METABOLIC EFFECTS

Amino-Terminal Pro-B-Type Brain Natriuretic Peptide: Screening for Cardiovascular Disease in the Setting of Alcoholism

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Abstract — Aims: N-terminal pro-BNP (NtBNP) has attracted attention as a biomarker for heart failure. The aims of our study are (a) to characterize the role of NtBNP as a biological marker in the setting of alcoholism; (b) to describe potential gender differences with respect to NtBNP; (c) to correlate NtBNP with other clinical and haemodynamic variables. Methods: We examined 83 alcohol-dependent patients according to International Classification of Disease 10th Revision (ICD-10) and Diagnostic and Statistical Manual of Mental Disorders Fourth Edition (DSM-IV; 59 males and 24 females, age: 50 ± 10.5 years) referred to the department of psychiatry for alcohol withdrawal therapy. In these patients, we determined NtBNP, markers of alcohol abuse and transthoracic echocardiography to determine systolic left ventricular ejection fraction (EF). These measurements were repeated after alcohol withdrawal. Results: At Day 1 of alcohol withdrawal, 43 patients (52%; 27 males and 16 females) had elevated NtBNP levels (394.4 ± 438.7 pg/ml; P < 0.01), despite unchanged EF (65.0 ± 5.8%; P = ns). This was the case in both males (228.6 ± 251.2 pg/ml; P < 0.05) and females (328.9 ± 355.5 to 216.7 ± 194.3 pg/ml; P < 0.05 vs. 492.7 ± 635.7 to 246.6 ± 327.7 pg/ml; P < 0.05). Elevated NtBNP levels were related significantly to the history of arterial hypertension (P < 0.05). Conclusion: This study highlights the fact that NtBNP can be elevated in the setting of alcoholism. The elevation in NtBNP is unrelated to EF and is reversible after alcohol withdrawal. We suggest a subclinical detrimental effect of alcohol abuse on cardiac function.

INTRODUCTION

N-terminal pro-BNP (NtBNP), which is mainly produced in the ventricular myocardium, has attracted attention as a biomarker for heart failure (Bergler-Klein et al., 2004; Costello-Boerrigter et al., 2006; Daniels and Maisel, 2007; Galasko et al., 2005; Markham and De Lemos, 2005; Moe, 2006).

NtBNP, with a 17-amino ring structure, is released in response to cardiomyocyte stretch that may occur during many pathophysiological processes (ventricular systolic and diastolic dysfunction, cardiac hypertrophy, increased intracardiac filling pressures-wall stress, ischaemia and renal insufficiency). NtBNP circulates as a cardiac hormone in the body inducing vasodilation, natriuresis and diuresis in various tissues (De Lemos et al., 2003).

Several studies show that the effect of ethanol on the cardiovascular system is dose-dependent: regular moderate consumption reduces the cardiovascular and overall mortality, whereas excessive or binge drinking is associated with detrimental effects on the cardiovascular system such as cardiomyopathy, hyperlipidaemia, vasoconstriction, arterial hypertension, a lower threshold for atrial fibrillation and may lead to an elevation of NtBNP (Costanzo et al., 2010; Di Castelnuovo et al., 2006; Mukamal, 2003). However, the role of alcohol dependence and alcohol withdrawal specifically on the neurohormone NtBNP is unclear.

The aims of our study are (a) to characterize the role of NtBNP as a biological marker in the setting of alcoholism; (b) to describe potential gender differences with respect to NtBNP; (c) to correlate NtBNP with other clinical and haemodynamic variables.

PATIENTS AND METHODS

Patients

A total of 83 consecutive male and female alcohol-dependent patients (according to International Classification of Disease 10th Revision (ICD-10) and Diagnostic and Statistical Manual of Mental Disorders Fourth Edition (DSM-IV)) aged 27–74 years, currently suffering from alcohol withdrawal, referred to the department of psychiatry for alcohol withdrawal therapy, were included in this prospective study. The study was performed as a collaboration project between the departments of psychiatry and cardiology of the Medical University of Vienna, Austria.

