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Ultrasound evaluation of long-term outcome in boys operated on due to testicular torsion

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Abstract

We aimed at verifying the usefulness of spectral Doppler ultrasonography in determining development of the testis after torsion in boys.

The study involved 28 patients and 30 control cases divided into 3 developmental groups: pre-pubertal, early pubertal, and pubertal. It presented surgical management in testicular torsion (TT), volume, and echogenicity of testes, as well as peak-systolic velocity (PSV), end-diastolic velocity (EDV), and vascular resistance index (RI) in the capsular and intra-testicular arteries, regarding developmental groups, detorsed testes, uninvolved ones, and testes in the control group.

Orchiectomy was performed in 13 boys with complete TT, in 11 lasting for over 24 hours, and in 2 lasting for 9 and 10 hours, respectively. Orchiectomy mainly involved patients aged up to 6 years, who at the time of the follow-up ultrasound belonged to the pre-pubertal group.

There is no clear correlation between the type of testicular torsion, its duration, and the echogenicity of the testis. Testicular torsion has a negative effect on the volume of detorsed testis with compensatory hypertrophy of the uninvolved testis. The study represents a new approach to the issue of long-term gonadal blood supply abnormalities after treatment of testicular torsion in childhood.

Abbreviations: 1 Q = the first quartile, 3 Q = the third quartile, C = complete, CT = testis in the control group, d = days, Dev. = developmental, DT = detorsed testis, EDV = end-diastolic velocity, F = fixation, H = homogenous, hrs = hours, IC = incomplete, L = left, Max. = maximal value, Me = median, Min. = minimal value, M–W = Mann–Whitney *U* test, NH = non-homogenous, O = orchiectomy, *P* = *P* coefficient, PSV = peak-systolic velocity, R = right, RI = vascular resistance index, TT = testicular torsion, US = ultrasound examination, UT = uninvolved testis, *X* = arithmetic mean, y = years, Z = value of statistics.

Keywords: boys, Doppler, echogenicity, testis, torsion, ultrasound, volume

1. Introduction

Testicular torsion (TT) is an urgent urological condition in which rotation of the testis around the axis of the spermatic cord takes place, causing a disturbance of venous and arterial blood flow, which may result in necrosis of the gonad.[1,2] That disease entity was described for the first time in 1840 by Delasiuave.[3]

TT occurs with a frequency of 1:4000 men up to 25 years of age.[3–7] In the developmental period, up to the age of 18, it affects 3.8% of the male population which is 150 times more often than in adults.[8] Two-peak incidence periods are observed: the neonatal-infant and the adolescent one.[2,5,6,8–13]

TT is the second cause of "acute scrotum" in boys, after torsion of the Morgagni appendix.[14,15] It can be complete (≥360°), incomplete (<360°) and recurrent.[1,5,11,16,17]

The testis, epididymis, vas deferens, and their coats are vascularized by 3 arterial vessels, the ends of which form a network of capillary connections: the internal spermatic artery, the external spermatic artery, and the deferential artery.[17–20] The internal spermatic artery—a branch of the abdominal aorta, is divided into the testicular artery and the epididymal artery. The testicular artery penetrates the tunica albuginea, close to the lower pole of the testis, and as a capsular artery forms a network around the gonad, called the vascular membrane. Its branches penetrate into the testicular septae as intra-testicular arteries. The external spermatic artery, a branch of the inferior epigastric artery, mainly vascularizes coats of the spermatic cord. The deferential artery, a branch of the upper bladder artery, supplies the vas deferens. With so many sources of vascularization, even in a complete torsion, the testis is still supplied with blood what gives some time for an urgent operation.

There are many publications discussing the results of treatment of various diseases of the testicular region, including measurements of peak-systolic velocity (PSV), end-diastolic velocity

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Data availability statement: The raw datasets generated and analyzed during the current study are available from the corresponding author.

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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(EDV), and vascular resistance index (RI) in gonadal vessels upon Doppler ultrasound examination (US).[18–30] On the other hand, only a few publications describe changes in echostructure, vascular flow, or gonadal volume in boys with TT.[2,4,5,10,16,31]

Therefore, we have undertaken research to explain the following issues. How the degree, duration of TT, and patient age at the time of surgery affect testicular viability? Assess the effect of TT on echogenicity, volume, and vascularization of testicular parenchyma.

2. Material and methods

2.1. Ethics

Informed, written consent for the purpose of studies, and publication of the manuscript was obtained for every patient from the parent or legal guardian and also from the patient over 16 years of age, due to the legal regulations in Poland. Studies were performed according to the Medical Association Declaration of Helsinki. The research protocol was approved by the Bioethics Commission of the Medical University of Lublin, Poland no. KE-0254/11/2017.

2.2. Participants

In years 2012 to 2016, in the Department of Pediatric Surgery and Traumatology, Medical University of Lublin in Poland 80 boys were operated on due to TT. Twenty eight of them were selected for the study group, those individuals regularly attended outpatient visits and had undergone the follow-up US examination. Patients were divided into 3 developmental groups at the time of the follow-up US:

1. pre-pubertal (age 1–8 years): 9 cases,
2. early pubertal (age 9–13 years): 3 cases,
3. pubertal (age 14–17 years): 16 cases.

The age of patients at the time of surgery was also given.

The control group consisted of 30 boys hospitalized in the same department due to diseases not related to the genitourinary system, also divided into 3 developmental groups (10 boys in each group).

2.3. The surgical technique

A transverse incision was made on the frontal surface of the hemiscrotum, due to pattern of skin vessels of the scrotal sac. The spermatic cord was detorsed and the degree of torsion was recorded. The testis and epididymis were wrapped in a warm, damp napkin to improve circulation and the spermatic cord was injected with 1% lignocaine solution to reduce the effect of painful stimuli from the operating field. After 10 minutes, testicular blood supply was assessed. If the testis has regained of its normal color or if its blood supply had improved, it was fixed with single, non-absorbable sutures on the lateral and medial sides. In cases where there was doubt, the tunica albuginea was cut. If arterial bleeding was seen after 5 minutes, the testis was left and fixed. In other cases, the testis was removed. Each boy had a prophylactic fixation of the uninvolved testis from a separate transverse scrotal incision.

The number of cases involving torsion of the right and left testes was recorded. The type of torsion was defined as complete ($\geq 360°$) or incomplete ($< 360°$), and the time of duration of TT was given in hours. The relationship between the type of torsion, its duration and surgical procedure (salvage of the testis or orchiectomy) was indicated.

