Juvenile cancer: improving care for adolescents and young adults within the frame of medical oncology

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Despite unique tumour epidemiology, a higher cancer incidence and modest survival improvement compared to paediatric patients, adolescents and young adults aged 15–30 have not been considered as a separate, ‘special’ group of patients in the frame of medical oncology. In an effort to emphasise this need, we review the particular characteristics of diagnosed tumours, aetiologic associations, nosologic classification, management, outcome and late toxic effects. Adolescents and young adults are in need of specialised care for intensive treatment of curable malignancies, skilled nursing care, interaction with peers, family and physicians as well as continuous psychosocial support. Enrolment in clinical research trials and close follow-up via the development of a cooperative infrastructure are imperative for the optimisation of management and avoidance of late effects. Similar to geriatric and paediatric oncology, we call for the intensification of treatment, support and research multidisciplinary efforts in order to better fulfil the pressing demands of this patient group.

Key words: adolescents, cancer, survival, treatment, young adults

Introduction

Over the last four decades, the need for intensive, specialised treatment, care and support along with the curability of childhood cancers have led to the establishment of paediatric oncology as a separate and well-recognised subspecialty in paediatrics. This has been the story of a spectacular success: cure rates have improved from <30% in the 1950s to ~75% in the 1990s [1], owing to financial investment, multidisciplinary research efforts and the development of a unique cooperative infrastructure throughout the world. Meanwhile, the next oldest age group of cancer patients have enjoyed much less of this focus for research and improved care than their younger counterparts. Despite the observation of both higher and accelerated cancer incidence in the 15–30 age group in comparison to children, along with only modest survival improvement [2], adolescents and young adults have not been considered any differently from their elderly counterparts. Although their most common malignancies are curable, 15–30-year-old cancer patients have seen the development of geriatric oncology taking place well before the need for recognising their unique characteristics, as well as optimising their care, has become evident. In this review, our aim is to emphasise the distinctive features of both tumours and patients in the 15–30 age group as well as the imperative need for specialised management, support and research by skilled personnel.

Epidemiology

Using a conventional age range of 15–19 for adolescents and 20–30 for young adults, there is ample evidence for an overall higher cancer incidence than that reported in paediatric populations. In fact, the incidence of cancer in young patients aged 15–30 years (220–250 cases per million youths) is higher and rising faster than in children. Data from the National Cancer Institute’s (NCI) Surveillance, Epidemiology and End Results (SEER) program and the UK cancer registry indicate that the incidence of cancer in adolescents 15–19 years old is 50% higher than in younger persons, for a rate of 203 new cases per million persons [3–5]. Overall, rates in both males and females were significantly higher at 20–24 years than at 15–19 years [6].

There is evidence of an increase in the cancer incidence rate in adolescents and young adults over the last quarter of the 20th century on both sides of the Atlantic. According to SEER and UK registry data, the overall rate of cancer incidence in adolescents has been rising at an average of 0.9% per year. Melanomas, non-Hodgkin lymphomas and testicular carcinoma have shown the greatest annual increases, over 5% for the former and 2% for the latter two [3, 5]. The most common tumours among 15–19-year-old adolescents are testicular cancer (15%) and Hodgkin’s disease (16%) by far, followed...
by central nervous system (CNS) tumours, non-Hodgkin’s lymphomas (NHL), thyroid cancer, malignant melanoma and acute lymphoblastic leukaemia (Figure 1). These tumours account for more than two-thirds of the total of malignant cases [5, 7]. Non-seminomatous germ cell tumours outnumber seminomas and most of the cases of Hodgkin’s disease are of the nodular sclerosis classical subtype [8]. Young patients with NHL usually have diffuse large B-cell or T-cell high-grade histology according to the World Health Organization (WHO) classification, in sharp contrast to the predominance of lymphoblastic and Burkitt types during early childhood [9]. This distribution is unique for this age group, as the paediatric embryonal tumours such as nephroblastoma, neuroblastoma, medulloblastoma, hepatoblastoma and retinoblastoma are seldom encountered. Rhabdomyosarcoma accounts for only a quarter of soft tissue sarcomas, the remainder including synovial sarcoma, liposarcoma and fibrous histiocytoma. Acute lymphoblastic leukaemia (ALL) (6%) is certainly less common than in children (30% of all cancers) [5]. Among patients in the 20–30 age group, the commonest tumours are reported to be Hodgkin’s disease, germ cell tumours, melanomas, CNS tumours, bone and soft tissue sarcomas [10, 11] (Figure 2). The first two tumours peak in incidence in this age group, whereas declining ALL is being surpassed by acute myelogenous leukaemia. Ovarian tumours are usually germ cell malignancies (60% of cases) in the 15–19-year-olds, whereas 20–24-year-old adult females have ovarian carcinomas in 70% of cases [12]. Characteristic in the 15–30 age group is the rare occurrence of common (for older ages) epithelial tumours of the aerodigestive and genitourinary tract. The relative frequency of epithelial tumours by age groups can be seen in Figure 3.