At the baseline day prior to withdrawal, blood samples were taken at 8 a.m., followed by an electrocardiogram (ECG), breath alcohol concentration (BAC) was determined and questionnaires were admitted. Transthoracic echocardiography was performed on the same day. After completion of withdrawal treatment (16.6 ± 7.8 days) in patients with elevated NtBNP levels at baseline, both blood samples and echocardiography were repeated.

Exclusion criteria were: pregnancy, advanced renal insufficiency (serum creatinine levels >2.0 mg/dl), atrial fibrillation on the ECG and liver cirrhosis because NtBNP levels can be affected.

Patient characteristics are given in Table 1. The patient sample consisted of 59 (73.5%) males and 24 females (26.5%) with a mean age of 50 ± 10.5 years (range 27–74 years); mean body mass index (BMI) was 24.5 ± 3.4 (range 18.3–37.2), whereas men had significantly higher values (25.1 ± 3.6 vs. 23 ± 2.4; P < 0.05); the alcoholism typology according to Lesch was distributed as follows: Type I (n = 16, 19.3%), Type II (n = 27, 32.5%), Type III (n = 21, 25.3%),...
Type IV ($n = 19, 22.9\%$); 35 patients (42.1\%) had a positive family drinking history and 21 patients (25.3\%) developed alcohol dependence before or at the age of 25 years (early onset); systolic blood pressure was 134.6 ± 17.2 mmHg (range 107–114); diastolic blood pressure was 86.2 ± 11.3 mmHg (range 64–114); 16 patients (19.3\%) had a history of hypertension and were on antihypertensive therapy; one patient suffered from coronary artery disease; no patient had atrial fibrillation on the ECG.

The study was approved by the local Ethics Committee (Nr: 123/2008), and written informed consent was obtained from all patients prior to the study.

Lesch Alcoholism Typology (www.lat-online.at)

At baseline, all patients were clinically assessed by a psychiatrist using the Lesch Alcoholism Typology (LAT) online program, a standardized structured interview assessing different areas of alcohol dependence (e.g. Lesch Typology, family history positive/negative, early ($\leq 25$ years)/late ($>25$ years) onset of dependence). Using the items, 4 (tremor), 5 (paroxysmal sweats) and 11 (anxiety) of the revised Clinical Institute Withdrawal Assessment for Alcohol Scale, which are integrated part of LAT, alcohol withdrawal syndrome was measured and verified by a score $\geq 12$ (severe withdrawal symptoms) in these three items (Lesch et al., 2011; Nimmerrichter et al., 2002).

Ethanol intake

At baseline, drinking behaviour was assessed using the time line follow back method (Sobell and Sobell 1992) and by measuring %BAC using the Dräger Alcotest 6510.

Echocardiography

A commercially available ultrasound system (Vivid 7, General Electric Inc) was used. All patients underwent a comprehensive examination, including M-mode, and 2D echocardiography and continuous-wave, pulsed-wave and colour Doppler. The ejection fraction (EF) was calculated with the biplane Simpson method (Otterstad et al., 1997).

Biomarkers

Determination of mean corpuscular volume (MCV), gamma glutamyl transferase (GGT), glutamat-oxalacetate-transaminase (GOT) and glutamat-pyruvate-transaminase (GPT) was performed using routine clinical laboratory methods.

Carbohydrate deficient transferrin (%CDT) (cut-off $\geq 2.3\%$) was determined using the HPLC kit from Bio-Rad Laboratories (Munich, Germany).

For the determination of NtBNP, venous blood samples were drawn into chilled EDTA Vacutainer test tubes, and an electrochemiluminescence immunoassay (proBNP Elecsys, Roche Diagnostics GmbH) was used. The cut-off value for elevated levels (males and females) was defined as $>100$ pg/ml (Galasko et al., 2005).

Statistical analysis

Data were expressed as frequencies or percentages for discrete variables and means ± standard deviation for continuous variables. Comparisons between groups were made using the $\chi^2$ test for categorical variables, and the Student t-test for continuous variables. Statistical analysis was performed with SPSS 15 (SPSS Inc., IL, USA). Statistical significance was considered if $P \leq 0.05$.