2.4. The follow-up ultrasound

The follow-up US was performed by 1 specialist in ultrasonography after at least 1 year, but on average 3 years after surgery due to TT. The Siemens Acuson S2000 ultrasound apparatus with a 9 to 12 MHz linear transducer and spectral Doppler was used (Siemens Medical Solutions USA, Inc., Mountain View, CA, 1230 Shorebird Way). The echogenicity of the testicular parenchyma was defined as homogeneous or non-homogeneous. The length, width, and thickness of the testis were measured and its volume was calculated from the ellipsoidal formula: volume = length × width × thickness in cm × 0.52.[24]

Peak-systolic velocity (PSV) and end-diastolic velocity (EDV) were measured in the capsular and intra-testicular arteries and vascular resistance index (RI) was calculated from equation: RI = (PSV – EDV): PSV × 100.[21]

For statistical comparisons, the mean testicular volume, PSV, EDV, and RI in each developmental group were calculated.

The following ultrasonographic comparisons were made in developmental groups:

1. between detorsed and uninvolved testes,
2. between detorsed testes and testes in the control group,
3. between uninvolved testes and testes in the control group.

The comparisons concerned:

- the mean testicular volume,
- the mean PSV, EDV, and RI in the capsular artery,
- the mean PSV, EDV, and RI in the intra-testicular artery.

Exemplary results of the follow-up US carried on the detorsed and uninvolved testes, illustrating vascularization are shown in Figs. 1–4. It was done due to present the necessity of precision in vessels' identification and measurement of blood flow velocity.

2.5. Statistical analysis

The values of the parameters analyzed due to the quotient scale of measurement were characterized by arithmetic mean (X), minimal value (Min.), the first quartile (1 Q), median (Me), the third quartile (3 Q), and maximal value (Max.).

The distribution of the examined parameters was evaluated on the basis of the Shapiro-Wilk W test.[32,33] The Mann–Whitney U test (M–W) was used to compare 2 independent groups, presenting Z-value of statistics (Z) and P coefficient (P).[34–36]

A 5% inference error and a significance level of $P \leq .05$ indicating statistically significant differences or dependencies were accepted. The statistical analysis was conducted using the Statistica v. 13.1 software (StatSoft, Poland) in the Department of Medical Informatics and Statistics with E-learning Lab of our University.

3. Results

Testicular torsion involved the right and left sides with the same rate of frequency (Table 1). Complete torsion was found in 20 and incomplete in 8 patients. In 11 patients, the torsion lasted for over 24 hours and was complete. In 4 of those patients, fetal TT was found. In the remaining 17 patients, the duration of torsion

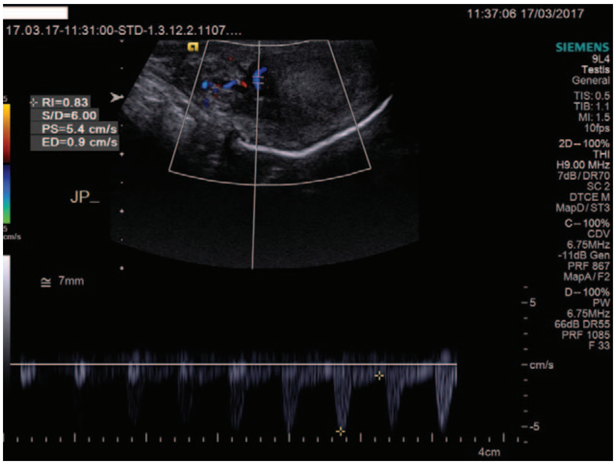

Figure 1. Detorsed testis, capsular artery: PSV=peak-systolic velocity, EDV=end-diastolic velocity, RI=vascular resistance index, S/D=PSV/EDV.

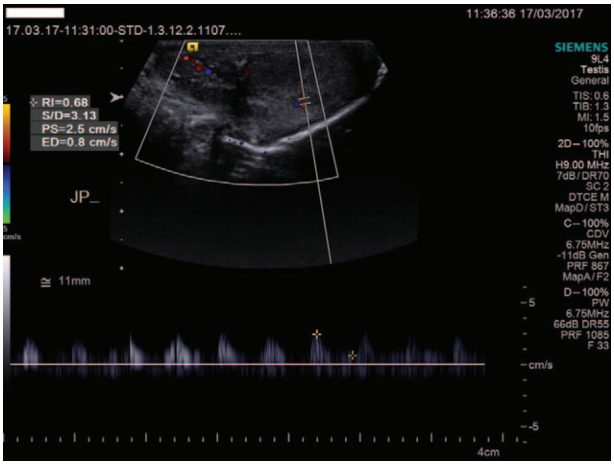

Figure 2. Detorsed testis, intra-testicular artery: PSV=peak-systolic velocity, EDV=end-diastolic velocity, RI=vascular resistance index, S/D=PSV/EDV.

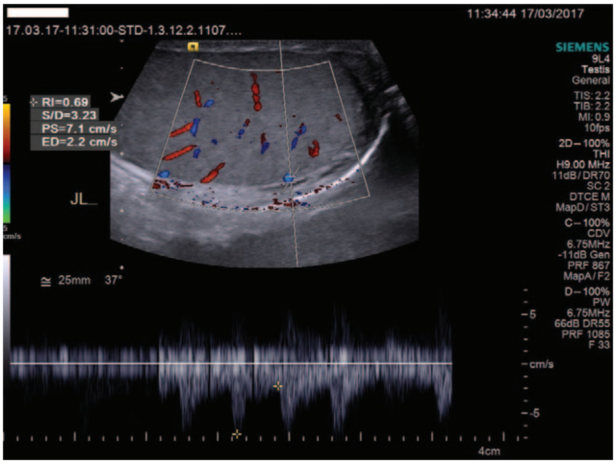

Figure 3. The uninvolved testis, capsular artery: PSV=peak-systolic velocity, EDV=end-diastolic velocity, RI=vascular resistance index, S/D=PSV/EDV.

