea, vomiting, diarrhea); skin manifestations (dry skin, pruritus, ecchymosis); fatigue, increased drowsiness; malnutrition and failure to thrive ; erectile dysfunction, decreased libido, and amenorrhea; Platelet dysfunction with consequent easy bleeding. Check for depressive symptoms in elderly patients with chronic kidney disease; 45% of patients on initial dialysis report depressive symptoms, sometimes with somatic symptoms.

Laboratory tests include complete blood count (CBC), basic metabolic panel, urinalysis, serum albumin levels, and lipid profile (Castro & Gourley, 2010). Evidence of renal-bone disease was obtained from serum calcium, 25-hydroxyvitamin D, alkaline phosphatase, and parathyroid hormone (PTH) levels. Imaging can be done to establish the diagnosis: renal ultrasound, retrograde pyelography, CT-scan, MRI, Renal radionuclide scanning.

Early diagnosis and management of the underlying disease is very important to slow the progression of the disease. Treatment that must be done includes slowing the progression of kidney disease, diagnosis and treatment of disease pathological manifestations (anemia, hyperphosphatemia, hypocalcemia, hyperparathyroidism, fluid overload, metabolic acidosis, uremia), long-term plans for renal replacement therapy (severe metabolic acidosis, hyperkalemia, pericarditis, encephalopathy, intractable fluid overload, malnutrition and failure to thrive , peripheral neuropathy, persistent gastrointestinal disturbances).

To prevent worsening of CKD, it is necessary to control blood pressure individually based on age, the presence or absence of heart disease and other comorbidities. Administration of antihypertensive drug regimens in the elderly must pay attention to age, comorbidities as well as other drugs that are being used. In patients with CKD, with blood pressure 140/90 mmHg and urinary albumin excretion < 30 mg/24 hours, antihypertensive drugs should be given to maintain blood pressure 140/90 mmHg. In patients with CKD, with blood pressure 130/80 mmHg and urinary albumin excretion >30 mg/24 hours, antihypertensive drugs should be given to maintain blood pressure 130/80 mmHg. The use of ACE-i is recommended when urinary albumin excretion is 30-300 mg/24 hours and is recommended when urinary albumin excretion is >30 mg/24 hours. Secondary hypertension accounts for 5-10% of hypertension cases. Included in this group are hypertension due to kidney disease (renal hypertension), endocrine hypertension, central nervous disorders, drugs and others. Renal hypertension may be reno- vascular hypertension, eg in renal artery stenosis, intrarenal cutaneous vasculature; and hypertension due to renal parenchymal lesions as in chronic glomerulonephritis (Becker et al. , 2012;) (Yoewono, Saputri and Maheasy, 2020) .

RESEARCH METHOD

This research is a case study research. This research was conducted for three months. The following is the case examined by the author. A 36-year-old man who works as an entrepreneur comes to the ER complaining of severe shortness of breath since 1 week before being admitted to the hospital. The tightness is felt to get worse during activity and the patient is more comfortable in a sitting position. Shortness of breath is not accompanied by cough or fever, nor is it accompanied by chest pain and complaints of coughing up blood . The patient also complains of swelling all over the body, orange urine, a lot of it, and does not look foamy, and there is a headache. but not pulsating. Complaints in the form of seizures, projectile vomiting, decreased consciousness, blurred vision, and weakness on one side were denied by the patient, bowel movements were within normal limits .

3 months of SMRS, the patient went to the Police Hospital because he often felt short of breath, especially during activities accompanied by complaints of swelling in the body. At that time the patient was admitted to the ICU for 3 weeks, and it was said the patient suffered from kidney failure and hypertension, but did not require dialysis . The patient's complaints of swelling in the body were initially felt by the patient since ± 4 months of SMRS, initially in both legs which then gradually spread up the stomach to the front, and was accompanied by fatigue during activities. On physical examination, the patient was aware of compost mentis, good general condition, weight 89 kg, height 170 cm with BMI 30.79 kg/m2 (obese class 1). Blood pressure 190/130 mmHg, pulse 132 x/minute, respiratory rate 26 x/minute, eyes looked pale conjunctiva +/+ with periorbital edema +/+. On auscultation of the lungs, vesicles were found to decrease at the bilateral lung bases, bilateral smooth wet rhonchi at the lung bases, no wheezing. On abdominal examination, the abdomen appeared distended, with positive shifting dullness. The extremities showed pitting edema in all four extremities.

