Introduction. Wilms’ tumour is one of the commonest malignant tumours of childhood. It appears mainly in the first 5 years of life. Incidental examples of nephroblastoma in adults have been described in literature (about 3% of all described cases). There are diagnostic and therapeutic difficulties in that older age group. The preoperative diagnosis of nephroblastoma in adults is difficult because there are no specific radiographic findings that allow to distinguish it from the more common adult renal tumors. Histopathologically, there is no difference between adult and childhood Wilms’ tumor.

Materials and methods. The PubMed database and current literature search was conducted for reports on clinical and histopathological features of nephroblastoma in adults. We also reviewed the literature in terms of treatment strategy, toxicity and prognostic factors.

Results. Up till now, several biological factors have been identified that may be in future new prognostic factors. Modern treatment regiments improved OS in this group of patients (OS rates of 90%). The prognosis remain still worse for about 25% of patients with anaplastic, bilateral and recurrent disease.

Conclusions. Due to the fact that nephroblastoma is a very rare type of cancer, adult patients should be treated in an individual way based on the available schemes used in children. Toxicity in adults is higher than in children.

Key Words: nephroblastoma • adults • diagnosis • treatment • toxicity • prognosis
basis of Wilms’ tumor is complicated. The WT1 gene (11p13) is mutated in 10% of tumors. Changes in 11p, at 7p, 16q, and 1p are also recognized. Classical histopathological pattern is triphasic: blastemal, epithelial, and stromal. Blastemal–predominant Wilms’ tumors are more aggressive than others types and has a poor outcome. Epithelial and stromal kinds represent intermediate risk tumors. Pathological diagnosis of adult nephroblastoma is based on criteria developed by Kilton et. al. that is: the presence of the tumor primarily originating from the kidney; the presence of primitive blastenic spindle or round cell component; the formation of abortive or embryonal tubules or glomerular structures or no area of renal cell carcinoma histopathology; confirmation of the diagnosis in the histopathological tests; and the age above 15 years old [3]. Additional diagnostics such as immunohistochemical staining for the presence of cytokeratin, vimentin, desmin, actin, and WT1 allows to distinguish between other rare cancer types such as: renal sarcoma, mesoblastic nephroma, clear cell sarcoma, or rhabdoid tumor. The WT1 expression is diagnosed in the blastemal and proliferating epithelial tissue, but not in mature stroma and mature epithelial tissue [4]. The classification to one of the three risk groups depends of the histopathologic features of the tumor. It is necessary for choice of adequate treatment schemes. The SIOP histologic classification reflects chemotherapy–induced changes including “regressive” changes. The NWTSG classifies nephroblastoma based on the presence of anaplasia [5, 6]. The revised SIOP classify Wims’ tumor into three risk groups such as low, intermediate, and high risk (Table 3).

TREATMENT

The classification of the tumor to one of the three risk groups allows to use of adequate treatment schemes (Table 3). Radical nephrectomy (the removal of the tumor along with the kidney with the adrenal gland and lymph nodes of the same side) is treatment of choice of one–sided nephroblastoma. According to SIOP, a partial kidney resection (nephron sparing treatment) is only allowed in precisely designated cases such as in the presence of developmental disadvantages in the other kidney, genetically predisposed diseases in which the risk of nephroblastoma development is high, and in patients who only have one kidney [7]. The SIOP strategy does not recommend the nephron sparing surgery in patients with one–sided nephroblastoma without the presence of the above–mentioned criteria [8]. In the NWTSG and COG studies treatment starts by surgical resection of the tumor. Surgical complica-

