

Role of the Plasma Brain Natriuretic Peptide in Differentiating Patients with Congestive Heart Failure from Other Diseases

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Abstract

Background : Heart failure (HF) is primarily a disease of the elderly. The incidence of congestive heart failure (CHF) in Thailand has been increasing during the last 10 years. Unlike other diseases, physicians have only rough patients' symptoms and physical findings to guide the adequacy of treatment. Recently, there has been evidence of the role of brain natriuretic peptide (BNP) and its use in HF concerning diagnosis, prognosis, and treatment follow-up. The purpose of this study was to determine the sensitivity and specificity of N-terminal part of brain natriuretic peptide plasma level (NT-proBNP) in the diagnosis of HF in Thai patients who presented with dyspnea.

Method : The design was a cross sectional study. The authors enrolled 50 consecutive patients from the Respiratory Unit with dyspnea from chronic obstructive pulmonary disease (COPD), asthma, or anxiety. The cardiovascular cause of dyspnea such as pulmonary emboli and poor left ventricular ejection fraction (LVEF) were excluded. Forty eight consecutive patients with evidence of HF who presented to the Cardiac Center with a history of dyspnea on exertion were assigned as cases. Five milliliters of venous blood samples were taken and sent together with 200 samples from a normal healthy population from the check up department for NT-proBNP measurement.

Results : In case and control groups, there were no statistical significances in sex (males 68.8% vs females 52.0%, $p > 0.05$) and age (63.3 ± 14.9 vs 55.6 ± 16.9 ; $p > 0.05$). The mean left ventricular ejection fraction in the case group was 32.4 ± 9.7 per cent. There was significant difference between value of NT-proBNP in the control group ($386 \pm 1,041$ pg/ml) and in the case group ($8,912 \pm 12,525$ pg/ml, $p < 0.001$). To diagnose HF in patients who presented with dyspnea using the cut-off value of NT-proBNP at > 150 pg/ml in patients with dyspnea the sensitivity was 96 per cent, and the specificity of 72 per cent; at > 200 pg/ml the sensitivity was 96 per cent and the specificity was 80 per cent and at > 300 pg/ml the sensitivity was 94 per cent and specificity of 82 per cent. Plasma level of NT-proBNP increased significantly with increasing New York Heart Association (NYHA) functional class (class II: $1,107 \pm 1,091$ pg/ml; class III: $5,097 \pm 4,201$ pg/ml, class IV: $19,389 \pm 15,966$ pg/ml $p < 0.01$). There was no significant difference of plasma NT-proBNP levels in patients with ischemic ($8,586 \pm 11,601$ pg/ml; $n = 35$) and those with non ischemic cardiomyopathy ($9,789 \pm 15,229$ pg/ml; $n = 13$). Plasma NT-proBNP was associated with neck vein distension ($p < 0.05$) but there was no

significant association with S3, paroxysmal nocturnal dyspnea, rales, cardiomegaly, acute pulmonary edema, serum sodium ($r = 0.22$), ejection fraction ($r = -0.18$) and subsequent hospital death ($p > 0.05$).

Conclusion : Measurement of plasma NT-proBNP proved to be a useful diagnostic test in differentiating HF from other causes in patients who presented with dyspnea.

Key word : Congestive Heart Failure, NT-proBNP, Sensitivity, Specificity, Predictive Value

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Congestive heart failure (CHF) is one of the major causes of morbidity and mortality in Thailand. It is primarily a disease of the elderly. In general practice heart failure is commonly misdiagnosed(1,2). The symptoms are non-specific and the clinical signs, although reasonably specific, are not at all sensitive. Consequently, even experienced physicians disagree on the diagnosis in individual cases, especially when the heart failure is mild(3). Two studies in primary care have shown that many patients with breathlessness thought to be mild heart failure have been misdiagnosed(1,2). The simplest confirmation of the diagnosis is provided by echocardiography, which is as reliable as other diagnostic methods such as radio-nuclide angiography(4). However it is not readily available to general practitioners and precise measurement of left ventricular function is possible in only 58 per cent of patients because of obesity or airway diseases(5). Recently, measurement of plasma brain natriuretic peptide (BNP) levels has been suggested as a method of screening for left ventricular (LV) dysfunction(6,7). ProBNP comprising 108 amino acids is secreted mainly by the ventricle and in this process is cleaved into physiologically active BNP (77-108) and the N terminal fragment NT-proBNP (1-76). In the pathophysiology of CHF, BNP participates in adaptive responses to hemodynamic alterations of heart failure. The increased BNP level in patients with LV dysfunction has generated considerable interest