On April 13 2017 laboratory examination, Hb 8.13 g/dl, Ht 22.2%, electrolyte sodium decreased by 131, urea 87.2 mg/dL. Creatinine 2.02 mg/dL, albumin 1.58 g/dL, MCH 32.7 pg, MCHC 56.6 g/dL, HDL 39 mg/dL. Urine analysis showed cloudy yellow urine, 3+ occult blood, 3.2 urobilinogen, 2+ protein, 1+ sediment, many erythrocytes, 1-2 hyaline cylinders, 1-2 leukocytes. Blood calcium examination showed 7.7 mg/dL. On X-ray examination showed cardiomegaly with signs of pulmonary congestion, possibly accompanied by pneumonia which could not be ruled out. Bilateral pleural effusion. Abdominal ultrasound examination revealed bilateral chronic parenchymal kidney disease. ascites. Echocardiography examination revealed an ejection fraction of 50%. Funduscopic examination revealed hypertensive retinopathy in both eyes. CT Brain examination on April 14, 2017 showed multiple infarcts in the right lateral periventricular, right basal ganglia and right posterior crus internal capsule. Lacunar basal ganglia and left thalamus infarct. left maxillary sinusitis. On the examination of Anti Nuclear Antibody (ANA) on April 19, 2017, the results were positive. The list of problems in this patient includes the presence of a cute respiratory distress syndrome , hypertension emergency , a cute on CKD , anasarca edema , hypoalbuminemia , normochromic normocytic anemia and hypertensive retinopathy grade III ODS . While in the treatment room the patient received therapy: Drip Furosemide 15 mg/hour ; Drip nitroglycerin 20 mcg/hour ; Amlodipine 1 x 10 mg ; Vitamin B 12 3 x 50 mg ; Ascardia 1 x 80 mg ; Simvastatin 1 x 20 mg ; Nicardipine titration up from 1 mcg/kg ; Ramipril 1 x 10 mg ; Folic acid 1 x 15 mg ; Clonidine 3 x 0.15 mg ; Vitamin B 6 2 x 10 mg ; Albumin 20% 100 ml . The following is a follow-up of the patient's treatment while in the treatment room.

RESULTS AND DISCUSSIONS

The patient was diagnosed with chronic glomerulonephritis, from the patient's anamnesis he complained of swelling all over his body since ± 4 months ago which then gradually spread to the abdomen to the front. It is said that the BAK is orange in color, in large quantities, and does not appear to be foamy. Based on physical examination, →anasarca edema appears; blood pressure

190/130 mmHg; pulse rate 132 x/minute, regular; respiratory rate 26 x/minute; pale conjunctival eyes +/+, periorbital edema; Lung breath sounds decreased eskiluer at bilateral lung bases, bilateral soft wet rhonchi at lung bases; distended abdomen , positive shifting dullness ; warm extremities , pitting edema in all four extremities . From laboratory examination, it was found that serum albumin: 1.75 g/dL, ANA: positive, quantitative urine protein: 5575 mg/24 hours, C3: 25, C4: 4 and anti-dsDNA: 0.7 IU/ml. Kidney biopsy was not performed because one of the contraindications for kidney biopsy is hypertension, which can increase the occurrence of bleeding (Becker *et al.* , 2012) .