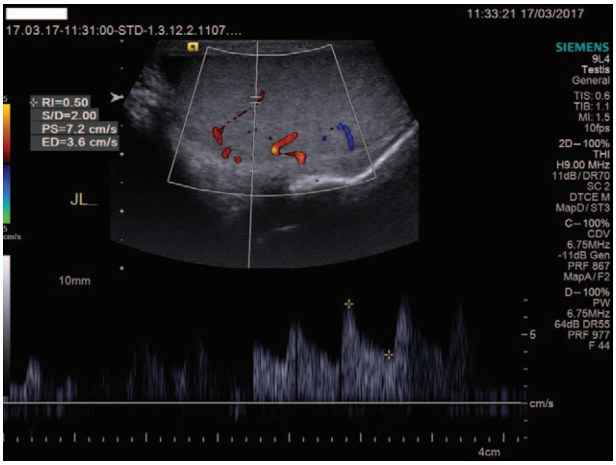

Figure 4. The uninvolved testis, intra-testicular artery: PSV=peak-systolic velocity, EDV=end-diastolic velocity, RI=vascular resistance index, S/D=PSV/EDV.

ranged from 2 to 15 hours, with an average of 7 hours. In 9 of them, the torsion was complete.

Orchiectomy was performed in 11 patients with complete TT lasting for over 24 hours and in 2 boys with complete torsion lasting for 9 and 10 hours, respectively. Orchiectomy was most often performed in patients aged up to 6 years, who at the time of the follow-up US belonged to the pre-pubertal group. In the remaining 15 cases, testicular detorsion and fixation were performed.

In 5 of 15 boys with salvaged testes, the follow-up US showed non-homogenous echogenicity of the parenchyma. In the other 10 cases, the echo was normal. There was no clear correlation between the type and duration of torsion and the echogenicity of the testicular parenchyma. Non-homogenous echogenicity was found in both complete torsion lasting for 15 hours and incomplete torsion lasting for only 5 hours.

3.1. The volume of testes

In the pre-pubertal group, it was found that the volume of the uninvolved testis was greater than the volume of the testis in the control group ($X=1.15$ mL vs $X=0.7$ mL, $Z=1.88$, $P=.06$). In the early pubertal group, the volume of the detorsed testis was less than the volume of the uninvolved testis ($X=4.6$ mL vs $X=5.27$ mL, $Z=-0.29$, $P=.78$) and greater than in the control group ($X=4.6$ mL vs $X=2.66$ mL, $Z=-0.25$, $P=.80$) respectively. The volume of the uninvolved testis was greater than in the control group ($X=5.27$ mL vs $X=2.66$ mL, $Z=-0.25$, $P=.80$). In the pubertal group, the volume of the detorsed testis was less than the volume of the uninvolved testis ($X=10.73$ mL vs $X=14.57$ mL, $Z=-0.26$, $P=.79$) and in the control group ($X=10.73$ mL vs $X=11.19$ mL, $Z=-1.24$, $P=.22$). The volume of the uninvolved testis was greater than the volume of testis in the control group ($X=14.57$ mL vs $X=11.19$ mL, $Z=1.84$, $P=.08$). The above relationships were not statistically significant (Fig. 5).

3.2. PSV in the capsular artery

In the pre-pubertal group, PSV in the uninvolved testis was statistically significantly lower than that in the control group ($X=4.01$ cm/s vs $X=5.23$ cm/s, $Z=-2.80$, $P=.04$). In the early

Table 1

Profile of torsion, surgery, and echogenicity of detorsed testes.

Patient no.	Dev. group	Age at surgery	Side	Type of torsion	Duration of torsion in hrs	Type of surgery	Echogenicity of detorsed testis
1	1	1 d	R	C	>24 (fetal torsion)	0	0
2	1	3 d	R	C	>24	0	0
3	1	1 d	L	C	>24 (fetal torsion)	0	0
4	1	1 d	R	C	>24 (fetal torsion)	0	0
5	1	2 d	R	C	>24 (fetal torsion)	0	0
6	1	6 y	L	C	>24	0	0
7	1	3 y	L	C	> 24	0	0
8	1	4 y	L	IC	12	F	NH
9	1	4 y	L	C	> 24	0	0
10	2	6 y	L	C	6	F	H
11	2	10 y	R	IC	7	F	H
12	2	11 y	L	C	6	F	H
13	3	12 y	L	C	15	F	NH
14	3	13 y	R	IC	9	F	H
15	3	14 y	L	C	> 24	0	0
16	3	14 y	R	C	2	F	H
17	3	13 y	R	C	5	F	H
18	3	15 y	R	IC	9	F	NH
19	3	15 y	L	IC	2	F	H
20	3	14 y	R	C	5	F	NH
21	3	12 y	R	IC	4	F	H
22	3	15 y	L	C	6	F	H
23	3	14 y	R	IC	6	F	H
24	3	15 y	L	C	>24	0	0
25	3	12 y	R	C	9	0	0
26	3	13 y	R	C	>24	0	0
27	3	14 y	L	IC	5	F	NH
28	3	13 y	L	C	10	0	0

C = complete, d = days, Dev. = developmental, F = fixation, H = homogenous, hrs = hours, IC = incomplete, L = left, NH = non-homogenous, O = orchiectomy, R = right, y = years.

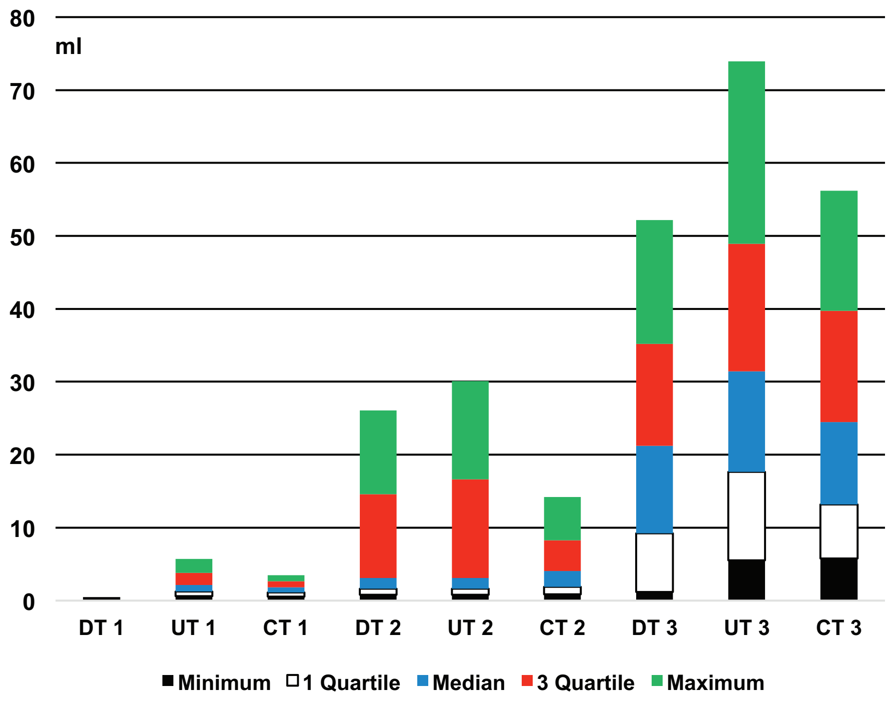

Figure 5. Profile of testicular volume. DT = detorsed testis, UT = uninvolved testis, CT = control testis. 1,2,3-developmental groups.

