Cryptorchidism: medical and surgical treatment in the 1st year of life

Abstract Since cryptorchidism can cause infertility and early orchiopexy can improve fertility, we tried to determine whether medical and surgical treatment in the 1st year of life can improve testicular fertility. We concluded that this is the best time to treat cryptorchid tests.

Key words Cryptorchism · Undescended testes · Treatment

Introduction

Cryptorchidism is an important cause of infertility [1–3]: cryptorchid testes show histologic alterations progressing from birth [4]. Many studies have demonstrated that early orchiopexy can improve fertility [5, 6]. The aim of our study was to evaluate whether medical and surgical treatment in the 1st year of life can improve testicular fertility.

Materials and methods

We treated 127 patients with 154 cryptorchid testes. Only full-term infants without other pathologies were included. The age range was between 6 months and 3 years. There were 104 unilateral and 50 bilateral cases. In group A, 30 testes were operated upon in the 1st year of life without previous medical treatment. Groups B–D received medical treatment before surgery: luteinizing hormone-releasing hormone (LHRH) nasal spray 200 μg in each nostril 3 times per day for 4 weeks, followed by human chorionic gonadotropin (HCG) 500 IU 3 times a week for 3 weeks.

The various testicular positions at surgery were defined as: abdominal when the testis was in the intraperitoneal position; intracanalicular when it was between the internal and external inguinal ring; and extracanalicular when it was outside the external inguinal ring. Ectopic testes were excluded. All patients were examined by the same pediatric endocrinologist and pediatric surgeon. Treatment was started after 6 months of life when the period of spontaneous testicular descent had passed [7]. Testicular dimensions were measured with a caliper and the volume was calculated using an ellipsoid formula: \(4/3 \cdot L/2 \cdot H/2 \cdot W/2\) (\(L = \) length, \(H = \) height, \(W = \) width).

Testicular biopsy specimens were fixed in 3% glutaraldehyde and embedded in Epon according to the method published previously [8]. Semi-thin sections were examined by the same pathologist without knowing the age and treatment of the subject. Each biopsy was studied under 50x magnification, and all spermatogonia and Ad spermatogonia on the slide were counted and divided by the number of tubules present. At least 30 tubules were identified on each biopsy specimen. Mean and standard deviation (SD) values of total spermatogonia and Ad spermatogonia numbers were compared with previous published data [8, 9].

Statistical analyses were performed by variance analysis, chi-square test, Kruskal-Wallis test, Spearman’s rank correlation, and linear regression and correlation. Rank values for clinical (I), surgical (II a and b), and histologic (III a and b) findings were graded as follows:

I Clinical: prescrotal location of the testis = 1, inguinal = 2, nonpalpable = 3.
IIa. Surgical: extracanalicular = 1, intracanalicular = 2, abdominal = 3.
IIb. Epididymal morphology: normal = 1, epididymis attached to the testis by its caput and tail = 2, attached only by its caput = 3, totally separate = 4.
IIia. Histologic appearance (tubular atrophy): no atrophy = 1, mild atrophy (collapsed tubuli with no atrophy of Sertoli cells) = 2, severe atrophy (partial degeneration of Sertoli cells in the tubulus with pronounced peritubular hyalinosis or fibrosis) = 3.
IIib. Leydig-cell atrophy: normal according to age = 1, mild (diminution of cytoplasm and increase in nuclear-membrane indentation) = 2, severe (almost no juvenile Leydig cells within the interstitium and, if observed, Leydig cells with minimal cytoplasm) = 3.

Results

According to age and treatment modality, our patients were divided into 4 groups: group A: 27 patients with 30 testes treated surgically in the 1st year of life; group B: 40 patients with 52 testes treated medically and surgically in the 1st year of life; group C: 38 patients with 45 testes treated medically and surgically in the 2nd year of
life; and group D: 22 patients with 27 testes treated medically and surgically in the 3rd year of life. Group A consisted of patients whose parents refused hormonal therapy. Although patients were not been randomized, but were biased by parent request, our data are statistically significant and remain very interesting.

The various testicular positions were equally present in the 4 groups. In evaluating the connection between the testis and epididymis (Fig. 1), we found normal morphology in 28 patients (18.4%), the epididymis attached to the testis by its caput and tail in 79 (51.2%), attached only by its caput in 43 (27.9%), and complete disjunction in 4 (2.5%). We found a positive correlation between the anatomic position of the testis and epididymal malformations (Fig. 2, Spearman: $P < 0.005$, $r = 0.28$). Extracanalicular, intracanalicular, abdominal

Tubular atrophy was found in 90 testes (58.4%) and correlated positively with age at surgery (Fig. 4 Spearman: $P < 0.001$, $r = 0.59$) and inversely with number of germ cells (Fig. 5, Spearman: $P < 0.001$, $r = 0.62$). Atrophy of Leydig cells was found in 134 testes (87%) and correlated inversely with the number of germ cells.