Chromosomal abnormalities and Y chromosome microdeletions in patients with azoospermia and cryptozoospermia

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Abstract

\textbf{Purpose:} To identify genetic abnormalities in patients with azoospermia and cryptozoospermia examined and treated at Da Nang Hospital for Women and Children.

\textbf{Materials and Methods:} A cross-sectional descriptive study was conducted at the Assisted Conception Unit, Da Nang Hospital for Women and Children from December 2020 to September 2023 including 72 cases of azoospermia and cryptozoospermia that met the inclusion and exclusion criteria and were included in the study.

\textbf{Results:} The average age of the study subjects was 31.8±5.5 years old. The average duration of infertility was 3.8±3.2 (years). There were ten cases of genetic abnormalities, accounted for 13.9\% (10/72), of which three cases of chromosomal abnormalities accounted for 4.2\% (3/72) and seven cases of Y chromosome microdeletions accounting for 9.7\% (7/72). All cases of Y chromosome microdeletions have normal karyotypes. Of the three cases of chromosomal abnormalities, there were two cases of Klinefelter syndrome accounting for 2.8\% and one case of chromosomal structural abnormality characterized by reciprocal translocation between the long arm of chromosome 9 and the short arm of chromosome 18 (46,XY,DER(9;18)(Q22;P11.3)). The rate of chromosomal polymorphism was 31.9\%. The rate of Y chromosome microdeletions in the AZFc region was highest at 71.4\%, of which 60\% of the deletions in the AZFc region were gr/gr and 40\% were b2/b4 deletions.

\textbf{Conclusions:} The genetic abnormalities in this study were similar to some studies in Asia and other regions around the world. Testing of karyotype and Y chromosome microdeletions is required in patients with azoospermia and cryptozoospermia.

\textbf{Keywords:} Azoospermia, Chromosomes, Cryptozoospermia, male infertility, Y chromosome microdeletions.

1. INTRODUCTION

Azoospermia is a condition in which there are no sperm in the ejaculate. It is estimated that the rate of azoospermia is 1 in 100 men of reproductive age and about 10\% of male infertility globally [1]. Cryptozoospermia is a condition in which no sperm are seen in the ejaculate but there are a few sperm in the centrifuged sediment. The rate of cryptozoospermia accounts for about 8.37\% of sperm abnormalities [2]. Genetic disorders occur in more than 30\% of male infertility cases. Chromosomal abnormalities are one of the most important causes of male infertility, accounting for 2.1\% - 28.4\% of infertile men and only 0.7\% - 1\% of men in the general population [3]. Y chromosome microdeletions are the second cause of male infertility following Klinefelter syndrome [2]. The human Y chromosome is essential for sexual differentiation and the development and maintenance of male germ cells. The azoospermia factor region is located on the long arm of the Y chromosome, plays an important role in male infertility genetics, and includes the three subregions AZFa, AZFb, and AZFc that have been described previously. The fourth region - the AZFd, was first discovered by Ken-First and colleagues using the multiplex PCR technique and is believed to be located between the AZF b and c regions. There is still debate about the existence of this region. Some authors believe that this region does not exist, while others believe that the deletion of this region is independent of the deletion of the AZFc region. The AZF region contains genes involved in spermatogenesis and testis development. Deletions in this region lead to spermatogenesis disorders and male infertility [4]. The rate of Y chromosome deletion in patients with azoospermia and severe oligozoospermia is 15.8\% and 11.5\%, respectively [5]. The most common deletion is in the AZFc region (approximately 80\%), followed by AZFb (1 - 3\%) and AZFa (0.5 - 4\%) [6].

With the development of assisted reproductive techniques, infertile men with Y chromosome microdeletions can still have biological children but there is an increased risk of genetic abnormalities for the next generation. Therefore, the American Society for Reproductive Medicine, the European Association of Andrology, and the European Association of Urology recommend genetic testing for patients with azoospermia and severe oligozoospermia [7]. To provide
a basis for genetic counseling for infertile men, this study was conducted to identify genetic abnormalities in men with azoospermia and cryptozoospermia examined and treated at Da Nang Hospital for Women and Children.