Although it is often stated that 15–19-year-old adolescents more commonly suffer from paediatric tumours, a closer look at epidemiological and incidence data do not support this observation. Hodgkin’s disease and germ cell tumours, the two commonest adolescent malignancies, occur at an incidence 3–6 times higher than the paediatric one. Epithelial carcinomas, thyroid cancer and melanomas are seldom, if ever, encountered in children, while ALL, NHL, CNS tumours, and osteosarcoma/Ewing family tumours are almost as common in adolescence as in childhood. Accordingly, neoplasias affecting teenagers should be better characterised as ‘adolescent-type’ tumours that also occur less often in childhood or tumours that are common in both age groups. Rhabdomyosarcoma and other embryonal sarcomas are a notable exception that represent true ‘paediatric-type’ tumours occurring in adolescence [13].

Ethnic/racial differences in incidence are apparent between Caucasian and black youths, with cancer appearing 50% more often in whites. The overall frequency of adolescent tumours seems to be equal between sexes in the USA [6] and higher for males in the UK [5]. Individual tumour types have unequal sex distributions with ALL, NHL, Ewing sarcomas, osteosarcomas and brain tumours more common in males. Thyroid
carcinomas, melanomas and Hodgkin’s disease are diagnosed more often in females.

Aetiology

The vast majority of cancers of adolescence and adulthood are sporadic events of unknown aetiology. Genetic conditions that are associated with an increased frequency of malignancies such as skin cancer, lymphoma, sarcoma, hepatic tumours and various carcinomas account for only a small proportion of malignant cases in these age groups. Indeed, neurofibromatosis, Li–Fraumeni syndrome, xeroderma pigmentosum, ataxiatelangiectasia, Fanconi pancytopenia, hereditary dysplastic nevus syndrome, Turner, Beckwith–Wiedemann, Gorlin, Bloom’s syndrome and multiple endocrine neoplasia (MEN) syndromes make up <10% of observed adolescent and young adult tumours [14, 15]. The rare breast/ovarian carcinomas encountered in young females aged <30 may be related to the presence of BRCA1/BRCA2 tumour suppressor gene mutations, similar to colorectal cancer cases associated with familial adenomatous polyposis or Lynch syndromes (hMLH1/hMSH2,6 mutations).

Similar to genetic factors, environmental factors have rarely been incriminated in the pathogenesis of malignancies of adolescents and young adults. Exceptions are clear-cell adenocarcinomas of the vagina–cervix in adolescent females, caused by prenatal exposure to diethylstilbestrol taken by their mothers, hepatic tumours caused by early hepatitis B/C infection, contraceptive use, aflatoxin exposure and human papilloma virus infection [16]. Early onset of active sexual life and multiple sexual partners lead to increased likelihood of chronic infection from high risk HPV strains and, consequently, increased risk for cervical cancer development at a young age. The latter could well turn out to be a preventable or treatable ‘juvenile’ tumour in view of hopes for complementarity of HPV DNA testing to cervical cytologic screening and effectiveness of HPV vaccination [17]. Adolescents and young adults exposed to radiation or chemoradiation during early childhood occasionally experience second tumours. In fact, a lot of ‘juvenile’ malignant cases that have been linked to aetiological factors are antineoplastic treatment-induced second tumours [18].