The patient was diagnosed with a hypertensive emergency in acute on CKD, where the definition of a hypertensive emergency according to JNC 7 is: a sudden increase in systolic blood pressure >180 mmHg or diastoic > 120 mmHg accompanied by target organ damage. In this patient was found on physical examination →BP: 190/130 mmHg; pale conjunctiva; on investigation, it was →found that Hb: 8.13 g/dL, urea: 87.2 mg/dL, creatinine: 2.02 mg/dL; eGFR : 20ml/min/1.73m ^{2}. Albumin: 1.75 g/dL, urine analysis: urine protein 2+, abdominal ultrasound: Chronic parenchymal kidney disease bilateral; CT brain: multiple infarcts in the right lateral periventricular, right basal ganglia and right posterior crus internal capsule. Lacunar basal ganglia and left thalamus infarct. left maxillary sinusitis. Funduscopy: hypertensive retinopathy in both eyes, chest X-ray showed cardiomegaly, abdominal ultrasound of bilateral chronic parenchymal kidney disease. Definition of *acute on CKD* : patients suffering from CKD with symptoms of acute kidney failure. This patient, based on the eGFR value of 20 mL/min/1.73 M2, →was classified as CKD stage IV and experienced symptoms such as acute kidney failure, namely: reduced urine production, nausea and vomiting, shortness of breath, accumulation of fluid in the body or edema, fatigue. (Becker *et al.* , 2012) Emergency antihypertensive therapy in patients with CKD is a decrease in systolic and diastolic blood pressure 10-15% but not more than 25% of initial blood pressure through parenteral administration of antihypertensive drugs, aiming to lower blood pressure as soon as possible to avoid damage to organs Furthermore. The following are selected drugs that can be adapted to the medical conditions and target organs involved; based on the management of hypertensive emergencies by the Department of Emergency Medicine Pennsylvania Hospital 2008 (Pollack, Rees and Chang, 2008) .

Table 1. Anti-hypertensive agents suggested for specific hypertensive emergency syndromes [2,17-18]

Syndrome	Suggested anti-hypertensive agents
Aortic Dissection	- Nitroprusside, often in combination with esmolol or labetalol - Nicardipine or clevidipine with esmolol or labetalol
Acute Pulmonary Edema	- Nitroglycerin may reduce pressure - Fenoldopam if renally impaired - Nicardipine - Clevidipine
Acute Coronary Syndrome	- ß-blocker - Nitroglycerin - Clevidipine
Hypertensive Emergency with Acute or Chronic Renal Failure	- Labetalol - Nicardipine - Fenoldopam - Clevidipine
Eclampsia	- Labetalol - Nicardipine - Hydralazine *(all in conjunction with magnesium sulfate)*
Acute Ischemic Stroke or ICH *(If expert guidance deems BP control necessary)*	- Nicardipine - Labetalol - Clevidipine
Hypertensive Encephalopathy	- Labetalol - Esmolol - Nicardipine - Fenoldopam if renally impaired - Nitroprusside *(only if necessary)* - Clevidipine

Table 2. Parenteral Agents Used for Hypertensive Emergencies [1,10,11]

DRUG	CLASS/MECHANISM	USUAL DOSE	ONSET	DURATION	ADVANTAGES	DISADVANTAGES/ADVERSE EFFECTS	COMMON USES	ED UTILITY + to ++++
Sodium nitroprusside	Direct arterial and venous vasodilator via cGMP.	0.25–10 µg/kg/min. Avg. effective dose 3 µg/kg/min.	1-2 min.	3-4 minutes after infusion stopped.	Large amount of experience with use.	- Nausea, vomiting, muscle twitching, diaphoresis. - Cyanide toxicity, especially with renal insufficiency and prolonged infusions (>48 hours). - Inactivated by light, requires light-protected delivery system. - Usually requires invasive intra-arterial blood pressure monitoring. - Use cautiously with ↑ ICP as can worsen. - Use cautiously in ACS as can cause coronary steal.	Has been used in all syndromes of hypertensive emergencies (HE).	+++
Nitroglycerin	Direct venous vasodilator.	5 µg/kg/min to max 100 µg/kg/min.	2-5 min.	5-10 minutes.	- Dilates coronary vessels.	- Ineffective arterial vasodilator. - HA, hypotension, tachycardia.	Not used for most HE, reserved for cardiac ischemia and cardiogenic pulmonary edema.	+
Fenoldopam	Peripheral, dopamine-1 receptor agonist, causes vasodilation esp. in renal, cardiac, splanchnic beds.	0.1 µg/kg/min. to max 1.6 µg/kg/min. Titrate in 0.05–0.1 µg/kg/min increments.	10 min.; Max effect in 30 min.	1 hour after stopping.	- Increases renal blood flow, improving CrCl esp. in setting of impaired renal function. - Used without invasive BP monitoring.	- Reflex tachycardia. - HA, dizziness, flushing, nausea - Worsening angina - A-fib - Tachyphylaxis after 48 hrs. - Contraindicated in glaucoma as causes dose-related increase in intraocular pressure.	Especially useful in HE syndromes complicated by renal insufficiency or failure.	++
Nicardipine	Dihydropyridine Ca-channel blocker. Vasodilator.	5 mg/hr can ↑ by 2.5 mg/hr to max 15 mg/hr.	Within 10 min.	2-6 hours after stopping.	- Dilates coronary vessels, can use with known CAD. - Used without invasive BP monitoring.	- HA, flushing, dizziness, hypotension, digital dysesthesias. - Abrupt withdrawal can cause or worsen angina or hypertension. - Metabolized in liver, use with caution in cirrhotics.	Has been used extensively in post-op hypertension, especially post CT surgery.	+++