2. MATERIALS AND METHODS

2.1. Study Population
Inclusion criteria were infertile men diagnosed with azoospermia and cryptozoospermia. Exclusion criteria were men with acute genital infection, undescended testicles, and use of chemotherapy. All patients who met the inclusion and exclusion criteria were included in the study sample.

2.2. Study design
A cross-sectional descriptive study was conducted at the Assisted Conception Unit, Da Nang Hospital for Women and Children from December 2020 to September 2023.

2.3. Study procedures
Following taking a medical history and personal and family histories, the patients were also subjected to clinical examinations and laboratory tests. Tests included semen analysis, endocrine testing, scrotal ultrasound, karyotype, and Y chromosome microdeletions.

In this study, we did not perform testicular biopsy in all patients, therefore the differential diagnosis between obstructive azoospermia and non-obstructive azoospermia was not based on histological results but on two factors including FSH concentration and testicular volume. According to a study on Asians (Taiwan) by Huang IS et al., azoospermia patients with FSH concentration > 9.2 mIU/mL and right testicular volume < 15 ml were classified into the group of non-obstructive azoospermia. The remaining cases are obstructive azoospermia [8].

Semen analysis
Semen was analyzed according to the WHO manual 2010. Azoospermia was diagnosed when no spermatozoa were found in semen samples after centrifugation at 3000g for 15 min, and semen analysis was performed twice 4 weeks apart. Cryptozoospermia is diagnosed as rare sperm that can only be detected after centrifugation of semen samples [2].

Scrotal ultrasound
Determine the volume and abnormalities of the testicles, such as varicocele, epididymal cysts, and spermatic hydrocele. Testicular volume was determined according to Lambert’s formula: V = length x width x height x 0.71 (cm$^3$) [9].

Endocrine testing
FSH, LH, testosterone, and prolactin levels were measured using a French electrochemiluminescence immunoassay-ECLIA machine. Patients were instructed to fast and have blood taken for testing in the morning from 8 am to 11 am. Reference ranges were divided according to the manufacturer’s instructions for men. Normal values of FSH, LH, testosterone, and prolactin are 1.5 - 12.4 mIU/mL; 1.7 - 8.6 mIU/mL; 1.5 - 6.6 ng/mL, and 2.6 - 13.1 ng/mL, respectively.

Karyotype testing
Karyotype testing was performed according to the instructions of ECA 2012, cytogenetic nomenclature according to ISCN (International System for Human Cytogenetic Nomenclature) 2020. Cell culture using PBMax medium, GTG 400 band staining, imaging, and analysis using the Lucia Cytogenetics automatic system. Analyze 30 metaphase clusters, and prepare 10 sets of karyotypes.

Y chromosome microdeletion testing
Investigation of AZF deletions was conducted by amplifying specific STS primers (sequence-tagged sites) in the AZFa (Sy84, Sy86, Sy82, Sy83, Sy1065, Sy88), AZFb (Sy127, Sy134, Sy105, Sy121, Sy1192, Sy153), AZFc (Sy254, Sy255, Sy1191, Sy1291, Sy160), and AZFd (sY152, BpY2) with the SRY and ZFY genes as controls. PCR (polymerase chain reaction) and sequencing techniques were performed respectively on the PCR-9700 and 3130/3130xl Genetic Analyzer machines from Applied Biosystems (USA).

2.4. Statistical Analysis
Data were collected and analyzed using Medcalc software. Descriptive statistics (frequency and percentage) were used to present baseline, clinical, and subclinical characteristics. Percentages and frequencies were computed to report categorical data. The Chi-square test and One-way Anova were utilized to evaluate discrepancies between groups.

2.5. Ethical Approval
The study protocol was approved by the Medical Ethics Committee of Da Nang Hospital for Women and Children (45/BVPSN-DN/ HDYD//2020). All patient provided written informed consent for the publication and use of their data.