### Table 1. Staging system for renal tumors according SIOP 2001 protocols (after chemotherapy)

| Stage   | Description                                                                                      |
|---------|-----------------------------------------------------------------------------------------------|
| Stage I | Tumor is limited to the kidney or surrounded with fibrous pseudocapsule. The renal capsule or pseudocapsule may be infiltrated with the tumor, but it does not reach the outer surface. |
|         | – Tumor is completely resected (resección margines „clear“).                                   |
|         | – Tumor may be protruding into the pelvic system and “dipping” into the ureter but not infiltrates its wall. |
|         | – The vessels of the renal sinus are not involved                                               |
|         | – Intrarenal vessel involvement may be present                                                  |
| Stage II| The tumor extends beyond kidney or penetrates through the renal capsule and/or fibrous pseudocapsule into perirenal fat but is completely resected (resección margines „cler“). |
|         | – The tumor infiltrates the renal sinus and/or invades blood and lymphatic vessels outside the renal parenchyma but is completely resected. |
|         | – The tumor infiltrates adjacent organs or vena cava but is completely resected.               |
| Stage III| Incomplete excision of the tumor which extends beyond resection margines                        |
|         | – Any abdominal lymph nodes are involved                                                       |
|         | – Tumor rupture before or intraoperatively                                                      |
|         | – The tumor has penetrated through the peritoneal surface                                       |
|         | – Tumor thrombi present at resection margines of vessels or ureter                             |
|         | – The tumor has been surgically biopsied prior to preoperative chemotherapy or surgery.        |
| Stage IV| Hematogenous metastases (Lung, Liver, bone, brain etc.) or lymph node metastases outside the abdomeno–pelvic region. |
| Stage V | Bilateral renal tumors at diagnosis.                                                           |
| Stage VI| Bilateral renal involvement at the time of initial diagnosis.                                   |

### Table 2. Staging system for renal tumors according NWTSG protocols (before chemotherapy)

| Stage   | Description                                                                                      |
|---------|--------------------------------------------------------------------------------------------------|
| Stage I | Tumor is limited to the kidney and completely resected (resección margines „clear“).           |
|         | – Tumor was not ruptured before or during removal                                                   |
|         | – The vessels of the renal sinus are not involved beyond 2 mm                                      |
|         | – There is no residual tumor apparent beyond the margins of excision                               |
| Stage II| Tumor extends beyond the kidney but is completely excised.                                        |
|         | – No residual tumor is apparent at or beyond the margins of excision                                |
|         | – Tumor thrombus in vessels outside the kidney is stage II if the thrombus is removed en block with the tumor. |
| Stage III| Residual tumor confined to the abdomen.                                                          |
|         | – Lymph nodes in the renal hilum or the periaortic chains                                          |
|         | – Diffuse peritoneal contamination by the tumor.                                                   |
|         | – Implants are found on the peritoneal surfaces                                                   |
|         | – Tumor extends beyond the surgical margins either microscopically or glossy                      |
|         | – Tumor is not completely respectable because of local infiltration into vital structures.        |
| Stage IV| Presence of hematogenous metastases or metastases to distal lymph nodes.                         |
| Stage V | Bilateral renal involvement at the time of initial diagnosis.                                     |
ations such as bowel obstruction (5.1%), extensive hemorrhage, wound infection (1.9% each), extensive vascular injuries (1.4%) and injuries to other visceral organs (1%) were observed in NWTS – 4 study. The risk factor of surgical complications were intravascular extension into the inferior vena cava, the atrium, or both, a tumor diameter greater than 10 cm [9].

Systemic treatment

The most effective chemotherapeutics in treatment of nephroblastoma are: actinomycin D (ACT), vincristine (VCR), doxorubicin (ADM), cyclophosphamide (ctx), ifosfamide (IFO), etoposide and carboplatin (as in monotherapy as in drug combination). The treatment schemes of nephroblastoma according to NWTS and SIOP were presented in Table IV. According SIOP strategy preoperative chemotherapy reduces the risk of tumor rupture during surgery and thereby reduce the probability of local and distant recurrence (recurrence and its treatement strategy will be shown later) [5, 6]. NWTS recommends polchemotherapy (ACT, VCR, ADM) for a period of 15 weeks in adjuvant treatment in tumor stage III. Less aggressive treatment using two medications (VCR and ACT) can be used in cancer stages I and II [10, 11]. No advantage was shown of the three–medication therapy including ACT, VCR and ADM comparing over the two–medication scheme ACT with VCR in stage II [12]. Adult patients can be treated according to pediatric protocols. The toxicity of such treatment is higher in adults than in children [13].

Radiotherapy

Nephroblastoma is a radiotherapy sensitive cancer. Currently, radiation therapy is usually part of treatment only for more advanced Wilms tumors (stages III, IV, and V) and for some earlier stages tumors with unfavorable histology. The recommended dose according to NWTS, SIOP, and PPGGL is 10, 15, and 20 Gy, respectively. The benefits from pre–operative radiotherapy in prevention of tumor rupture and in improving the stage distribution were confirmed in several SIOP trials such as: SIOP1, SIOP2 and SIOP5. Initially irradiation and later chemotherapy cause the shrinkage of the tumor [14]. Treatment regiments for Wilms’ tumor from NWTS and SIOP studies shows Table 4.