in its diagnostic and prognostic properties in CHF (8,9). Previous studies showed that BNP was more accurate in diagnosing CHF than atrial natriuretic peptide (ANP) and N-terminal atrial natriuretic peptide (NT-ANP); which, being subject to more acute regulation(10,11) and more rapid degradation(12,13). The latter two substances are more likely to fluctuate than BNP. Indeed plasma N-terminal brain natriuretic peptide (NT-ANP) increases to a much greater extent than ANP because of lower renal clearance(14). Thus, the purpose of this study was to determine the utility of NT-proBNP level in the diagnosis of CHF for Thai patients who presented with dyspnea.

MATERIAL AND METHOD

Ethical approval for this study was granted by the hospital ethical committee. The authors enrolled 48 consecutive patients with a diagnosis of CHF according to Framingham criteria (by two cardiologists) from Bangkok Heart Institute and 50 consecutive patients from the Respiratory Unit who presented with dyspnea and diagnosis of asthma or chronic obstructive airway disease or anxiety. Those respiratory patients with underlying cardiovascular disease such as pulmonary emboli, cor pulmonale and poor left ventricular function were excluded. The blood samples of those 98 patients were analyzed for NT-proBNP. The laboratory staff was blinded for clinical status of the patients and controls.

Measurement of plasma NT-proBNP

Five millimeters of venous blood samples were taken and centrifuged within 60 min. The plasma fraction was stored at -20°C until analysis. Plasma NT-proBNP concentration was measured using the Elecsys proBNP reagent kit. Elecsys proBNP contains polyclonal antibodies with recognized epitopes located in the N terminal part (1-76) of proBNP (1-108). In the laboratory, the interassay coefficient of variation within run and between run less than 5 per cent, cross reactivity for other substances e.g. ANP, NT-proANP, BNP, CNP, adrenomedullin, etc. were specified by the manufacturer as < 0.001 per cent.

Statistical analysis

All data were presented as the mean value \pm SD or percentage as appropriate. Chi-square test was used for comparison of categorical variables; continuous variables were compared by using independent Student *t*-test or ANOVA. Post-hoc testing was performed using Scheffe or Tamhane method where appropriate. The relationship between the two continuous variables was assessed by calculation of Pearson's correlation coefficient. Sensitivity and specificity was calculated by using ROC curve. A two tail *p*-value < 0.05 was considered statistically significant.

RESULTS

Demographic data and clinical profile of the CHF patients are shown in Table 1 and 2. The mean value of left ventricular ejection fraction in CHF group was 32.4 ± 9.7 per cent. The etiology by ischemic

heart failure was 35 of 48 (72.9%). The New York Heart Association of functional class in the CHF groups divided into functional class 2 was 15/48 (31.3%), class 3 was 16/48 (33.3%), and class 4 was 17/48 (35.4%). The mean value of NT-proBNP in control was $386 \pm 1,041$ pg/ml, and in case was $8,912 \pm 12,525$ pg/ml (*p* < 0.001) (Table 2, Fig. 1).