3. RESULTS
From December 2020 to September 2023, there were 58 cases of azoospermia and 14 cases of cryptozoospermia that met the inclusion and exclusion criteria and were included in the study. According to the classification of Huang IS et al [8], there were 48 cases of obstructive azoospermia and 10 cases of non-obstructive azoospermia.
3.1. Baseline Characteristics of study population

Table 1. Baseline Characteristics of study population

<table>
<thead>
<tr>
<th>Features</th>
<th>Semen analysis</th>
<th>OA</th>
<th>NOA</th>
<th>Cryptozoospermia</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean)</td>
<td></td>
<td>32.6</td>
<td>29.4</td>
<td>31.1</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Mean infertility duration (years)</td>
<td></td>
<td>3.9</td>
<td>4.2</td>
<td>3.0</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td></td>
<td>23.1</td>
<td>21.4</td>
<td>23.3</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Testicular volume (cm³)</td>
<td></td>
<td>17.9</td>
<td>6.5</td>
<td>13.0</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>FSH (mIU/ml)</td>
<td></td>
<td>5.1</td>
<td>31.2</td>
<td>19.3</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>LH (mIU/ml)</td>
<td></td>
<td>4.9</td>
<td>15.5</td>
<td>12.1</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Testosteron (ng/mL)</td>
<td></td>
<td>5.8</td>
<td>5.4</td>
<td>5.4</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Prolactin (ng/mL)</td>
<td></td>
<td>13.0</td>
<td>10.1</td>
<td>15.7</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

* One-way ANOVA test

There was no statistically significant difference in the average age, average duration of infertility, and BMI between the obstructive azoospermia and non-obstructive azoospermia, and cryptozoospermia groups (p > 0.05). There was no statistically significant difference in average FSH and LH levels in the non-obstructive azoospermia and cryptozoospermia groups (p > 0.05). FSH, LH levels in the cryptozoospermia and non-obstructive azoospermia groups were significantly higher than in the obstructive group (p < 0.0001). There was no statistically significant difference in testosterone and prolactin levels between the groups of non-obstructive azoospermia, obstructive azoospermia and cryptozoospermia (p > 0.05).

3.2. Genetic Characteristics

Table 2. Genetic Characteristics

<table>
<thead>
<tr>
<th>Genetic Characteristics</th>
<th>Semen analysis group</th>
<th>OA</th>
<th>NOA</th>
<th>Cryptozoospermia</th>
<th>Total</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>n (%)</td>
<td>n (%)</td>
<td>n (%)</td>
<td>100.0</td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td></td>
<td>45 (72.6)</td>
<td>5 (8.1)</td>
<td>12 (19.3)</td>
<td>62</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Abnormal</td>
<td></td>
<td>3 (30.0)</td>
<td>5 (50.0)</td>
<td>2 (20.0)</td>
<td>10</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Karyotype</td>
<td></td>
<td>0</td>
<td>2 (66.7)</td>
<td>1 (33.3)</td>
<td>3</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>KS (47, XXY)</td>
<td></td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Reciprocal translocation</td>
<td></td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Y chromosome microdeletion</td>
<td></td>
<td>3 (42.9)</td>
<td>3 (42.9)</td>
<td>1 (14.2)</td>
<td>7</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

* Chi-square test

There were 10 cases of genetic abnormalities (13.9%) (10/72), of which 3 cases of chromosomal abnormalities (4.2%) (3/72) and 7 cases of Y chromosome deletions accounted for 9.7% (7/72). All cases of Y chromosome microdeletion have normal karyotypes. Of the three cases of chromosomal abnormalities, there were two cases of Klinefelter syndrome accounting for 2.8%, and one case of chromosomal structural abnormalities characterized by reciprocal translocation between the long arm of chromosome 9 and the short arm of chromosome 18 (46,XY,DER(9;18)(Q22;P11.3)). The difference in the rate of genetic abnormalities, chromosomal abnormalities, and Y chromosome microdeletions among the three groups was not statistically significant (p > 0.05).