Most known carcinogens (tobacco, sunlight, diet and chemicals) cause DNA damage in somatic cells resulting in cancer after a delay of more than two decades. Accordingly, one would expect to observe a reduced incidence of such common carcinomas in the adolescent and young adult population, which is indeed the case. The emerging concept of accelerated carcinogenic effects of environmental toxins affecting developing tissues during childhood with resulting early epithelial tumours in young adulthood is not widely accepted yet. A notable exception is the frequent occurrence of melanoma in youths in sunny countries, a finding that lends weight to the possibility of carcinogenesis after a relatively short latent period secondary to intermittent, intense sun exposure during childhood and adolescence [19].

Nosologic classification

Classification of paediatric tumours is based on morphology (histology) [20], whereas adult tumours are classified according to organ site of the primary (International Classification of Disease, ICD-O) [21]. As stated before, the epidemiology of malignancy in adolescents and young adults represents a transitional phase between that of paediatric embryonal tumours and carcinomas/sarcomas of older adults. In order to develop services tailored to their needs, it is necessary to define patient characteristics through precise analyses of relevant population-based data. Since both paediatric and adult classification schemes seem to be inappropriate for the juvenile group, a diagnostic classification scheme is needed so as to meet epidemiological and service planning purposes, as well as be applied to international cancer registration procedures.

Some authors have advocated use of the paediatric classification system, at least for adolescents [22]. However, a number of major childhood cancer groups are irrelevant in adolescents and young adults. Moreover, cancers of older patients, especially carcinomas, do occur in adolescents and youngsters. Carcinomas, as specified in the childhood cancer classification, are inappropriately subdivided and do not describe adequately the pattern observed in young adults. On the other hand, the adult ICD classification cannot differentiate carcinomas, soft tissue sarcomas and germ cell tumours that arise in many different anatomical sites. On top of this, it is unable to define the important differences between morphological subtypes of carcinomas, CNS and bone tumours.

Consequently, neither histology nor organ of origin provides an accurate basis on which to classify the cancers of adolescents and young adults. Some authors have suggested changes of the paediatric classification, such as separate enumeration of colorectal, salivary, lung and breast carcinomas from the ‘other carcinoma’ group [22]. In an effort to produce a separate ‘juvenile’ nosologic system, Birch et al. [5] created a morphology-based classification scheme with revised tumour

<table>
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<tr>
<th>Tumour group</th>
<th>Definition</th>
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<tr>
<td>Group 1</td>
<td>Leukaemias</td>
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<td>Group 2</td>
<td>Lymphomas</td>
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<td>Group 3</td>
<td>CNS tumours</td>
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<td>Group 4</td>
<td>Bone tumours</td>
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<td>Group 5</td>
<td>Soft tissue sarcomas</td>
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<td>Group 6</td>
<td>Germ cell tumours</td>
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<td>Group 7</td>
<td>Melanoma and skin carcinoma</td>
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<td>Group 8</td>
<td>Carcinomas (except of skin)</td>
</tr>
<tr>
<td>Group 9</td>
<td>Miscellaneous specified neoplasms (including embryonal paediatric tumours)</td>
</tr>
<tr>
<td>Group 10</td>
<td>Unspecified malignant neoplasms</td>
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Data from Birch et al. [5].

CNS, central nervous system.
groups specifically for the 15–24 age group malignant epidemiology (Table 1). Hopefully this scheme, which also facilitates comparisons of cancer data in children with those in adolescents, will become a standard format for data presentation. It may also facilitate international comparisons and encourage research in juvenile oncology.

Special management issues

The majority of juvenile tumours are potentially curable. Five-year survival rates for ALL, Hodgkin’s disease, NHL, sarcomas, germ cell and CNS tumours range from 45% to 90% and usually correspond to a cure [23, 24] (Table 2). Accordingly, treatment given to a youngster with malignancy often aims for long-term disease control and survival. This is an aim paediatric oncologists are familiar with, but ‘adult’ medical oncologists are not as only too often palliation of symptoms and modest prolongation of survival are the only realistic targets they can hope for. On the other hand, paediatric oncologists may not have the experience to deal with the different types of lymphomas, soft tissue sarcomas, melanomas, germ cell tumours and carcinomas that affect adolescents and young adults more often than children.