The ability of plasma NT-proBNP levels to identify the risk of a clinical event in the CHF and control groups was assessed by comparison of receiver-operating characteristic curves (ROC) (Fig. 2). When the authors used the cut-off value of NT-proBNP at > 150 pg/ml in patients with dyspnea NT-proBNP was able to discriminate patients with a sensitivity of 96 per cent, the specificity was 72 per cent, the positive predictive value was 77 per cent with a negative predictive value of 95 per cent at > 200 pg/ml the sensitivity was 96 per cent, the specificity was 80 per cent, the positive predictive value was 82 per cent with a negative predictive value of 95 per cent and when using the cut-off value > 300 pg/ml the sensitivity was 94 per cent, specificity was 82 per cent, positive predictive value was 83 per cent with a negative predictive value of 93 per cent in the diagnosis of CHF (*p* < 0.0001). As a screening tool the level of NT-proBNP > 200 pg/ml looks appropriate for screening patients with dyspnea.

Plasma level of NT-pro BNP increased significantly with increasing NYHA functional class (class II: $1,107.2 \pm 1,091.2$, class III: $5,097.1 \pm 4,201.0$, class IV: $19,389.2 \pm 15,966.9$ pg/ml; *p* < 0.01). Plasma NT-proBNP levels were not different between patients

Table 1. Demographic data of 48 CHF patients and 50 control subjects.

Clinical profile	CHF patients (N = 48)	%	Control subjects (N = 50)	%	P-value
Sex (male)	33	68.8	26	52	NS
Age (mean \pm SD) yr	63.3 ± 14.9		55.6 ± 16.9		NS

Table 2. The NT-proBNP concentrations of 48 CHF patients and 50 control subjects.

Test	CHF patients (N = 48)	Control subjects (N = 50)	P-value
NT-proBNP (pg/ml)	$8,912 \pm 12,525$	$386 \pm 1,041$	< 0.001

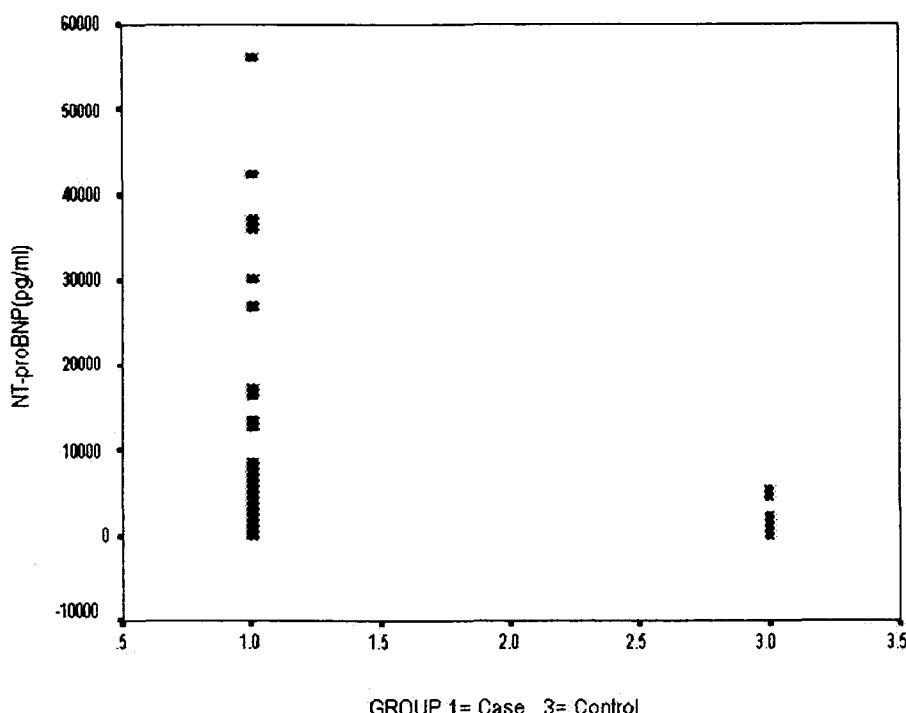


Fig. 1. NT-proBNP level in the two patient groups.

with ischemic ($8,586 \pm 11,601$ pg/ml; $n = 35$) and those with non ischemic cardiomyopathy ($9,789 \pm 15,229$ pg/ml; $n = 13$). Plasma NT-proBNP was associated with neck vein distension ($p < 0.05$) but there was no significant association with S3 gallop, paroxysmal nocturnal dyspnea, rales, cardiomegaly, acute pulmonary edema (Table 3), serum sodium ($r = 0.22$) (Fig. 3A), ejection fraction ($r = -0.18$) (Fig. 3B) and death ($p > 0.05$).