3.3. Karyotype distribution in cases with chromosomal polymorphism

Table 3. Karyotype distribution in cases with chromosomal polymorphism

<table>
<thead>
<tr>
<th>Karyotype</th>
<th>n</th>
<th>No of chromosomes with polymorphism</th>
</tr>
</thead>
<tbody>
<tr>
<td>46 XY, 21ps+</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>46 XY, 9qh+, 15 ps+, 22 ps+</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>46 XY, 9qh+, 14 ps+, 15 ps+, 22 ps+</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>46XY, 14 ps+, 15 ps+, 21 ps+</td>
<td>1</td>
<td>3</td>
</tr>
</tbody>
</table>
There were 23 cases of chromosome polymorphisms, accounting for 31.9%. Chromosomal polymorphisms include increased length of the heterochromatin region on the long arm of chromosomes 1 (1qh+), 9 (qh+), Y (Yqh+) and increased length of the short arm satellites of chromosomes 13 (13ps+), 14 (14ps+), 15 (15ps+), 21 (21ps+), 22 (22ps+). Among them, there was 1 case of increased length of the heterochromatin region on the long arm of the Y chromosome (46, XY, Yqh+), accounting for 4.3% (1/23).

### 3.4. Distribution of Y chromosome microdeletions according to the AZF region

<table>
<thead>
<tr>
<th>Y chromosome microdeletions according to the AZF region</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>AZFc</td>
<td></td>
<td></td>
</tr>
<tr>
<td>gr/gr deletions</td>
<td>3</td>
<td>71.4</td>
</tr>
<tr>
<td>b2/b4 deletions</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>AZFd</td>
<td></td>
<td>14.3</td>
</tr>
<tr>
<td>AZFcD</td>
<td></td>
<td>14.3</td>
</tr>
<tr>
<td>Total</td>
<td>7</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Among seven cases of Y chromosome microdeletion, there were five cases of Y chromosome deletion in the AZFc region accounting for 71.4%, one case of chromosome microdeletion in the AZFd region accounting for 14.3%, and one case of AZFc+d Y chromosome microdeletion (14.3%).

### 3.5. Factors affecting Y chromosome microdeletions

<table>
<thead>
<tr>
<th>Factors</th>
<th>Y Chromosome microdeletions</th>
<th>OR</th>
<th>CI 95%</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>31.3 ± 4.8</td>
<td>31.9 ± 5.6</td>
<td>0.979</td>
<td>0.846 - 1.132</td>
</tr>
<tr>
<td>Mean testicular volume (cm³)</td>
<td>10.7 ± 4.2</td>
<td>15.8 ± 6.9</td>
<td>0.883</td>
<td>0.773 - 1.008</td>
</tr>
</tbody>
</table>
4. DISCUSSION

Genetic abnormality is one of the main causes of male infertility. These abnormalities can be numerical or structural, connected to autosomal or sex chromosomes. In this study, the rate of genetic abnormalities was 13.9%. This rate is similar to that found in Arafá MM et al. (10.78%) (35/325) [10] and Akbarzadeh Khiavi M et al. (14.0%) (29/209) [6] but lower than the study of Chen SW et al 19.86% (15/78) [11]. The rate of genetic abnormalities in male infertility ranges from 3-19% and is 10 times higher than in the general population (0.4-0.6%) [12]. This rate in azoospermic patients varies from 11-24% and 2-16% in oligozoospermic patients [13]. These differences in the rate of genetic abnormalities are considered to be influenced by race, geography, inclusion criteria, and laboratory testing methods whether or not the cells that are categorized and analyzed include chromosomal polymorphism.

The rate of chromosomal abnormalities in our study was 4.2% which is equivalent to the studies of Suganya J et al (4.6%) [14] but lower than the studies conducted by Lee TH et al (23.1%) [5], Arafá MM et al (9.59%) [10]. This difference may be due to different inclusion criteria and chromosomal analysis techniques between studies. Research by Lee TH et al showed that the rate of chromosomal abnormalities in the azoospermia group was 30.0% (27/90) higher than the severe oligospermia group, which was 7.5% (p=0.0097) [5]. In our study, the difference in the rate of chromosomal abnormalities among the three groups was not statistically significant (p>0.05). This result can be explained by the insignificant difference in sperm concentration between the azoospermia and cryptozoospermia groups.