RESULTS

Alcohol consumption

The mean %BAC was $0.6 \pm 0.9$ (range 0–3) at the time of admission, MCV $95.8 \pm 6.1$ (range 77.2–109.4) fl, mean de-Ritis-ratio (GOT/GPT) $1.3 \pm 0.6$ (range 0.23–3), mean GGT $275.7 \pm 410.7$ (range 20–2254) U/l and mean %CDT $3.8 \pm 4.9$ (range 0.43–27.69) %, whereas 42% were CDT positive ($\geq 2.3\%$). The patients consumed a mean of 1202.3...
At Day 1 of alcohol withdrawal, the mean NtBNP level for the entire population was 223.1 ± 329.6 pg/ml (range 7.9–2488). Forty-three patients (63% of males and 37% of females) had elevated NtBNP levels (394.4 ± 438.7 pg/ml, range 102.1–2488). NtBNP was significantly (P < 0.05) related to age. Patients with a history of hypertension were more likely to have elevated NtBNP values (P < 0.05; Table 3). All patients had normal EF (64.7 ± 6.0%, range 51–76). There was no difference in EF between males and females (64.7 ± 6 vs. 64.8 ± 6.2%; P = ns). The 40 patients with normal NtBNP levels (54.3 ± 27.6 pg/ml, range 7.9–99.7) did not differ in EF to the group with elevated NtBNP levels (64.7 ± 5.9 vs. 64.7 ± 6.2%; P = ns). These two patient groups did also not differ with respect to: age, BMI, GGT, %CDT, GOT/GPT, MCV, BAC and grams of ethanol intake in the last 7 days (Table 4).

**NtBNP and EF after alcohol withdrawal**

After withdrawal was completed (16.6 ± 7.8 days), the group with elevated NtBNP levels showed a significant decrease of this biomarker (P < 0.01). This was the case both in males and females (328.9 ± 235.5–216.7 ± 194.3 pg/ml; P < 0.05 vs. 492.7 ± 635.7–246.6 ± 327.7 pg/ml; P < 0.05; Fig. 1). The EF remained unchanged (65.0 ± 5.8%, range 54–75; P = ns).

**Family history of alcohol dependence**

In the total patient cohort, no differences could be found between family positive and family negative patients (P = ns; Table 5).

**Early (≤ 25 years) and late onset (>25 years) of alcohol dependence**

Besides lower NtBNP levels in younger patients (P < 0.05), no differences could be found between early and late onset of alcohol dependence (Table 5). Early alcohol dependence was not related to positive family drinking history (P = ns).

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**Table 3. Comparison of patient groups with normal and elevated NtBNP (cardiovascular parameters) at baseline**

<table>
<thead>
<tr>
<th></th>
<th>All</th>
<th>Elevated NtBNP</th>
<th>Normal NtBNP</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RR systolic (mmHg)</td>
<td>134.6 ± 17.2</td>
<td>139 ± 15.9</td>
<td>130.5 ± 17.7</td>
<td>NS</td>
</tr>
<tr>
<td>RR diastolic (mmHg)</td>
<td>86.2 ± 11.3</td>
<td>87.6 ± 12.3</td>
<td>84.8 ± 10.3</td>
<td>NS</td>
</tr>
<tr>
<td>Antihypertensive therapy</td>
<td>15 (18.1)</td>
<td>13 (30)</td>
<td>2 (5)</td>
<td>P &lt; 0.01</td>
</tr>
</tbody>
</table>

The elevated NtBNP group shows significantly more patients with treated arterial hypertension (P < 0.05).

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**Table 4. Comparison of patient groups with normal and elevated NtBNP (patient characteristics, hepatic function and alcohol consumption) at baseline**