The curative purpose of treatment in the juvenile patient group necessitates that it is given in a ‘state of the art’ fashion, as intensive as it needs to be so as to maximise the chances for cure. Management often consists of combined-modality treatment, incorporating multi-agent chemotherapy, high-dose radiotherapy and aggressive surgery. Chemotherapy is often dose-intense, dose-dense or high-dose with autologous marrow/stem cell rescue. Differences in outcome as large as cure dose-intense, dose-dense or high-dose with autologous marrow/stem cell rescue. Differences in outcome as large as cure

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<tr>
<td>ALL</td>
<td>35%</td>
<td>51%</td>
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<tr>
<td>AML</td>
<td>22%</td>
<td>42%</td>
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<tr>
<td>Hodgkin’s disease</td>
<td>88%</td>
<td>90%</td>
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<td>NHL</td>
<td>56%</td>
<td>69%</td>
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<tr>
<td>Astrocytoma</td>
<td>62%</td>
<td>75%</td>
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<tr>
<td>Medulloblastoma</td>
<td>63%</td>
<td>75%</td>
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<tr>
<td>Osteosarcoma</td>
<td>49%</td>
<td>59%</td>
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<tr>
<td>Ewing sarcoma</td>
<td>36%</td>
<td>56%</td>
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<tr>
<td>Soft tissue sarcoma</td>
<td>70%</td>
<td>63%</td>
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<tr>
<td>Rhabdomyosarcoma</td>
<td>40%</td>
<td>45%</td>
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<tr>
<td>Germ cell tumours</td>
<td>79%</td>
<td>90%</td>
</tr>
<tr>
<td>Thyroid carcinoma</td>
<td>99%</td>
<td>99%</td>
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<tr>
<td>Melanoma</td>
<td>84%</td>
<td>92%</td>
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<tr>
<td>Total</td>
<td>69%</td>
<td>77%</td>
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ALL, acute lymphocytic leukaemia; AML, acute myelocytic leukaemia; NHL, non-Hodgkin’s lymphoma.

It is a common experience of young patients and attending personnel that adolescents and young adults do not ‘fit’ in either paediatric wards or medical wards with elderly patients. Dedicated units for patients of the 15–30 age group within the context of medical oncology offer an ideal environment for treatment, interaction with physician, family and peers, skilled nursing care, individualised psychosocial support and coordinated clinical research [29]. Educational activities could also be set up, though quite difficult to organise. Nowadays, most countries recommend referral of adolescents with cancer to specialised centres (model of centralised care) for more effective multidisciplinary treatment along the example of paediatric oncology [30]. Such units should be developed as a component of a broader structure of care that includes recognition of local providers, resources available in the community and awareness of factors that can ensure access to care for these patients. Some degree of decentralisation may be more realistic for models of care suitable for sparsely populated or remote areas. Adolescent oncology units already function successfully in the UK, a country that spearheaded the way for their development [31].

Psychologic and social support is another pivotal issue for 15–30-year-old patients. These youths face several challenges, such as independence, education, sexual maturation, employment, marriage, reproduction, parenting on top of a struggle for reconciliation with cancer diagnosis, for cure and survival [32]. Isolation from peers and society, carrying the ‘stigma’ of cancer is often devastating [33]. The adverse effects of therapy such as mutilating surgery, alopecia, acne, weight gain, stunted growth, disruption of normal sexual function may be overwhelming to individuals at a time in their life when they would normally try to define and accept their self-image. Time away from school is hard to accept [34, 35]. At the same time, the youth going through these hardships has to rely on the family’s help at a period when his/her important target would be financial and emotional independence from it. Skilled, individualised psychosocial support of these young patients is imperative for unhindered management and smooth re-incorporation into active socioeconomic life [36]. Evidence suggests that it should be long-term, continuing for years after successful treatment [37]. It is also pivotal for the patient with relapsed disease or the terminally ill youngster. Support should also encompass the family, friends, educational
and work environment. Psychiatrists, psychologists, social workers and medical personnel should be actively involved with it, working as a team.

Long-term follow-up of youngsters treated for malignant diseases, their education and adherence to health promotional measures are crucial for both timely diagnosis of relapse as well as prevention of related or unrelated comorbidity. Transition of care and identification of the long-term follow-up provider are critical issues: lifelong follow-up in the treating specialised unit, the use of joint clinics staffed by juvenile and medical oncologists, implementation of a ‘transition programme’ to transfer the patient’s care to either the medical oncologist or the general practitioner have been provided in various parts of the world. At present no gold standard can be identified, as each model has to take into consideration the resources available, the quality, interest and geographical location of juvenile, adult oncologic and primary health services [38].