DISCUSSION

In the present study, the authors demonstrated that measurement of plasma NT-proBNP provides a useful screening tool in differentiating patients with CHF from other diseases who presented with symptoms of dyspnea. Because the plasma concentrations of the natriuretic peptides rise as heart failure develops, there is increasing clinical interest in their measurement in the evaluation of left ventricular dysfunction and in prognosis(7). The report by Mc Donagh *et al*(7) and that by Cowie *et al*(6) 4 years ago contributed to the understanding of the part that natriuretic peptides play in the primary diagnosis of heart failure

or in screening for LV dysfunction. Mc Donagh's study is useful in that the population was large and unselected and that it quantified the value of B-type natriuretic peptide (BNP) especially when a high-risk population is targeted. Both studies show that BNP is more accurate than N-terminal atrial natriuretic peptide (NT-ANP) in the detection of left-ventricular dysfunction and the presence of clinical heart failure. BNP is also a sensitive indicator of heart failure as the cause of dyspnea in patients admitted to hospital(15), on admission plasma BNP concentration more accurately reflected the final diagnosis of HF (93% sensitivity and 90% specificity when $BNP \geq 76$ pg/ml) than LVEF or plasma ANP, and in primary care by chosen to give negative predictive values for HF of 98 per cent, when the BNP level was ≥ 76.8 (6). Indeed plasma NT-proBNP which increases to a much greater extent than BNP as the heart fails(16) - proves in other hands to be as good or better than any other non-invasive measure in the assessment of heart function, and prognosis after myocardial infarction(17). To date, BNP, and particularly its aminoterminal portion (NT-proBNP) appears to be the most powerful neurohormonal

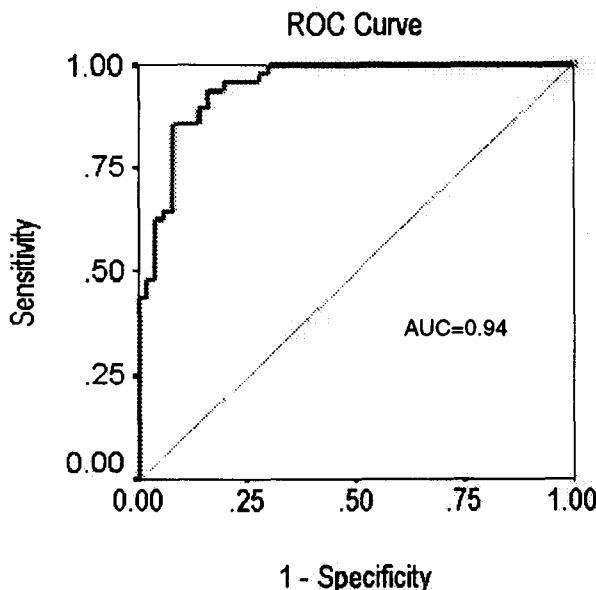


Fig. 2. Receiver-operating characteristics for identification of HF.

Table 3. Correlation of NT-proBNP with symptoms and signs.

Parameter	NT-proBNP Level (pg/ml)	P-value
Neck vein distension (+ or -)	20,095 vs 5,513	0.03
S3 gallop (+ or -)	12,527 vs 6,929	NS
PND (+ or -)	7,946 vs 17,216	NS
Rales (+ or -)	9,679 vs 7,663	NS
Cardiomegaly (+ or -)	8,865 vs 9,981	NS
Acute pulmonary edema (+ or -)	13,328 vs 6,261	NS
Hospital death (+ or -)	17,223 vs 7,945	NS

predictor of left ventricular function and prognosis (15-18), thus, much research interest has focused on the use of these assays in screening, diagnosis, assessment of clinical status, assessment of therapeutic efficacy and prognosis with regard to both morbidity and mortality. Concentration of these peptides are commonly used as surrogate endpoints in monitoring the clinical course or response to therapy in the individual patient or in clinical trials(19-22).