pubertal group, PSV in the detorsed testis was lower than in the uninvolved testis (X=4.17 cm/s vs X=4.9 cm/s, Z=−1.82, P=.07), and statistically significantly lower than in the control group (X=4.17 cm/s vs X=8.85 cm/s, Z=–2.45, P=.01). PSV in the uninvolved testis was lower than in the control group (X=4.9 cm/s vs X=8.85 cm/s, Z=–1.27, P=.20). In the pubertal group, PSV in the detorsed testis was statistically significantly lower than in the uninvolved testis (X=6.7 cm/s vs X=6.9 cm/s, Z=–3.03, P=.002) and in the control group (X=6.7 cm/s vs X=15.06 cm/s, Z=–4.19, P=.00003). PSV was also statistically significantly lower in the uninvolved testis than in the control group (X=6.9 cm/s vs X=15.06 cm/s, Z=–4.08, P=.00004) (Fig. 6).

3.3. EDV in the capsular artery

In the pre-pubertal group, no statistically significant differences were found between EDV in the uninvolved testis and in the control group (X=1.58 cm/s vs X=1.53 cm/s, Z=0.12, P=.90). In the early pubertal group, EDV was greater in the testis in the control group than in the detorsed testis (X=2.73 cm/s vs X=1.7 cm/s, Z=–0.93, P=.35) and in uninvolved testis (X=2.73 cm/s vs X=1.9 cm/s, Z=–0.75, P=.46) respectively. EDV was lower in the detorsed testis than in the uninvolved testis (X=1.7 cm/s vs X=1.9 cm/s, Z=–0.74, P=.46). In the pubertal group, EDV in the detorsed testis was statistically significantly lower than in the uninvolved testis (X=2.21 cm/s vs X=2.47 cm/s, Z=−1.99, P=.046) and in the control group (X=2.21 cm/s vs X=5.09 cm/s, Z=–4.14, P=.00004). EDV was also statistically significantly lower in the uninvolved testis than in the control group (X=2.47 cm/s vs X=5.09 cm/s, Z=–3,72, P=.0002) (Fig. 7).

3.4. RI in the capsular artery

In the pre-pubertal group, RI was lower in the uninvolved testis than in the control group (X=0.61 vs X=0.7, Z=−1.80, P=.07). In the early pubertal group, RI was lower in the detorsed testis than in the uninvolved testis (X=0.59 vs X=0.62, Z=−1.68, P=.09) and statistically significantly lower than in the control group (X=0.59 vs X=0.71, Z=–2.03, P=.04). RI was lower in the uninvolved testis than in the control group (X=0.62 vs X=0.71, Z=−1.69, P=.09). In the pubertal group, no statistically significant differences in RI values were found: the detorsed testis versus the uninvolved testis (X=0.67 vs X=0.63, Z=−1.17, P=.24), the detorsed testis versus the control group (X=0.67 vs X=0.66, Z=−1.16, P=.25), the uninvolved testis versus the control group (X=0.63 vs X=0.66, Z=–0.74, P=.46) (Fig. 8).

3.5. PSV in the intra-testicular artery

In the pre-pubertal group, PSV in the uninvolved testis was statistically significantly greater than in the control group (X=3.87 cm/s vs X=2.49 cm/s, Z=–2.08, P=.04). In the early pubertal group, no statistically significant differences in PSV values were found: the detorsed testis versus the uninvolved testis (X=3.07 cm/s vs X=2.9 cm/s, Z=0.27, P=.79), the detorsed testis versus the control group (X=3.07 cm/s vs X=3.15 cm/s, Z=0.08, P=.93), the uninvolved testis versus the control group (X=2.9 cm/s vs X=3.15 cm/s, Z=–0.59, P=.55). In the pubertal group, PSV in the detorsed testis was statistically significantly lower than in the uninvolved testis (X=3.73 cm/s vs X=4.41 cm/s, Z=–3.65, P=.0003) and in the control group (X=3.73 cm/s vs X=9.7 cm/s, Z=–4.19, P=.00003). PSV was also statistically significantly lower in the uninvolved testis than in the control group (X=4.41 cm/s vs X=9.7 cm/s, Z=–4.19, P=.00003) (Fig. 9).

3.6. EDV in the intra-testicular artery

In the pre-pubertal group, EDV in the uninvolved testis was greater than in the control group (X=1.89 cm/s vs X=1.2 cm/s,

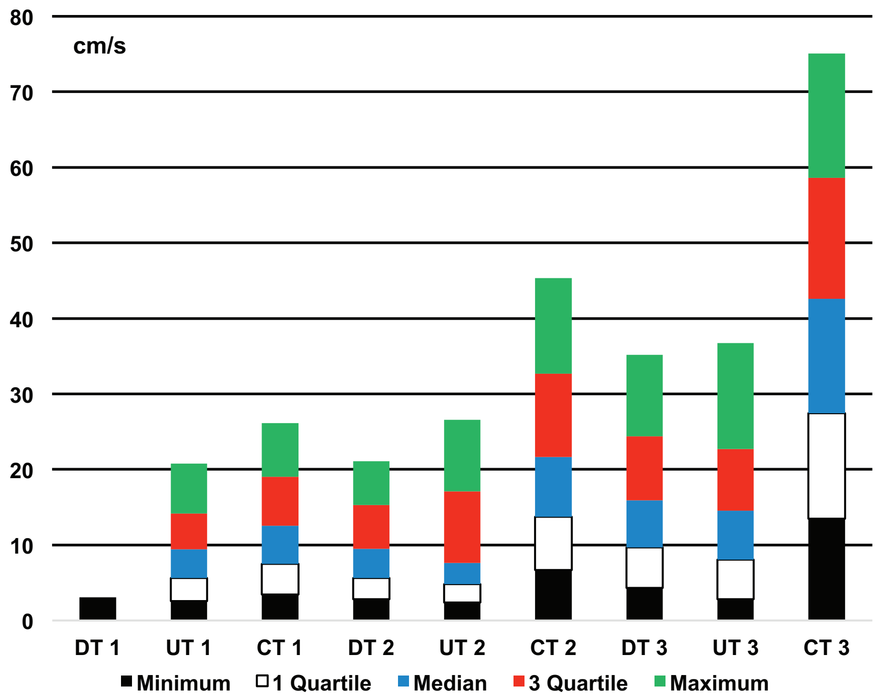

Figure 6. Peak-systolic velocity in the capsular artery. DT=detorsed testis, UT=uninvolved testis, CT=control testis. 1,2,3-developmental groups.