<table>
<thead>
<tr>
<th></th>
<th>Elevated NtBNP</th>
<th>Normal NtBNP</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>27 (63)</td>
<td>32 (80)</td>
<td>NS</td>
</tr>
<tr>
<td>Age (years)</td>
<td>51.8 ± 11.6</td>
<td>48.1 ± 8.7</td>
<td>NS</td>
</tr>
<tr>
<td>BMI</td>
<td>24.1 ± 3</td>
<td>25 ± 3.9</td>
<td>NS</td>
</tr>
<tr>
<td>GGT</td>
<td>290.2 ± 410.5</td>
<td>260.4 ± 415.9</td>
<td>NS</td>
</tr>
<tr>
<td>%CDT</td>
<td>3.7 ± 4.7</td>
<td>3.9 ± 5.1</td>
<td>NS</td>
</tr>
<tr>
<td>De-Ritis-ratio (GOT/GPT)</td>
<td>1.4 ± 0.7</td>
<td>1.1 ± 0.5</td>
<td>NS</td>
</tr>
<tr>
<td>MCV</td>
<td>96.5 ± 6.5</td>
<td>95.1 ± 5.6</td>
<td>NS</td>
</tr>
<tr>
<td>BAC (%)</td>
<td>0.6 ± 0.9</td>
<td>0.6 ± 0.9</td>
<td>NS</td>
</tr>
<tr>
<td>Gram ethanol intake in the last 7 days</td>
<td>1207.3 ± 621.5</td>
<td>1193.9 ± 503.9</td>
<td>NS</td>
</tr>
</tbody>
</table>

No significant group differences could be found (P = ns).

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± 574 (range 392–2993) g of ethanol in the last 7 days prior to hospitalization (Table 2).

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**Table 2. Alcohol consumption**

<table>
<thead>
<tr>
<th></th>
<th>All, 83 (100)</th>
<th>Male, 59 (73.5)</th>
<th>Female, 24 (26.5)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BAC (%)</td>
<td>0.6 ± 0.9 (0–3)</td>
<td>0.6 ± 0.9 (0–2.8)</td>
<td>0.6 ± 0.9 (0–2.8)</td>
<td>NS</td>
</tr>
<tr>
<td>MCV</td>
<td>95.8 ± 6.1 (77.2–109.4)</td>
<td>95.5 ± 5.4 (86.7–108.7)</td>
<td>96.5 ± 5.4 (86.7–108.7)</td>
<td>NS</td>
</tr>
<tr>
<td>De-Ritis-ratio (GOT/GPT)</td>
<td>1.3 ± 0.6 (0.2–3)</td>
<td>1.2 ± 0.6 (0.2–3)</td>
<td>1.5 ± 0.6 (0.7–2.8)</td>
<td>NS</td>
</tr>
<tr>
<td>GGT</td>
<td>275.7 ± 410.7 (20–2254)</td>
<td>277 ± 395.9 (20–2254)</td>
<td>272.6 ± 453.5 (24–2071)</td>
<td>NS</td>
</tr>
<tr>
<td>% CDT</td>
<td>3.8 ± 4.9 (0.4–27.7)</td>
<td>4.1 ± 5.1 (0.4–27.7)</td>
<td>3.1 ± 4.3 (0.61–19)</td>
<td>NS</td>
</tr>
<tr>
<td>Gram ethanol intake in the last 7 days</td>
<td>1202.3 ± 574 (392–2993)</td>
<td>1302.5 ± 643.9 (467–2993)</td>
<td>1037.1 ± 399.6 (392–1680)</td>
<td>NS</td>
</tr>
</tbody>
</table>

BAC, breath alcohol concentration.
DISCUSSION
The biomarker NtBNP has become an important tool in diagnosis and prognosis of heart failure due to left ventricular dysfunction. Several studies have highlighted the role of NtBNP measurement for risk stratification in cardiovascular disease (CVD) associated with acute and chronic heart failure. NtBNP levels are more closely associated with mortality than New York Heart Association class or left ventricular EF (Maeda et al., 2000; Richards et al., 1999; Stanek et al., 2001). The prognostic value of NtBNP has been shown for patients after acute myocardial infarction (Nagaya et al., 1998) and unstable angina (de Lemos et al., 2001). When screening patients for elevated NtBNP, age and sex should be considered (Redfield et al., 2002; Wang et al., 2002). Therefore, our findings of higher NtBNP in females and older patients are not surprising.

It is well established that alcohol exhibits both acute and chronic toxic effects on the heart. Our finding of elevated NtBNP values in this population of patients with acute alcohol abuse confirms these findings. After completion of alcohol withdrawal treatment, NtBNP decreased significantly, despite unchanged EF. This strongly supports the association between NtBNP and alcohol. To our knowledge, this study is the first to evaluate the role of NtBNP in the setting of alcoholism in a psychiatric patient population regarding plasma level changes during alcohol withdrawal treatment.