In the present study, patients with high circulating levels of BNP had a substantially higher probability of deterioration of their functional class compared with those patients who needed more extensive risk stratification. Beside their functional class, neck vein distension also correlated with NT-proBNP level.

Although there was no association with ventricular ejection fraction, serum sodium and other symptoms and signs that might be from several reasons such as small sample size, heterogeneous population including acute pulmonary edema from coronary artery disease (CAD), valvular regurgitation, hypertensive heart disease, chronic dyspnea from chronic CHF. Ejection fraction in particular may not accurate because of the timing record. Increase sample or selected homogeneous population and precise measurement such as radionuclide angiography may have a correlation between NT-proBNP with LV dysfunction as several papers have mentioned that natriuretic peptide concentrations reflect the severity of left ventricular hemodynamic dysfunction(23-26) and are of prognostic

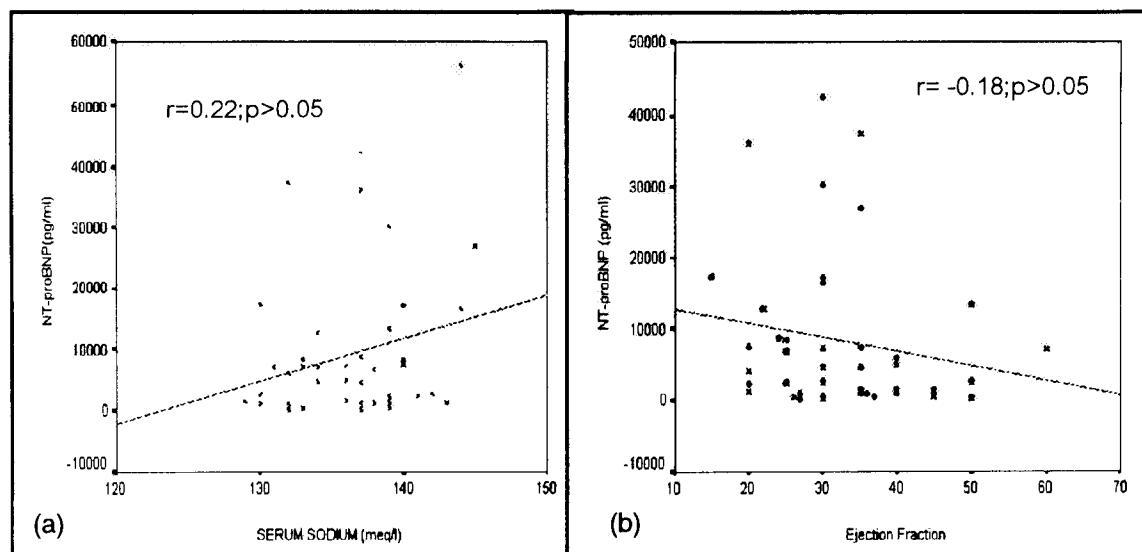


Fig. 3. Correlation of plasma sodium and ejection fraction with plasma NT-proBNP.

value. Apart from LV dysfunction or increased intracardiac pressure and wall tension affect BNP release, other factors may affect cardiac hormone secretion, including chronic renal impairment(10,27), pulmonary arterial hypertension(28) and severe hypoxemia(29, 30). The authors hypothesized that in acute dyspnea of cardiac origin the concentrations of plasma NT-proBNP would exceed those observed in severe primary lung disease and would therefore offer a potentially useful diagnostic aid.