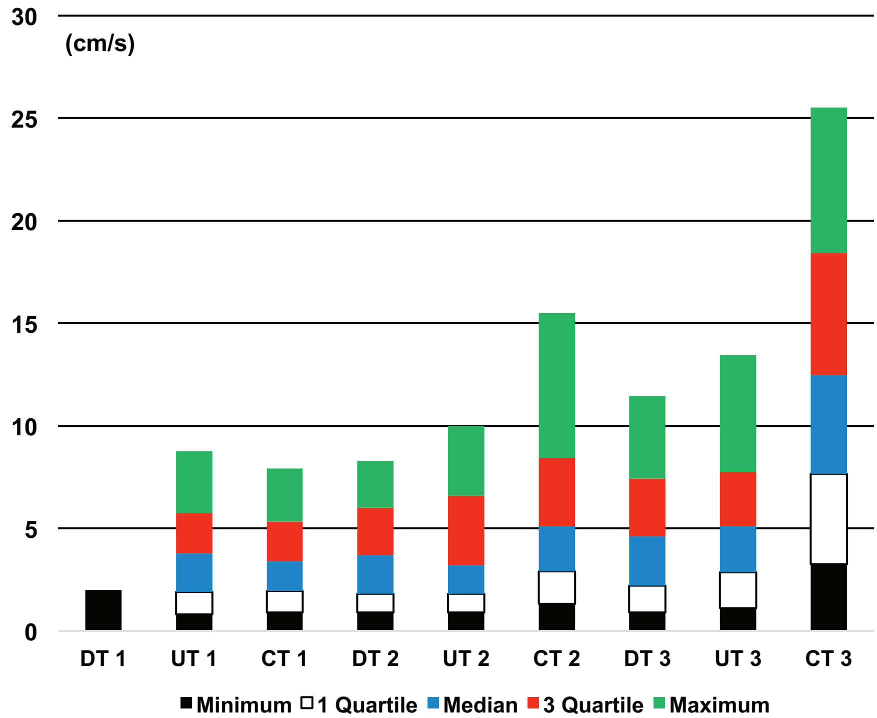

Figure 7. End-diastolic velocity in the capsular artery. DT=detorsed testis, UT=uninvolved testis, CT=control testis. 1,2,3-developmental groups.

$Z=0.75$, $P=.49$). In the early pubertal group, no statistically significant differences in EDV values were found: the detorsed testis versus the uninvolved testis ($X=1.37$ cm/s vs $X=1.27$ cm/s, $Z=0.11$, $P=.99$), the detorsed testis versus the control group ($X=1.37$ cm/s vs $X=1.37$ cm/s, $Z=0.10$, $P=.99$), the uninvolved testis versus the control group ($X=1.27$ cm/s vs $X=1.37$ cm/s, $Z=0,75$, $P=.49$). In the pubertal group, EDV in the detorsed testis was statistically significantly lower than in the uninvolved testis ($X=1.62$ cm/s vs $X=1.89$ cm/s, $Z=-3.56$, $P=.0004$) and in the control group ($X=1.62$ cm/s vs

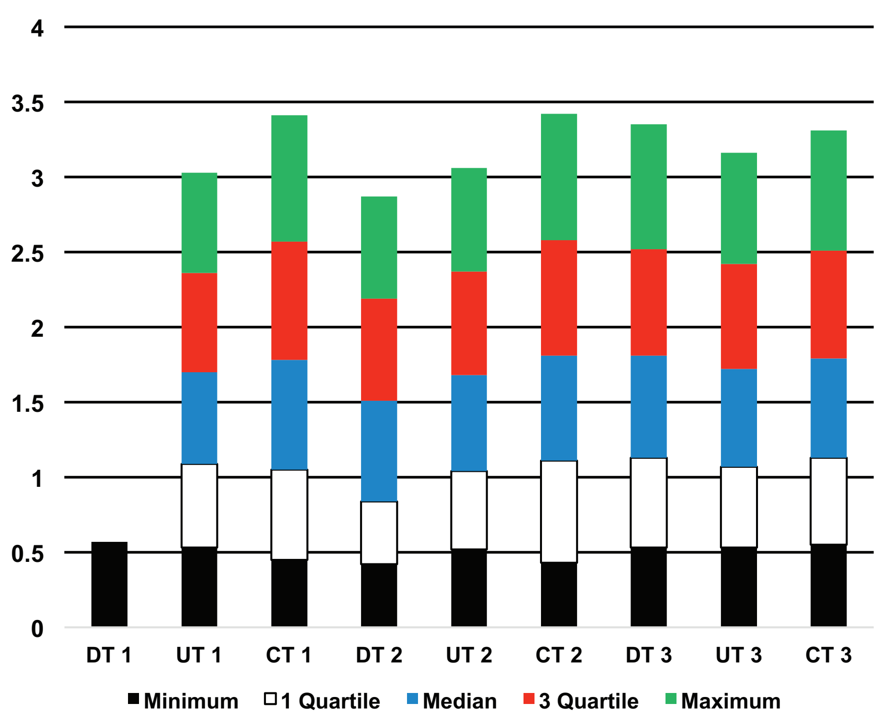

Figure 8. Vascular resistance index in the capsular artery. DT=detorsed testis, UT=uninvolved testis, CT=control testis. 1,2,3-developmental groups.

