INTRODUCTION
Congenital idiopathic clubfoot is a complex congenital deformity and ranges approximately 40% in the structure of congenital diseases of the lower extremities in children [1,2]. The frequency of congenital clubfoot is approximately from 0.6 to 3 cases per 1000 live births [3]. It is three times more common in males. In 30-50% present bilateral lesion of the feet. Combination with other congenital defects (hip dysplasia, infant torticollis etc.) is observed in about 10% [4,5]. Congenital clubfoot can be an idiopathic malformation or accompany systemic diseases, arthrogryposis, diastrophic dysplasia, Freeman-Sheldon syndrome, Larsen syndrome [6].

The anomaly occurs in the 3rd month of intrauterine life and is characterized by dysfunction of the posterior and medial aspects of the lower leg, ankle and foot [7,8]. The muscles are smaller and there is increased collagen synthesis resulting in fibrosis in the posteromedial tarsal ligaments, deep fascia, Achilles tendon and tibialis posterior tendon. Congenital clubfoot develops under the influence of endogenous and exogenous pathological factors. Basic theories of clubfoot development: mechanical, embryonic, neuromuscular. Mechanical theory, which postulates that clubfoot results from an elevated intrauterine pressure during pregnancy. Embryonic theory associated with toxicosis during pregnancy, viral infection, toxoplasmosis, avitaminosis during embryogenesis. Some authors suggested that the etiology of clubfoot is neuromuscular in origin and has genetic implications. A genetic basis for isolated clubfoot is supported by the fact approximately 25% of all patients with isolated clubfoot report a positive family history for clubfoot [9]. The role for genetic factors in clubfoot is also supported by a twin study that demonstrates a higher concordance rate for identical twins compared to fraternal twins (33% versus 3%). Further evidence for a genetic basis for clubfoot is the differences in clubfoot prevalence across ethnic populations, with the lowest prevalence in Chinese (0.39 cases per 1000 live births) and highest in the Hawaiians and Maoris (7 per 1000) [10,11]. The neuromuscular etiological concepts in congenital clubfeet yield important information regarding recurrent deformities, especially dynamic supination [12,13,14].

For almost 3 decades prior to 2010, we treated congenital clubfoot with manipulation (following the modified T. Zatsepin protocol, 1947) [15] and casting from 3-4 and up to the age of 6-8 months followed by modified posteromedial release, subtalar or/and plantar incision. Since 2010, we started to use Ponseti method and serial casting from first days after birth.

THE AIM
The aim of this study was to analyze the outcome, recurrence rate and complications between Ponseti method and soft-tissue release 3 years after the initial treatment.

RESULTS OF TREATMENT OF IDIOPATHIC CONGENITAL CLUBFOOT IN CHILDREN: A 3-YEAR FOLLOW UP STUDY

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ABSTRACT
The aim: Was to analyze the outcome, recurrence rate and complications between Ponseti method and soft-tissue release 3 years after the initial treatment.

Materials and methods: This prospective cohort study was conducted in congenital idiopathic clubfoot patients who underwent primary treatment by either Ponseti serial casting or soft tissue release between 2006 to 2016 at department of traumatology and orthopedics National Children’s Specialized Hospital “Okhmatdet”. Total of 113 feet in 95 patients (61 males and 34 females), sixty-two feet (62 patients) were in the Ponseti group and thirty-three feet (33 patients) were in the surgical treatment group. For both groups, descriptive statistics were calculated Pirani score (2004) result before and 3 years after treatment, recurrence rate and complications. The comparison of the Pirani score result and complications between the two groups was analyzed by nonparametric tests (Mann-Whitney U-tests). Statistical data processing was performed in SPSS 17.0 program.

Results: The results of Pirani score reveal satisfactory outcomes for both groups. But Ponseti method has the more conservative approach and lower complication rate (11,29±5,27% and 24,24±11,74%, p=0,52).

Conclusions: Ponseti method is a safe, effective method for treatment of congenital idiopathic clubfoot in children from first days after birth. Open surgery should be reserved for deformity that cannot be completely corrected or for treatment of recurrences.

KEY WORDS: clubfoot, treatment, Ponseti method, Zatsepin protocol, surgical treatment
RESULTS AND DISCUSSION

Patients in the soft tissue surgical treatment group were treated with manipulation, serial casting by modified Zatsepin technique [15] then underwent one or more of the following procedures such as posteromedial release and plantar release with modified Carrol incision and plantar incision for cavus deformity correction (average age at the time of surgery, 8.5 months; range, 4.7–11.4 months). After correction, the talonavicular and the talocalcaneal joints were pinned using smooth three Kirschner wires and toe to groin plaster casts were applied. Preventive antibiotics were given intravenously for 5 days. The average hospital stay was 14 days (range, 12–15 days). There were no neurovascular complications or skin problems in any of the cases. Kirschner wires were removed 12 weeks after surgery. Orthotic management was applied after removed of the Kirschner wires and included shoes with insoles and nighttime splinting. All patients of both groups were followed up for at least three years.