Figure 1. Management of Hypertensive Emergencies by the Department of Emergency Medicine Pennsylvania Hospital 2008

Based on the figure above, the selected emergency antihypertensive drugs are: nitroglycerin and nicardipine. Administration of initial NTG →, 5 mcg/kg/min IV, increase by 5 mcg/kg/min IV, every 2-5 minutes up to 20 mcg/kg/min until clinical response if no response at 20 mcg/kg/min , increase the dose by 10 mcg/kg/min every 2-5 minutes to 100 mcg/kg/min. If a clinical response is obtained (the decrease in BP is 25% from the initial MAP value), then the dose of nitroglycerin is tapered off by decreasing the dose slowly and gradually over 24 hours by 5-10 mcg/kg/min until a minimum dose of 5 mcg/kg/min is obtained. min within 24 hours, then IV nitroglycerin is discontinued. Because the side effect of stopping nitroglycerin suddenly can cause rebound hypertension (Yoewono, Saputri and Mahcepat, 2020) (Becker *et al.* , 2012) (Pollack, Rees and Chang, 2008) .

Along with the administration of emergency antihypertensive drugs intravenously, it can also be given orally as a BP *maintenance* , the recommended drug classes according to the KDIGO *Clinical Practice Guideline for the Management of Blood Pressure in Chronic Kidney Disease* 2012 are ACE inhibitors and furosemide. Administration of amlodipine along with nicardipine as a treatment for hypertensive emergencies because amlodipine is long acting so that it supports the work of nicardipine, with peak levels only being reached within 6-9 hours, half-life within 35-48 hours (Huang *et al.* , 2019) .

Administration of clonidine as an oral emergency antihypertensive drug because of clonidine as a second or third drug when BP reduction with a diuretic is not optimal, to replace other adrenergic blockers in a 3-drug combination with a diuretic and vasodilator, and for some hypertensive emergencies. (Yoewono, Saputri and Maheasy, 2020) . After the hypertensive emergency resolved (at D-16, BP: 150/90mmHg) →the parenteral antihypertensive drug was slowly replaced with an oral antihypertensive drug. The administrations given were Ni fedipine, Clonidine and Ramipril (Sunaryo *et al.* , 2019) .

According to the JNC 7 algorithm, hypertensive patients with a *compelling indication* of CKD are the preferred choice of an ACE inhibitor or ARB. Ramipril belongs to the class of ACE inhibitors. The administration of nifedipine is useful for treating hypertension with pulmonary edema. Management of anasarca edema ec. Chronic glomerulonephritis: this patient received antihypertensive therapy, furosemide, spironolactone, albumin, and dialysis was performed three times a week during the treatment. However, because refractory edema still occurs (patients are resistant to loop diuretic therapy in treating anasarca edema), the dose of furosemide given 2 x 40 mg was increased to 160 mg to 200 mg intravenously slowly, in order to avoid the side effects of

tinnitus. Once the edema resolves, IV furosemide can be switched to oral furosemide. Albumin therapy together with furosemide according to several studies can increase the delivery of diuretics to the kidneys while keeping furosemide in the vascular space. In a preliminary report, this approach led to a substantial increase in sodium excretion in some patients. The administration of 60 mg furosemide infused in 200 ml of 20% albumin solution can increase sodium excretion compared to single furosemide administration. (Hsu and Hsu, 2016) .

Administration of spironolactone→ Hypertension is more common in heart failure patients with good ejection fraction function (50%), so spironolactone as a restrictive therapy for heart failure can be given. In addition, spironolactone is also synergistic when given together with furosemide, and can also reduce the hypokalemic effect of furosemide. (Smf, Vascular and Medicine, 2015) . Administration of vitamins B6 and B12 and folic acid in these patients; based on the 2013 AHA/ASA guidelines, therapy for cerebral infarction can be given neuroprotective therapy. Vitamins B6 and B12 are neuroprotective for conditions of cerebral infarction. Vitamin B12 and folic acid are also given to patients with CKD, to avoid folic acid deficiency and megaloblastic anemia due to CKD. (Roccella, 2005) .