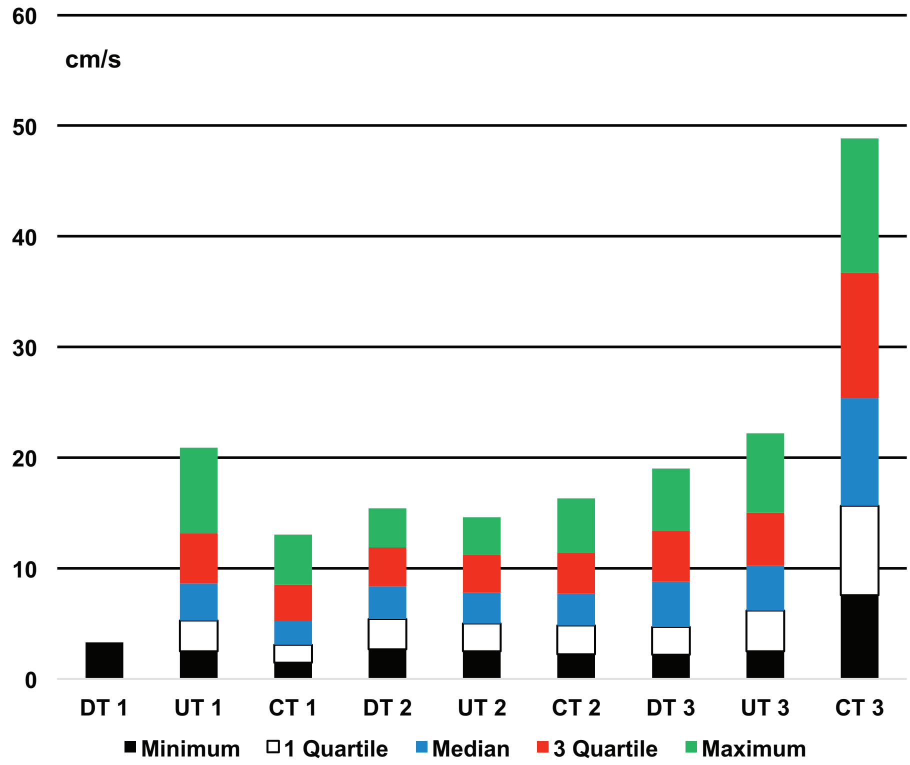

Figure 9. Peak-systolic velocity in the intra-testicular artery. DT=detorsed testis, UT=uninvolved testis, CT=control testis. 1,2,3-developmental groups.

$X=3.46$ cm/s, $Z=-3.55$, $P=.00004$). EDV was also statistically significantly lower in the uninvolved testis than in the control group ($X=1.89$ cm/s vs $X=3.46$ cm/s, $Z=-2.04$, $P=.04$) (Fig. 10).

3.7. RI in the intra-testicular artery

The pre-pubertal group showed no statistically significant differences between RI in the uninvolved testis and in the control group ($X=0.52$ vs $X=0.53$, $Z=0.75$, $P=.49$). In the early-

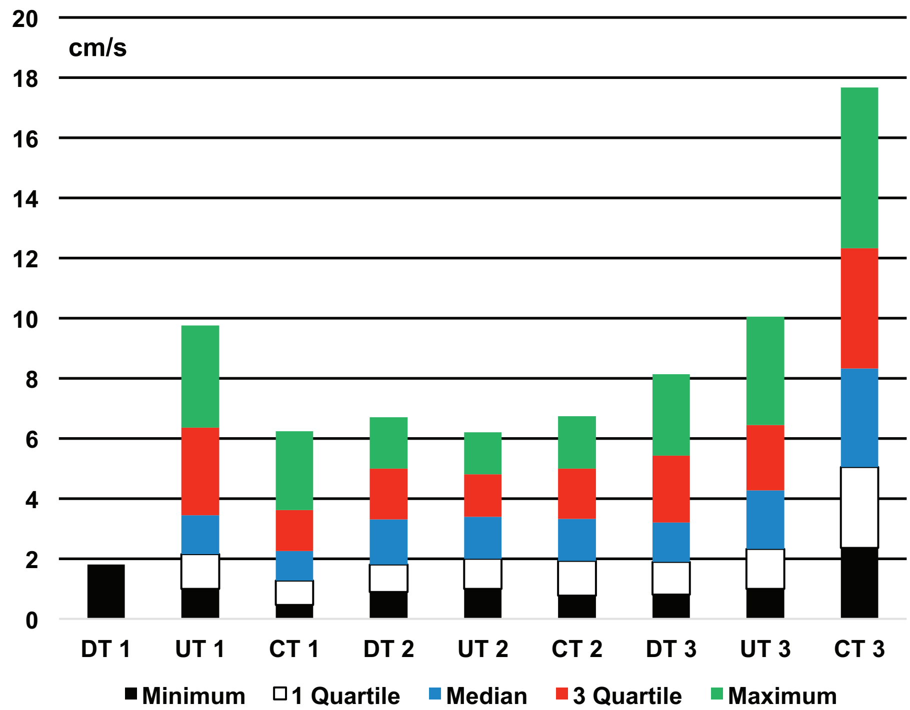

Figure 10. End-diastolic velocity in the intra-testicular artery. DT=detorsed testis, UT=uninvolved testis, CT=control testis. 1,2,3-developmental groups.

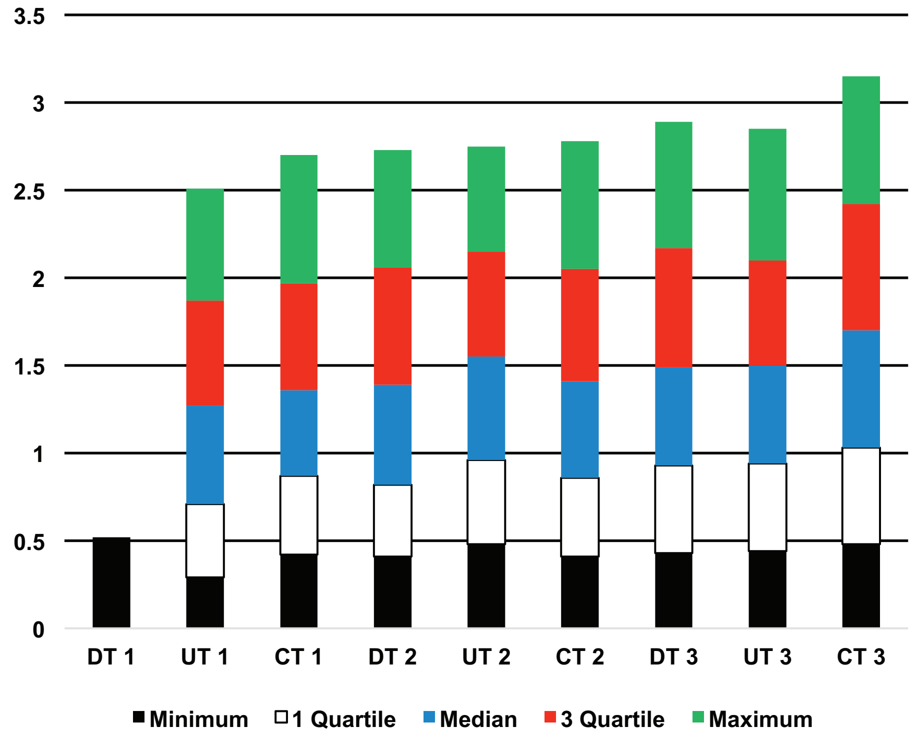

Figure 11. Vascular resistance index in the intra-testicular artery. DT=detorsed testis, UT=uninvolved testis, CT=control testis. 1,2,3-developmental groups.