In this study, there were two cases of Klinefelter Syndrome (both with karyotype 47, XXX) accounting for 2.8%. This rate is similar to the study by Jafari-Ghahfarrokh H et al (3.3%) (2/60) [15] but lower than the study by Zhao P et al (21.2%) (7/33) [13]. 80-90% of Klinefelter Syndrome is characterized by a karyotype of 47, XXX, the remaining 10-20% are mosaic (47, XXY/46,XY), high-level aneuploidy (48, XXXY or 48, XXY) or an unusual X chromosome structure (eg 47,i(Xq,Y). The prevalence and incidence of this syndrome are about 0.15% (150/100,000 men) and 1/500-1/1000 (0.1-0.2%) in male live births, respectively, which varied by region and race. Klinefelter syndrome is the most common chromosomal abnormality in infertile men (3-4%) and accounts for 8-12% of azoospermic men. The majority of Klinefelter cases are infertile (99%), and azoospermic (91%), and the remaining 8% have few sperm in the semen (severe oligospermia). The detriment in spermatogenesis often progresses according to time until testicular atrophy is found [16].

Our study also recorded a case with a chromosomal pattern of 46,XY,DER(9;18)(Q22;P11.3). This is a chromosomal structural abnormality characterized by reciprocal translocation between two segments: the long arm of chromosome 9 and with the short arm of chromosome 18. Chromosomal translocations are the most common structural abnormalities in men, occurring at a frequency of 1.23/1000, and the rate is 10 times higher among infertile men. This abnormality involves the transfer of genetic material from one chromosome to another, including two main types: reciprocal and Robertsonian translocations. Chromosomal translocations cause a variety of sperm production phenotypes, ranging from normal spermatogenesis to spermatogenic failure [10, 17].

Chromosomal polymorphism includes different sizes of heterochromatin regions, satellite regions, and regions with repetitive sequences. This is considered a normal and heritable variation. However, there are more and more reports on the impact of chromosome polymorphisms on fertility, but the mechanism is still unknown [18]. The rate of chromosomal polymorphism in our study was 31.9%. This rate is equivalent to the study of Suganya J et al (37.2%) [14] but higher than the study of Atl El et al (6.0%) [19] and lower than the study of Minocherhomji S et al (58.68%) [20]. Differences in chromosomal polymorphisms between studies may be due to sample size, inclusion criteria, testing methods, and racial factors. In our study, 4.3% of cases had increased heterochromatin length on the long arm of the Y chromosome (46, XY, Yqh+). This rate is similar to that reported by Jafari-Ghahfarrokh H et al. (6%) [15]. The increased length of the heterochromatin region on the long arm of the Y chromosome is believed to play an important role in the reproductive process. This may be related to the inhibition of gene expression, especially genes related to reproduction during spermatogenesis. Recent studies show that Y chromosome polymorphism can affect homologous chromosome pairing and chromosome segregation, leading to cell division
disorders, embryonic development disorders, fetal demise, and miscarriage [19]. The association between chromosomal polymorphisms and male infertility is still a controversial topic because the role of heterochromatin regions has no clear clinical relevance. Therefore, research is needed to clarify this issue [21].

Y chromosome microdeletion has been identified as the most common cause of spermatogenesis failure. The phenotype associated with AZF deletions varies from azoospermia to normal semen analysis depending on the location and size of the deletion. The rate of Y chromosome microdeletions in our study was 9.7%. This rate is similar to the research of Sharma H et al (9.27%) (18/194) [22], of Waseem AS et al. (10.93%) (33/302) [23], but higher than the study of Arafa MM et al (2.2%) (7/325) [10], Damdinsuren E et al (2.66%) [24] and lower than the study of Lee TH et al (14.1%) [5], Akbarzadeh Khivi M et al (32.05%) (25/78) [6], Elsaid HOA et al (64%) (16/25) [25]. The global incidence of AZF microdeletions in infertility patients is estimated at 7%. This rate is lowest in Europe (3%), Australia (5.3%), and the rest of the world averages 8-9%. Among Asians, the rate of Y chromosome deletions is highest in East Asians and Southeast Asians, and lowest in West Asians. The frequency of Y chromosome microdeletions varies between studies, possibly due to the small number of samples in some studies, different sample selection criteria, the number of primer sequences used, and racial and environmental factors [25, 26].