Bilateral Wilms’ tumor

There are distinguished synchronous and metachronous bilateral Wilms’ tumor. Synchronous WT occurred in about 6–7% of the tumors and metachronous WT in approximately 2% of all nephroblastoma [15, 16]. Treatment strategy rely on kidney – preserving resection (NSS, nephron sparing surgery) after preoperative chemotherapy which often results in significant reduction of tumor size. NWTS–2 and NWTS–3 trials showed no differences in survival between initial surgical resection and initial biopsy with preoperative chemotherapy [15]. The NWTS–5 trial recommend initial biopsy, chemotherapy and second – look surgery at week 5 [16]. The surgery is recommended within 12 weeks of diagnosis to limit the risk of chemoresistant clonal expansion.

Recurrent Wilms’ tumor

The prognosis and treatment for patients with recurrent Wilms tumor depends on their prior treat-

Table 3. Histological classification of Wilms’ tumor according to the risks groups – SIOP 2001 protocols

| Low risk tumor (LR) | Intermediate risk tumor (IR) | High risk tumor (HR) |
|---------------------|------------------------------|----------------------|
| mesoblastic nephroma| epithelial type               | blastema type        |
| completely necrotic nephroblastoma | stromal type       | diffuse anaplasia   |
| cystic partially differentiated nephroblastoma | regressive type     | clear cell sarcoma of kidney |
|                     | mixed type                  | rhabdoid tumor of kidney |

Table 4. Treatment regiments for Wilms’ tumor from NWTS and SIOP studies

| Stage | Chemotherapy | Radiotherapy |
|-------|--------------|--------------|
|       | Preoperative | Postoperative | Radiotherapy |
| NWTS–5 |             |              |              |
| I     | VA x 18 weeks | –            | VA x 4 weeks | VA x 4 weeks | No |
| II    | VA x 18 weeks | –            | VA x 4 weeks | VDA x 27 weeks | Node negative: negative: 15 Gy |
| III   | VDA x 24 weeks | 10.8 Gy      | VA x 4 weeks | VDA x 27 weeks | 15 Gy |
| IV    | VDA x 24 weeks | 12 Gy (lung if lung metastasis) 10.8 Gy (if local stage III) | CR after 9 weeks | VDA x 27 weeks | No CR after 9 weeks | None if lung lesions disappear by week 9 otherwise 12 Gy |
| SIOP –01 |          |              |              |
| I     | –            |            | VA x 4 weeks | VA x 4 weeks | – |
| II    | –            |            | VA x 4 weeks | VDA x 27 weeks | – |
| III   | –            |            | VA x 4 weeks | VDA x 27 weeks | – |
| IV    | –            |            | VDA x 6 weeks | –            | – |
ment, the histology (favorable or unfavorable), localization of recurrence. The outcome for recurrent disease is better if following features are present: favorable histology (low-risk tumors), initial stage of I or II, initial chemotherapy with vincristine and actinomycin D, recurrence at least 12 months after initial diagnosis and lack of previous radiotherapy [17, 18]. The use of etoposide, carboplatin and ifosfamide as single agents have shown anti–tumor activity in children with relapsed Wilms’ tumor [19]. The combination of either etoposide/carboplatin or ifosfamide/etoposide is also examined in many phase II clinical trials in children with recurrence of solid tumors. ICE (ifosfamide/carboplatin/etoposide) treatment was found to be associated with a response in over 80% of the patients, including those with CR (complete remission) in 27% and those with PR (partial remission) in 55% [20]. The response rate and most common toxic effects are shown in Table 5.

Toxicity

In three drug regiment with vincristine, dactinomycin and adriamycin the main acute toxicity was neuropathy due to vincristine. Grade 4 hematomal toxicity occurred in patients with higher stages. Hepatotoxicity was rare. Another side effect was mucositis [21]. During chemotherapy conducted according to ICE (ifosfamide/carboplatin/etoposide) scheme all patients had hematomal toxicity such as neutropenia and thrombocytopenia in stage IV toxicity. The described nonhematomal toxicity include septic shock, complications of the digestive tract, hepatotoxicity, proteinuria, hypophosphatemia, low concentration of potassium and chronic renal insufficiency [22]. The type, timing and dosage of chemotherapy have been major risk factors in the combined treatment. The clinically significant late side effects are predominantly cardiotoxicity, reproductive problems, renal dysfunction and the development of benign and malignant second tumours [23]. Clinical heart failure is the commonest presentation and can occur acutely or many years following treatment. Posttherapy left ventricular fractional shortening was reduced in 2.5% patients. An additive effect might occur with radiation involving the heart, as in patients requiring lung radiotherapy or left flank radiotherapy for upper pole WT. Regular monitoring with echocardiograms is recommended [24, 25].