The accuracy of NT-proBNP in this study for the detection of dyspnea from cardiac origin (area under curve 0.94) is similar to that of prostate-specific antigen for detection of the prostate cancer (AUC 0.94)(31) and is superior to that of mammography for

breast cancer (0.85)(32) and Papanicolaou smears for cervical cancer (AUC 0.70)(33).

CHF is a common serious disorder with a recognizable latent phase that has early treatment known to decrease both mortality and progression. Screening of symptomatic or high-risk individuals for left-ventricular systolic dysfunction by measurement of plasma concentrations of natriuretic peptides and echocardiography now offers the possibility of improved accuracy of diagnosis of heart failure and left-ventricular dysfunction and guidance of treatment. Importantly, assay of natriuretic peptide concentrations may be a useful addition to the tests available to general practitioners to help in the decision about referral and further cardiological assessment.

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REFERENCES

1. Remes J, Miettinem H, Reunanan A, Pyorala K. Validity of clinical diagnosis of heart failure in primary heart failure in primary health care. *Eur Heart J* 1991; 12: 315-21.
2. Wheeldon NM, MacDonald TM, Flucker CJ, Mc Kendrick AD, Mc Devitt DG, Struthers AD. Echocardiography in chronic heart failure in the community. *Q J Med* 1993; 86:17-20.
3. Hlatky MA, Fleg JL, Hinton PC, et al. Physician practice in the management of congestive heart failure. *J Am Coll Cardiol* 1986; 8: 966-70.
4. Albin G, Rahko PS. Comparison of echocardiographic quantification of left ventricular ejection fraction to radionuclide angiography in patients with regional wall motion abnormalities. *Am J Cardiol* 1990; 65: 1031-2.
5. Francis CM, Caruana L, Kearney P, et al. Open access echocardiography in management of heart failure in the community. *BMJ* 1995; 310: 634-6.
6. Cowie MR, Struthers AD, Wood DA, et al. Value of natriuretic peptides in assessment of patients with possible new heart failure in primary care. *Lancet* 1997; 350: 1349-53.
7. Mc Donagh TA, Robb SD, Murdoch DR, et al. Biochemical detection of left- ventricular systolic dysfunction. *Lancet* 1998; 351: 9-13.
8. Davidson NC, Naas AA, Hanson JK, Kennedy NS, Coutie WJ, Struthers AD. Comparison of atrial natriuretic peptide, B-type natriuretic peptide and N-terminal proatrial natriuretic peptide as indicators of left ventricular systolic dysfunction. *Am J Cardiol* 1996; 77: 828-31.
9. Yamamoto K, Burnett JC, Jougasaki M, et al. Superiority of brain natriuretic peptide as a hormonal marker of ventricular systolic and diastolic dysfunction and ventricular hypertrophy. *Hypertension* 1996; 28: 988-94.
10. Yandle TG, Richards AM, Glibert A, Fisher S, Holmes S, Espiner EA. Assay of brain natriuretic peptide (BNP) in human plasma: Evidence for high molecular weight BNP as a major plasma component in heart failure. *J Clin Endocrinol Metab* 1993; 76: 832-8.