CONCLUSION

Management of anasarca edema in patients with refractory edema by administering furosemide iv up to the maximum dose; ie from a dose of 2 x 40 mg increased to 160 mg to 200 mg intravenously, the dose is increased slowly to avoid the side effect of tinnitus. After the edema resolves, the intravenous administration of furosemide can be switched to oral .

References

Becker, GJ *et al.* (2012) 'Kidney disease: Improving global outcomes (KDIGO) blood pressure work group. KDIGO clinical practice guidelines for the management of blood pressure in chronic kidney disease', *Kidney International Supplements* , 2(5), pp. 337–414. doi: 10.1038/kisup.2012.46.

Boyd, L. M., Langston, C., Thompson, K., Zivin, K., & Imanishi, M. (2008). Survival in cats with naturally occurring chronic kidney disease (2000–2002). *Journal of Veterinary Internal Medicine*, *22*(5), 1111-1117.

Castro, C., & Gourley, M. (2010). Diagnostic testing and interpretation of tests for autoimmunity. *Journal of Allergy and Clinical Immunology*, *125*(2), S238-S247.

Hsu, RK and Hsu, C. yuan (2016) 'The Role of Acute Kidney Injury in Chronic Kidney Disease', *Seminars in Nephrology* , 36(4), pp. 283–292. doi: 10.1016/j.semnephrol.2016.05.005.

Huang, QF *et al.* (2019) 'A randomized controlled trial on the blood pressure– lowering effect of amlodipine and nifedipine-GITS in sustained hypertension', *Journal of Clinical Hypertension* , 21(5), pp. 648–657. doi: 10.1111/jch.13543.

Guo, X., Qin, Y., Zheng, K., Gong, M., Wu, J., Shou, W., ... & Qiu, L. (2014). Improved glomerular filtration rate estimation using new equations combined with standardized cystatin C and creatinine in Chinese adult chronic kidney disease patients. *Clinical biochemistry*, *47*(13-14), 1220-1226.

Jonsson, A. J., Lund, S. H., Eriksen, B. O., Palsson, R., & Indridason, O. S. (2020). The prevalence of chronic kidney disease in Iceland according to KDIGO criteria and age-adapted estimated glomerular filtration rate thresholds. *Kidney International*, *98*(5), 1286-1295.

Kraut, J. A., & Kurtz, I. (2005). Metabolic acidosis of CKD: diagnosis, clinical characteristics, and treatment. *American journal of kidney diseases*, *45*(6), 978-993.

Pollack, C. V, Rees, CJ and Chang, AM (2008) 'Emergency Department Evaluation and Management of HYPERTENSIVE EMERGENCES : Objectives ' :, 3 (October).

Roccella, E. J. (2005). The National High Blood Pressure Education Program. In *Hypertension* (pp. 7-15). WB Saunders.

Smf, B., Vascular, K. and Medicine, F. (2015) 'Handling the Hypertension Crisis', *Idea Nursing Journal* , 6(3), pp. 61–67.

Soi, V., & Yee, J. (2017). Sodium homeostasis in chronic kidney disease. *Advances in chronic kidney disease*, *24*(5), 325-331.

Sunaryo, EYAB *et al.* (2019) 'Overview of body mass index, blood pressure, and blood sugar levels while in Dusun Jaten, Sinduadi, Mlati, Sleman, DIY', *Journal of Community Empowerment for Health* , 1(2), p. 115. doi: 10.22146/jcoemph.41375.

Webster, A. C., Nagler, E. V., Morton, R. L., & Masson, P. (2017). Chronic kidney disease. *The lancet*, *389*(10075), 1238-1252.

Yoewono, EP, Saputri, R. and Mahmudah, R. (2020) 'Effectiveness of Antihypertensive Therapy in Cases of Hypertension Crisis in the Emergency Installation of Ulin Hospital Banjarmasin', *Journal of Pharmaceutical Care and Science* , 1(1), pp. 19–27.

Vaidya, S. R., & Aeddula, N. R. (2021). Chronic renal failure. In *StatPearls [Internet]*. StatPearls Publishing.