Onads are particularly sensitive to radiation. In some cases fertility and a successful pregnancy outcome may be impaired, especially in girls who have abdominal radiation in which both ovaries or the uterus are within the field [26].

Development of renal disease can be observed in patients with progression of bilateral nephroblastoma or receiving irradiation in the opposite kidney in unilateral disease [27]. Chronic renal insufficiency has been reported in 19–73% of WT patients. The most important risk factors are: nephrectomy, abdominal radiotherapy and less compensatory renal hyperthropy [28]. The risk of nephrotoxicity may be reduced by avoiding nephrotoxic chemotherapy, optimizing radiation therapy and nephron–sparing surgery for bilateral disease [29]. The types of second cancers include bone and soft–tissue sarcomas, breast cancer, lymphoma, tumours of the digestive tract, melanoma and acute leukae- mias [30, 31]. In some studies secondary tumors increase with the increase in radiation dose and the use of doxorubicin intensified the effect of radiotherapy [32].

Prognosis

Wilms’ tumor metastasis occurs in children and adults in 10% and 29% of the cases, respectively [33, 34]. Nephroblastoma in adults is considered worse than

Table 5. The treatment of recurrent solid tumors in children – response and toxicity

| Chemo-therapy | Dose of medication | Treatment response | The most common toxic effect |
|---------------|--------------------|--------------------|-----------------------------|
| Etoposide     | 200 mg/m²/day for 5 days | CR in 7% PR in 35% | Neutropenia Thrombocytopenia |
| CarboPlatin  | 550 mg/m² every three weeks | CR in 26% PR in 26% | Neutropenia Thrombocytopenia |
| Ifosfamide    | 3 mg/m² for 2 days, every two weeks | CR in 28% PR in 24% | Leukopenia |
| Etoposide with carboplatin | 100 mg/m² for 5 days of etoposide and 160 mg/m² for 5 days of carboplatin with a 21–day interval between the two courses. | CR in 30% PR in 43% | Thrombocytopenia Anemia Neutropenia |
| Ifosfamide with etoposide | 2 g/m² of ifosfamide and 100 mg/m² of etoposide with 500 mg/m² of mesna every 3 hours x 3 intravenously for 3 days with a 21–day interval between the two courses | CR in 31% PR in 20% | Neutropenia Vomiting Thrombocytopenia |
| ICE (ifosfamide, carboplatin, and etoposide) | 1800 mg/m² for 5 days of ifosfamide; 400 mg/m² for 2 days of carboplatin; and 100 mg/m² for 5 days of etoposide | CR in 27% PR in 55% | Neutropenia Thrombocytopenia Non–hematological |
in children. Stages III and IV are present in 50% of adults and 30% of children. A higher advancement stage and common metastatic events are the reasons for worse treatment outcomes in adults compared to children. The prognosis depends on the primary advancement stage, the histopathology, time since the first remission, type of therapy, and the recurrence location. In patients with recurrence three–year survival is about 30%, especially in the presence of poor prognostic factors such as advanced stage greater than I, abdominal relapse at the site of previous radiotherapy, early recurrence (<12 months), and after previous three–medication therapy [8]. The use of etoposide, carboplatin and ifosfamide as single agents have shown anti–tumor activity in children with relapsed Wilms’ tumor [9]. The combination of either etoposide/carboplatin or ifosfamide/etoposide were also examined in many phase II clinical trials in children with disease recurrence. ICE treatment (ifosfamide/carboplatin/ etoposide) was found to be associated with a overall response in 80% of the patients, including those with CR in 27% and those with PR in 55% [10].

Biologic prognostic factors

Up till now some potential molecular factors have been identified. One is loss of heterozygosity (LOH) at chromosomes 1p and 16q. Children with LOH at 16q had greater risk of relapse and mortality than did children without these changes [35]. A similar results applied to LOH at chromosome 1p [36]. LOH for both chromosomes 1p and 16q was identified in approximately 5% of Wilms tumors. Other promising prognostic factors are; an increase in gene copy number or expression at chromosomes 1q [37], and telomerase expression level [38].

CONCLUSIONS

Wilms’ tumor in adulthood is extreme rare. The cure rates for adult Wilms’ tumor are improving. Therapy schemes based on pediatric protocols leads to similar results as observed in children. Treatment toxicity in adults is higher than in children. Up till now, several molecular prognostic factors have been identified. The novel treatment approaches development are necessary.

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