11. Nicholson S, Richards M, Espiner E, Nicholls G, Yandle T. Atrial and brain natriuretic peptide response to exercise in patients with ischemic heart disease. *Clin Exp Pharmacol Physiol* 1993; 20: 535-40.
12. Ruskoaho H. Atrial natriuretic peptide: Synthesis, release, and metabolism. *Pharmacol Rev* 1992; 44: 479-602.
13. Holmes SJ, Espiner EA, Richards AM, Yandle TG, Frampton C. Renal, endocrine and hemodynamic effects of human brain natriuretic peptide in normal man. *J Clin Endocrinol Metab* 1993; 76: 91-6.
14. Sundsfjord JA, Thibault G, Larochelle P, Cantin M. Identification and plasma concentrations of the N-terminal fragment of proatrial natriuretic factor in man. *J Clin Endocrinol Metab* 1988; 6: 605-10.
15. Davis M, Espiner E, Richards G, et al. Plasma brain natriuretic peptide in assessment of acute dyspnea. *Lancet* 1994; 343: 440-4.
16. Hunt PJ, Richards AM, Nicholls MG, Yandle TG, Doughty RN, Espiner EA. Immunoreactive amino-terminal pro-brain natriuretic peptide (NT-proBNP) a new marker of cardiac impairment. *Clin Endocrinol* 1997; 47: 287-96.
17. Richards AM, Nicholls MG, Yandle TG, et al. Plasma N-terminal pro-brain natriuretic peptide and adrenomedullin: New neurohormonal predictors of left ventricular function and prognosis after myocardial infarction. *Circulation* 1998; 97: 1921-9.
18. Yasue H, Yoshimura M, Sumida H, et al. Localization and mechanism of secretion of B-type natriuretic peptide in comparison with those of A-type natriuretic peptide in normal subjects and patients with heart failure. *Circulation* 1998; 97: 1921-9.
19. Hunt PJ, Espiner EA, Nicholls MG, Richards AM, Yandle TG. The role of the circulation in processing pro-brain natriuretic peptide (proBNP) to amino-terminal BNP and BNP32. *Peptides* 1997; 18: 1475-81.
20. Richards AM, Nicholls MG, Yandle TG, et al. Neuroendocrine prediction of left ventricular function and heart failure after acute myocardial infarction. The Christchurch Cardioendocrine Research group. *Heart* 1999; 81: 114-20.
21. Kenneth D. Commentary, Natriuretic peptides in detection of heart failure. *Lancet* 1998; 351: 4.
22. Koglin J, Pehlivanli S, Schwaiblmair M, Vogeser M, Cremor P, von Scheidt W. Role of brain natriuretic peptide in risk stratification of patients with congestive heart failure. *J Am Coll Cardiol* 2001; 38: 1934-41.
23. Troughton RW, Frampton CM, Yandle TG, Espiner EA, Nicholls MG, Richards AM. Treatment of heart failure guided by plasma aminoterminal brain natriuretic peptide (NT-BNP) concentrations. *Lancet* 2000; 355: 1126-30.
24. Kinnunen P, Vuolteenaho O, Ruskoaho H. Mechanism of atrial and brain natriuretic peptide release from rat ventricular myocardium: effect of stretching. *Endocrinology* 1993; 132: 1961-70.
25. Richards AM, Crozier TG, Yandle TG, Espiner EA, Ikram H, Nicholls MG. Brain natriuretic factor: regional plasma concentrations and concentrations with hemodynamic in cardiac disease. *Br Heart J* 1993; 69: 414-7.
26. Magga J, Vuolteenaho O, Tokola H, Marttila M, Ruskoaho H. B-type natriuretic peptide: a myocyte-