Our study showed that the Y chromosome deletions in the AZFc region accounted for the highest rate at 60%. This result is similar to the study of Lee et al in which microdeletion of the AZFc region is the most common (59.2%) [5], Akbarzadeh Khivi M et al (80.0%) [6]. This is appropriate given the literature stating that the AZFc region is the most common deletion site in infertility patients (60-70%). However, there is an important point that is worth noting, the frequency of AZF site deletions in infertile patients is different between population groups. While the frequency of AZFc deletions was lower in Indian patients than in Western patients (45% vs. 60%), the frequency of AZFa region deletions was double (11% vs. 5%), respectively. In addition, the frequency of two region deletions (AZFa+b, b+c) in Indians is as twice as the rest of the world [26]. AZFc deletion mutations reduce sperm count and concentration which are associated with a variety of phenotypic features ranging from azoospermia or mild to severe oligozoospermia. Patients with this AZFc microdeletion have a higher chance of successful sperm retrieval than patients with any other type of deletion in the AZF region of Yq. For the group with microdeletion of AZFa, men with complete deletion of AZFa were azoospermic and could not retrieve sperm by any method. Deletion of the AZFb fragment shows more variable phenotypes. Patients with large AZFb deletions lead to no sperm, while partial AZFb deletions show many infertility phenotypes, including azoospermia, mild and severe oligozoospermia, and the most common pathological damage in this group is maturation arrest [4, 27].

In our study, gr/gr AZFc deletions accounted for 60% and the remaining were deletions of the b2/b4 segment (40%). The research results are in accordance with many studies in Vietnam and around the world on the prevalence of gr/gr AZFc deletions. Research by Rozen et al (2012) found that there were two types of partial AZFc deletions in Vietnamese people including deletions of the gr/gr type accounting for 94.11% (16/17) and b2/b3 (1/17 cases) (5.89%) [28]. Olesen et al (2017) found that partial AZFc loss of the gr/gr type accounted for the highest rate of 57.89% (33/57 cases); The lowest is the b2/b3 type, accounting for 36.84% (21/57 cases) and the lowest is the b1/b3 type, accounting for 5.27% (3/57 cases) [29]. The role of gr/gr deletions in male infertility remains controversial. Some studies have shown that the frequency of gr/gr deletions in infertile patients is significantly higher than in controls. However, some other studies show no correlation [7]. A Meta-analysis by Colaco S et al showed that the frequency of deletions gr/gr in male infertility patients was twice as high as in cases of normal fertility/normal semen analysis (OR =1.8). It is worth noting that gr/gr deletions are more common in Asians and Africans (10-15%) than in other populations (less than 5%). The association between gr/gr deletion and male infertility also depends on race, which is strongest in Caucasian men, weaker in Mongolians, and has no association in Dravidian and Nigro-Caucasian groups. Studies show that men with gr/gr deletions have significantly lower sperm count and motility compared to the group without deletions. This suggests that deletion of the gr/gr segment may not be a direct cause of infertility but damages spermatogenesis leading to a reduced sperm count [26].

In our study, no statistically significant association between age, BMI, testicular volume, and concentrations of FSH, LH, testosterone, and prolactin was found (Table 5). This is also appropriate to the research of Lee TH et al [5], Sharma H et al [21], and Uzay E et al [30].

5. CONCLUSION
The genetic abnormalities in our study are remarkably similar to those in other studies in Asia and around the world. There is a requirement for karyotype analysis and Y chromosomal microdeletion tests for infertile men characterized by azoospermia and cryptozoospermia.

REFERENCES