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MATERIALS AND METHODS

This prospective cohort study was conducted in congenital idiopathic clubfoot patients who underwent primary treatment by either Ponseti serial casting or soft tissue release between 2006 to 2016 at department of traumatology and orthopedics National Children's Specialized Hospital “Okhmated”. Clubfeet in both groups were of comparable severity at the start of the treatment as documented by the scores of Pirani et al. (2004) [16].

Inclusion criteria – healthy infants younger than 2 weeks born with clubfoot/clubfeet and without any other congenital anomalies.

Exclusion criteria included non-congenital idiopathic clubfoot, such as postural clubfoot, neuropathic or another syndromic clubfoot, and incomplete medical data.

Treatment of patients in the Ponseti group followed the standart protocol (1963) [17,18]. Cast applications were performed on an outpatient basis with weekly changes. In all cases, a persistent hindfoot equinus made a tenotomy of the tendo-Achilles under local anesthesia.

The tenotomies were performed according to the method described by Ponseti using a No. 11 blade, and no neurovascular compromise was experienced. After tenotomy of the tendo-Achilles, plaster casts were applied for 3 weeks. Orthotic management for abduction and external rotation of the feet started once correction of the deformity was achieved (braces with 70 external rotation for clubfoot and to 45 external rotation for the normal foot in unilateral cases).

Patients in the soft tissue surgical treatment group were treated with manipulation, serial casting by modified Zatsepin technique [15] then underwent one or more of the following procedures such as posteromedial release and plantar release with modified Carrol incision and plantar incision for cavus deformity correction (average age at the time of surgery, 8.5 months; range, 4.7–11.4 months). After correction, the talonavicular and the talocalcaneal joints were pinned using smooth three Kirschner wires and toe to groin plaster casts were applied. Preventive antibiotics were given intravenously for 5 days. The average hospital stay was 14 days (range, 12–15 days). There were no neurovascular complications or skin problems in any of the cases. Kirschner wires were removed 12 weeks after surgery. Orthotic management was applied after removed of the Kirschner wires and included shoes with insoles and nighttime splinting. All patients of both groups were followed up for at least three years.

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Table I. Pirani scores before and three years after treatment.

<table>
<thead>
<tr>
<th>Pirani score</th>
<th>Start of the treatment</th>
<th>Three years after treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Surgical treatment</td>
<td>Ponseti group</td>
</tr>
<tr>
<td></td>
<td>group (n=33)</td>
<td>(n=62 surgery)</td>
</tr>
<tr>
<td>0 – 0,5</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>0,5 – 1,5</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>1,5 – 2,5</td>
<td>3 (9,09±3,24%)</td>
<td>12 (8,35±3,81%)</td>
</tr>
<tr>
<td>2,5 – 5,0</td>
<td>12 (6,37±2,61%)</td>
<td>24 (18,71±9,69%)</td>
</tr>
<tr>
<td>5,0 – 6,0</td>
<td>18 (9,54±4,81%)</td>
<td>26 (17,94±7,19%)</td>
</tr>
</tbody>
</table>

Table II. Complications associated with treatment.

<table>
<thead>
<tr>
<th>Complications</th>
<th>Surgery group n = 33</th>
<th>Ponseti group n = 62</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cast complication</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cast loosening</td>
<td></td>
<td></td>
<td>17 (27,41%)</td>
</tr>
<tr>
<td>Cast-associated pressure sore</td>
<td>3 (9,09%)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Surgical complication</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infection</td>
<td>2 (6,06%)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Wound edge necrosis</td>
<td>3 (9,09%)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Recurrence</td>
<td>8 (24,24%)</td>
<td>7 (11,29%)</td>
<td>0,52</td>
</tr>
</tbody>
</table>

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Conclusions

Ponseti method is a safe, effective method for treatment of congenital idiopathic clubfoot in children from first days after birth. After analysis of the results, we changed the standard treatment of congenital clubfoot at our hospital to the Ponseti method of treatment. Open surgery should be reserved for deformity that cannot be completely corrected or for treatment of recurrences.

References

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Conflict of interest:
The Authors declare no conflict of interest.

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A – Work concept and design, B – Data collection and analysis, C – Responsibility for statistical analysis, D – Writing the article, E – Critical review, F – Final approval of the article