specific marker for characterizing load-induced alterations in cardiac gene expression. Ann Med 1998; 30 (Suppl 1): 39-45.

27. Buckley MG, Sethi D, Markandu ND, Sagnella GA, Singer DR, MacGregor GA. Plasma concentrations and comparisons of brain natriuretic peptide and atrial natriuretic peptide in normal subjects, cardiac transplant recipients and patients with dialysis-dependent chronic renal failure. Clin Sci 1992; 83: 437-44.

28. Anand IS, Chandrashekhar Y, Ferrari R, et al. Pathogenesis of congestive state in chronic obstructive pulmonary disease. Studies of body water and sodium, renal function, hemodynamic and plasma hormones during edema and after recovery. Circulation 1992; 86: 12-21.

29. Baertschi AJ, Adams JM, Sullivan MP. Acute hypo-
xemia stimulates atrial natriuretic factor *in vivo*. Am J Physiol 1988; 255: H255-300.

30. Lang CC, Coutie WJ, Struthers AD, Dhillon DP, Winter JH, Lipworth BJ. Elevated levels of brain natriuretic peptide in acute hypoxemic chronic obstructive pulmonary disease. Clin Sci 1992; 83: 529-33.

31. Jacobsen SJ, Bergstrahl EJ, Guess HA, et al. Predictive properties of serum prostate-specific antigen testing in community-based setting. Arch Intern Med 1996; 156: 2462-8.

32. Swets JA, Getty DJ, Picker RM, D'orsi CJ, Seitz SE, McNeil BJ. Enhancing and evaluating diagnosis accuracy. Med Decis making 1991; 11: 9-18.

33. Fahey MT, Irwig L, Macaskill P. Meta-analysis of Pap test accuracy. Am J Epidemiol 1995; 141: 680-9.

บทบาทของพลาสม่า เบรน แอนทิทรียูเรติก เป็นไทร์ ในการแยกโรคหัวใจจากโรคอื่น ๆ

จีระศักดิ์ สิริอัญญานท์, พบ*, วัฒนา เลี้ยวัฒนา, พบ**,
ยศรีร์ สุขุมalgorithm, พบ***, สมศักดิ์ ชัยคุกุมงคลลาภ, พบ***, เสริมกิจ วัฒนาวารุณ, พบ***,
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ที่มาของการศึกษา : การวินิจฉัยภาวะหัวใจล้มเหลวจากการซักประวัติ ตรวจร่างกาย อาจทำได้ยากในผู้ป่วยบางราย คุณผู้ว่าจัดต้องการศึกษาประযุกชน์ของการตรวจ NT-proBNP ในการวินิจฉัยภาวะหัวใจล้มเหลว

วิธีการศึกษา : เป็นการศึกษา cross sectional study ในผู้ป่วย 48 คนที่หน่อยจากภาวะหัวใจล้มเหลวและ 50 คนที่มีอาการเหนื่อยจากสาเหตุอื่น เทียบกับกลุ่มควบคุมที่มารับการตรวจสุขภาพ 200 คน ทุกคนได้รับการเจาะเลือด 5 มิลลิลิตร ตรวจ NT-proBNP

ผลการศึกษา : ในกลุ่มควบคุม ระดับ NT-proBNP ไม่ต่างกัน ระหว่าง เพศชายและเพศหญิง อายุไม่มีผลต่อระดับ NT-proBNP, left ventricular ejection fraction เฉลี่ยของผู้ป่วยหัวใจล้มเหลวเป็น 32.4 ± 9.7 เปอร์เซ็นต์ ระดับ NT-proBNP ของผู้ป่วยหัวใจล้มเหลวสูงกว่ากลุ่มควบคุมอย่างมีนัยสำคัญทางสถิติ ($8,912 \pm 12,525$ pg/ml vs $386 \pm 1,041$ pg/ml, $p < 0.001$) ถ้าใช้ระดับ NT-proBNP > 150 pg/ml ในการวินิจฉัยภาวะหัวใจล้มเหลวจะได้ sensitivity 96 เปอร์เซ็นต์ specificity 72 เปอร์เซ็นต์และ sensitivity จะเป็น 96 เปอร์เซ็นต์และ specificity 80 เปอร์เซ็นต์ ถ้าใช้ระดับ NT-proBNP > 200 pg/ml, sensitivity เป็น 94 เปอร์เซ็นต์และ specificity 82 เปอร์เซ็นต์ ถ้าใช้ระดับ NT-proBNP > 300 pg/ml, ระดับ NT-proBNP มีความสัมพันธ์กับ functional class ของผู้ป่วยและไม่แตกต่างกันระหว่าง ischemic และ non-ischemic cardiomyopathy

สรุป : การตรวจ NT-proBNP เป็นการตรวจที่สามารถช่วยในการวินิจฉัยภาวะหัวใจล้มเหลวในผู้ป่วยที่มาด้วยอาการหอบเหนื่อย

คำสำคัญ : หัวใจหาย, เอ็นพี-โปรบีเอ็นพี, ความไว, ความจำเพาะ, ค่าคาดหมาย

จีระศักดิ์ สิริอัญญานท์, วัฒนา เลี้ยวัฒนา, ยศรีร์ สุขุมalgorithm, และคณะ
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