Our findings are, however, in contrast to animal studies in rats that did not show an elevation in NtBNP neither in, nor after, alcohol provocation (Kim et al., 2001, 2003).

In contrast to our study, this study used a model, which studied more the chronic and not acute effects of alcohol consumption. In addition, the effects of alcohol could be different between humans and animals.

In this context also, the dose-dependent effect of ethanol is of great interest: existing data provide evidence that a dose of already >20 mg ethanol per day substantially increases the risk of cardiovascular and overall mortality in CVD patients (Janszky et al., 2008). Our patients consumed a total of 1202.3 ± 574 g of ethanol per week (corresponding to an amount of ~170 g per day). Nevertheless, only one of our patients suffered from coronary artery disease.

In our findings, 52% of the alcohol-dependent patients had elevated NtBNP levels at the time of admission, independent of cardiac function. Possible explanations for this finding are: (a) the EF is not sensitive enough to detect subtle contractile abnormalities, which might be present in alcohol abuse. (b) Patients with alcohol abuse show adrenergic activation, which affects NtBNP values. (c) The altered volume state in alcohol-dependent individuals affects NtBNP values. (d) Metabolism and secretion of NtBNP is directly affected by alcohol or liver function.

There is compelling evidence that NtBNP has a high specificity and sensitivity in detecting cardiac dysfunction opposed to the EF (Costello-Boerrigter et al., 2006).

Our finding that a history of hypertension is associated with elevated NtBNP levels favours the hypothesis that indeed discrete abnormalities of contractile function, which are often present in hypertension, play an important role and that abnormalities of cardiac dysfunction are important factors that lead to NtBNP elevation in alcohol abuse.

This finding is especially relevant since the prevalence of hypertension in the general population rises linearly with alcohol consumption (Sesso et al., 2008). Several studies show an association between NtBNP elevation and elevated blood pressure (de Lemos et al., 2003).

Even though all patients were in sinus rhythm, we cannot exclude that paroxysmal atrial fibrillation contributes to the elevation in NtBNP. It is well established that heavy alcohol consumption is associated with an increased risk for atrial fibrillation and consequently NtBNP elevation (Bai et al. 2009; Conen et al., 2008; Djoussé et al., 2004; Mukamal et al., 2007).

Furthermore, liver cirrhosis has been associated with elevated NtBNP levels (Padillo et al., 2010; Radvan et al., 2009). However, none of our patients were in an advanced stage of cirrhosis. Parameters of hepatic function were similar in the group of patients with and without elevated NtBNP. In addition, NtBNP elevation was reversible after withdrawal. All these findings suggest that cirrhosis was not the main factor for NtBNP elevation. However, we cannot exclude that impairment of hepatic function caused by excessive alcohol consumption contributes to NtBNP elevation.

In the last decades, a wide variety of models to classify alcoholism have been applied in different clinical trials confirming that alcohol dependence is a heterogeneous disorder (Babor et al., 1992; Cloninger et al., 1981; Leggio et al., 2009; Lesch et al., 1988).

It is evident that different subgroups, even if the subtype classification of alcoholism remains controversial, need different therapy approaches, concerning medical withdrawal and relapse prevention treatment (Johnson, 2010; Lesch et al., 2011).

Table 5. Comparison of patients with positive and negative family history and early and late onset of alcohol dependence