pubertal group, no statistically significant differences in RI values were found: the detorsed testis versus the uninvolved testis ($X=0.55$ vs $X=0.56$, $Z=0.09$, $P=.99$), the detorsed testis vs the control group ($X=0.55$ vs $X=0.55$, $Z=0.10$, $P=.99$), the uninvolved testis vs the control group ($X=0.56$ vs $X=0.55$, $Z=0.25$, $P=.80$). In the pubertal group, no statistically significant differences in RI values were found: the detorsed testis versus the uninvolved testis ($X=0.58$ vs $X=0.56$, $Z=-0.56$, $P=.57$), the detorsed testis versus the control group ($X=0.58$ vs $X=0.64$, $Z=-1.62$, $P=.11$), the uninvolved testis versus the control group ($X=0.56$ vs $X=0.64$, $Z=-1.79$, $P=.07$) (Fig. 11).

4. Discussion

Testicular torsion in boys involves more frequently the left testis, rarely both testes.[2,6,10,13,16] In our study it occurred with the same frequency on the right and left side. TT most often affects boys in the neonatal and adolescent periods.[4,6,8,11] This is also confirmed by the current research.

Scrotal US with the Power or Color Doppler option is currently the standard diagnostics of TT. According to Pepe et al[31] US exam should be performed in every patient with an "acute scrotum" for medical and legal reasons. Surgical exploration of the scrotum must be done in cases of suspected testicular torsion in spite of normal result of preoperative US exam. Preoperative US with evaluation of testicular blood supply was always performed in our patients.

Bandarkar and Blask[11] recommend an attempt to detorse the testis percutaneously after the administration of analgesics, while awaiting for the surgery. TT usually takes place in the medial direction, but one has to consider the possibility of reverse rotation and various anomalies of the testicular region. This is why such treatment is not recommended by our group.

According to Bennett et al,[14] failure of testicular torsion treatment is primarily due to late reporting of symptoms to the appropriate specialist. However, it should be stressed that older boys usually manifest their ailments immediately after they start, so the percentage of orchiectomies is lower in them than in younger boys.[16] The results of our research confirm these observations.

Access to the torsed testis depends on knowledge and experience of the surgeon. Median raphe incision is recommended by Yang et al[2] and Tajchner et al.[42] Lorenzo et al[6] and Boettcher et al[8] prefer transverse scrotal incisions, separate for each testis. Günther and Rübben[1] and Arda and Ozyaylali[37] write about inguinal incision. Only Günther explains that inguinal access is good in neonates and infants due to supravaginal testicular torsion, more common in this developmental period. In our opinion transverse, separate scrotal incisions give good insight and opportunity of easy manipulation with testes, as well as a form of surgical "sterility" for the uninvolved testis, especially if there is a massive inflammatory-congestive process or necrosis of the torsed testis.

During the operation, the degree of torsion is assessed, and the testis should be detorsed and warmed up, as this speeds up the return of blood flow.[2,37] The gonadal blood supply assessment and surgical procedures described in the literature are diversified (Table 2). Our management is broadly described in the chapter "Material and methods" and is based on many years of experience in our department.

In the literature, there are discrepancies between the authors regarding the time that must pass from the beginning of a

Table 2

Assessment of blood supply and surgical management in testicular torsion according to references.

Author	Management
Arda and Ozyaylali[37]	Three grades of bleeding after detorsion and incision of the testis: 1st grade—arterial bleeding within 10 minutes of the incision, 2nd grade—arterial bleeding after 10 minutes of the incision, 3rd grade—no arterial bleeding after 10 minutes. Testes assessed as 3rd grade are removed.
Boettcher et al[8]	Testes dark or black after detorsion are removed.
Chu et al[4]	Detorsion and incision of the testis. Tunica vaginal flap (TVF) technique if arterial bleeding is present—the flap from vaginal casing is sutured into the incision, which reduces intra-testicular hypertension and stabilizes the testis. In cases of traces of venous bleeding or no bleeding—orchiectomy.
Grimsby et al[5]	Detorsion of the testis. 6-grade color scale from normal pink to black testis. All gonads saved.
Günther and Rübben[1]	They remove only these testes that show demonstrably macroscopic necrosis.
Yang et al[2]	Assessment of testicular blood flow 20 minutes after detorsion, incision only in cases of doubt, orchiectomy only in cases of no bleeding.
All above authors	Use non-absorbent threads for fixation, recommend and do simultaneous fixation of the second testis for prophylactic purposes.

complete torsion to necrosis of the testis: Bandarkar and Blask[11]—4 hours, Al-Hunayan et al[16]—12 hours, Benedetto et al[38]—15 hours, Abdul et al[9]—24 hours, Chu et al[4]—48 hours, Yang et al[2]—48 hours. On the basis of own research, it was found that 9 hours of a complete torsion is enough for testicular necrosis.

In testicular torsion, not only the germ cells are damaged, but the hormonal apparatus in the form of Leydig cells is also affected. According to Arda and Ozyaylali,[37] after 10 hours of torsion, these cells are irreversibly damaged. This somewhat contradicts the report by Arena et al,[10] which recommends that both gonads should be detorted and fixed in cases of bilateral fetal TT, because of the chance of some Leydig cells to survive. Zerin et al[13] think that orchiectomy in cases of fetal torsion and necrosis, confirmed with ultrasound exam, can be performed on a scheduled basis. In our study, 5 newborns were operated on immediately because US was not sensitive enough to clearly determine the condition of testes.

According to Günther and Rübben[1] and Arena et al,[10] TT activates a cascade of cytokines, which can damage both the ill and the uninvolved testis. Laboratory experiments have shown that a compound called N-acetylcysteine has a protective effect on the torsed testis and may have a future therapeutic use.[1]

According to the literature, follow-up US after detorsion of the testis is performed at different intervals: from 1 month to 7 years.[2,5,10,16,37] According to our experience 1 year of observation is a long enough time to assess the development of gonads after TT, although the optimal solution would be US study after the age of 18. Unfortunately, these patients are already under the care of specialists for adults.

Only Arena et al[10] raises the problem of echogenicity of the testicular parenchyma after detorsion. His study states that it was heterogeneous in some cases. In our study, heterogeneous echogenicity was recorded in 33% of cases of rescued testes and did not show dependence on the type of TT nor its duration.

The literature gives different methods of assessing the development of the testis after detorsion surgery. Al-Hunayan et al[16] determines atrophy on the follow-up US if the volume of the detorsed testis is <50% of the volume of the uninvolved testis. According to Chu et al,[4] atrophy simply means a reduction in the size of the detorsed testis compared the uninvolved one based on physical examination or US. Grimsby et al[5] determines atrophy on the follow-up US exam if the volume of the detorsed testis is <80% of the volume of the uninvolved testis. According to Yang et al,[2] 1/3 of patients have atrophy of the detorsed testis, and in each case the uninvolved testis shows compensatory hypertrophy.

The above publications do not refer to standards of testicular volume or control groups, and testicular atrophy is a relative concept if such a large discrepancy as 50% to 80% is assumed. In addition, it is uncertain whether the uninvolved testis is a healthy organ if it is known that TT activates cytokines that can damage both gonads.[1,10]

We did not use the concept of atrophy, but precisely determined and compared volumes of the detorsed, uninvolved and the control group testes with statistical conclusions. Our results indicate to abnormal development of the testis after detorsion and compensatory hypertrophy of the uninvolved testis regardless of the surgery—orchiectomy or detorsion. Developmental disturbances in the form of a smaller volume of the detorsed testis compared with the uninvolved and control ones are independent of the duration and type of TT.

Only 3 publications give standards for flow parameters in testicular microcirculation, including 1 item in boys. According to Bandarkar and Blask,[11] the normal vascular resistance index in the male intra-testicular artery is 0.48 to 0.75 (mean 0.62). According to Dudea et al,[17] the RI standard in the capsular and intra-testicular artery in boys with a testicular volume <4 mL is 0.87 on average and >4 mL is 0.57 on average. Middleton et al[39] reports the following testicular flow standards for men aged 28 to 35 years: the capsular artery PSV: 5.0 to 23.4 cm/s (mean 11.9 cm/s), EDV: 1.8 to 9.2 cm/s (mean 4.0 cm/s), RI: 0.46 to 0.78 (mean 0.66), the intra-testicular artery PSV: 4.0 to 19.1 cm/s (mean 9.7 cm/s), EDV: 1.6 to 6.9 cm/s (mean 3.6 cm/s), and RI: 0.48 to 0.75 (mean 0.62). In general, microcirculation in the normal testis has high-flow and low-resistance pattern.

Preoperative ultrasound in boys with TT were performed only by Pepe et al,[31] the average age of patients was 9 years. In the intra-testicular artery, PSV was 0 to 2 cm/s (mean 1.5 cm/s) and EDV was 0 to 1 cm/s (mean 0.5 cm/s).

In the available literature there are no data on US of microcirculation in the testis after operation due to TT in boys. Therefore, in order to understand the changes that occur within the testicular arterial network after surgeries performed in its vicinity, it is necessary to present references on the treatment of varicocele and inguinal hernia.

In publications of Akand et al[18] and Ener et al,[19] varicose vein ligation increases PSV and EDV, as well as reduces RI in the capsular and intra-testicular arteries. This should be seen as an indicator of improved blood microcirculation in the testis.

When examining patients operated on due to inguinal hernia, David and Goldenberg[24] and Stula et al[28] found no significant late abnormalities of PSV, EDV, and RI in the capsular and intra-testicular arteries. However, according to Çelebi et al[22] and Palabiyik et al,[26] a statistically significant increase in RI and decrease in EDV in these vessels indicates an early, transient testicular blood supply disorder after hernia surgery associated with vasospasm caused by surgical trauma.

Our research with use of US spectral Doppler shows that PSV and EDV in the capsular and intra-testicular arteries of the detorsed testis were lower than in the uninvolved and control testes, which indicates damage of the gonad, but RI was not increased. Analyzing abnormal blood flow parameters in the microcirculation of the uninvolved testis, we are submitting for discussion the theory of the occurrence of unknown circulatory disturbances caused by torsion or the earlier existing pathologies, for example in the form of testicular dysplasia.

The diagnostics of TT is still being extended with new research methods. According to Yamaguchi et al,[40] US with contrast significantly improves the imaging of vascular disorders in TT before surgery and in follow-up studies. Sun et al[7] introduces US with elastography, which precisely determines the elasticity of the testicular parenchyma disturbed by torsion, describing an increased value of Young modulus. The next step is the introduction of scintigraphy, which is much more sensitive in detecting TT than US examination, with a sensitivity of close to 100%.[3,9,41,42] Unfortunately, the study is time-consuming, uses ionizing radiation, and not always available. Rivers et al,[3] recommending the determination of levels of interleukin 6 in the differentiation of epididymitis from TT, indicates the need for the development of laboratory diagnostics. An innovative method in the diagnostics of TT is scrotoscopy with use of neonatal cystoscope described by Ye et al[43] and Yin et al.[44]

It should be stated that patients after surgery due to TT require many years of specialistic care with imaging and laboratory examinations, including semen assessment.

Strengths of our study: a novel, comprehensive study, assessing the influence of torsion on testicular echogenicity, volume, and vascularisation, depending on the degree and duration of torsion, and patients' age. It presents comparisons of detorsed testes with uninvolved testes and the control group.

Limitations of our study: the lack of a preoperative assessment of peak-systolic velocity, end-diastolic velocity, and vascular resistance index due to patient's anxiety and pain, the necessity to perform a quick surgery, and the relatively small size of the study and control groups.

5. Conclusions

There is no clear correlation between the type of testicular torsion, its duration and the echogenicity of the testis.

Testicular torsion has a negative effect on the volume of detorsed testis with compensatory hypertrophy of the uninvolved testis.

The study represents a new approach to the issue of long-term gonadal blood supply abnormalities after treatment of testicular torsion in childhood.

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