<table>
<thead>
<tr>
<th></th>
<th>Family positive</th>
<th>Family negative</th>
<th>P-value</th>
<th>Early onset (≤25 years)</th>
<th>Late onset (&gt;25 years)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>48.4 ± 9.4</td>
<td>51.2 ± 12</td>
<td>NS</td>
<td>46.4 ± 11.6</td>
<td>50.4 ± 9.5</td>
<td>NS</td>
</tr>
<tr>
<td>BMI</td>
<td>24.1 ± 3</td>
<td>24.3 ± 3.2</td>
<td>NS</td>
<td>24.3 ± 3.2</td>
<td>24.1 ± 3.1</td>
<td>NS</td>
</tr>
<tr>
<td>GGT</td>
<td>254.8 ± 294.6</td>
<td>256.9 ± 423.7</td>
<td>NS</td>
<td>282.6 ± 516.6</td>
<td>284.8 ± 408.6</td>
<td>NS</td>
</tr>
<tr>
<td>% CDT</td>
<td>2.6 ± 2.6</td>
<td>4.6 ± 4.4</td>
<td>NS</td>
<td>3.9 ± 3.9</td>
<td>4.5 ± 6.1</td>
<td>NS</td>
</tr>
<tr>
<td>De-Ritis-ratio</td>
<td>1.2 ± 0.7</td>
<td>1.3 ± 0.6</td>
<td>NS</td>
<td>1.1 ± 0.6</td>
<td>1.2 ± 0.6</td>
<td>NS</td>
</tr>
<tr>
<td>MCV</td>
<td>95.7 ± 6.7</td>
<td>95.7 ± 6.1</td>
<td>NS</td>
<td>96.7 ± 5.1</td>
<td>96.6 ± 2.2</td>
<td>NS</td>
</tr>
<tr>
<td>BAC [%]</td>
<td>0.5 ± 0.8</td>
<td>0.7 ± 1</td>
<td>NS</td>
<td>0.5 ± 0.8</td>
<td>0.7 ± 0.9</td>
<td>NS</td>
</tr>
<tr>
<td>Gram ethanol intake in the last 7 days</td>
<td>1224.6 ± 496.1</td>
<td>1179 ± 740.9</td>
<td>NS</td>
<td>1285.5 ± 570.9</td>
<td>1191.1 ± 622.3</td>
<td>NS</td>
</tr>
<tr>
<td>NtBNP (pg/ml)</td>
<td>205.5 ± 231.4</td>
<td>288.2 ± 439.6</td>
<td>NS</td>
<td>119.8 ± 90.9</td>
<td>261.0 ± 412.2</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

NtBNP is significantly lower in early onset compared with late-onset alcohol-dependent patients (P < 0.05).
In the literature, a simple binary model named early-onset alcoholism (EOA) and late-onset alcoholism is described. Based on a careful history of self-reported problems related to drinking and the age of onset (≤25 years and >25 years), a classification can be made for each individual on a priori basis. EOA, which is characterized by high family loading, could not be confirmed in our study. Nevertheless, this classification seems to be useful in treatment of EOA concerning especially serotonergic medications (e.g. ondansetron; Dawes et al., 2005). Also the genetic research in this subgroup seems to be hopeful to improve future therapy approaches (Dahmen et al., 2005; Lesch et al., 2011).

Considering the earlier-mentioned differentiated treatment of alcohol-dependent patients, we compared patients with the history of alcohol abuse before and after the age of 25 years (early vs. late onset) and patients with and without family history of alcohol abuse (family positive vs. negative) concerning age, BMI, liver parameters, blood cell count, and levels of ethanol intake in the last 7 days prior to hospitalization. Nevertheless, no group differences could be found.

Among the limitations of the study, are the relatively small patient numbers and the lack of follow-up of the patient group with normal NtBNP levels at the beginning of alcohol withdrawal. In addition, as already mentioned, we did not screen for paroxysmal atrial fibrillation.

Including the fact that individuals with mental disorders have higher incidence and prevalence rates for CVD, an early detection and treatment of physical illness is essential, particularly in this patient population (Weiser et al., 2009). Despite better and increasing pharmacological strategies for its treatment, alcoholism in connection with CVD is still a serious cause of morbidity and mortality. Elevated NtBNP values in the setting of alcohol abuse must be interpreted different than in other clinical conditions. It does not necessarily reflect overt heart failure and it is potentially reversible. However, the cause of NtBNP elevation in these patients needs further assessment.

Because NtBNP has a high potential to detect myocardial dysfunction and is easy to determine at a reasonable cost, it should be used in the setting of alcohol abuse. Our study also suggests that a closer cooperation between psychiatric and internal/ cardiologic departments should be anticipated to optimize the diagnosis and treatment in this risk group.

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