Outcome and clinical research

The overall 5-year survival for adolescents with cancer has been reported to be 77%, with similar outcome data existing for the young adult age group (19–24 year-olds) [6]. The survival gains that have been accomplished for most of the adolescent tumour types during the 1980s and 1990s are encouraging (Table 2). The worse outcomes are seen in AML, ALL and sarcomas (bone and soft-tissue), with data on carcinomas being scant. Each of the above tumours is associated with a lower mean 5-year survival rate than that in younger patients [2]. Cancer mortality burden in adolescents is mainly due to leukaemias, followed by lymphomas, CNS tumours, sarcomas and, to a lesser extent, germ cell tumours [39]. This parameter is a function of both survival and incidence rates. Most embryonal paediatric tumours (neuroblastoma, nephroblastoma, rhabdomyosarcoma) have a worsening outcome with increasing age, a fact that probably reflects differing tumour biology and host factors [40]. Children with CNS tumours fare better than adolescents, with the exception of those of very young age (<4 years). At this age, inability to administer full doses of radiation therapy because of vulnerability of the developing brain adversely affects outcome [41]. In contrast, the prognosis of children and adolescents with osteosarcoma seems to improve with age. Adolescents and adults with germ cell tumours seem to fare as well as children, with young adult patients tolerating bleomycin better than older adults who may experience pulmonary toxicity. Of note, mediastinal teratomas may behave in a more benign manner in children and young teenagers but are more aggressive and often fatal in older patients [42].

Adolescents and young adults with Hodgkin’s disease have a more favourable prognosis than older adults (>45 years) but an inferior prognosis when compared to children, who fare better because of higher frequency of lymphocyte predominant histology, stage I disease, lower frequency of B symptoms and more common use of combined modality therapy [43]. Children with lymphoblastic lymphoma and Burkitt’s lymphoma have a superior outcome when compared to the juvenile patient group, probably due to more intensive therapy [44]. Acute lymphoblastic leukaemia exhibits an age-related prognosis with children faring better than adolescents, who, in turn, fare better than adults. This differential prognosis is mainly due to differing biology of the malignancy with adverse karyotypic and molecular markers appearing more often in adolescents and adults, although the higher dose-intensity of paediatric chemotherapy protocols may also contribute to the favourable prognosis of children, as shown by recent clinical trials [45, 46].

There is a lack of epidemiological data on particular characteristics of the natural history and survival of very young patients with epithelial carcinomas. Young women aged <25 are diagnosed with localised ovarian carcinoma in 60% of cases, the reason for early diagnosis being heightened screening of the ovary among girls with delayed menarche or amenorrhea. Little is known of the biology and prognosis of very young women with breast carcinoma. An inferior prognosis has been suggested for women aged <35 when compared to older women, due to a combination of factors such as increased frequency of hormone-receptor negative disease, aggressive malignant phenotype and germine mutations (BRCA1, BRCA2) [47]. Anecdotal evidence indicates aggressive clinical course of lung and gastrointestinal carcinomas in young patients. Generally young patients with ‘adult’ common carcinomas are more likely to carry acquired or germine genetic lesions responsible for early carcinogenesis, a genetic bias that may confer inferior survival.

Although the improvements in outcome of youngsters with cancer seem to be satisfactory at first glance, they are inferior to what has been achieved in paediatric oncologic patients. The relative improvement in 5-year survival rates from 1974 to 1995 has been in excess of 30% for children and only 19% for adolescents aged 15–19. In the recently published EURO-CARE-3 study, the annual improvement rate of survival in patients aged 15–24 was shown to be inferior to those observed both in children and in adults over the age of 40. In fact, young patient mortality rates were increasing for some tumour types (soft tissue sarcomas) in the last quarter of the 20th century. In 1974, 5-year adolescent survival rates were superior when compared to paediatric patients (64% versus 55%) [3]. In sharp contrast, respective figures for the year 2000 are 80% for adolescents versus 85% for children, a reversal in the survival order from a 10% advantage to a 5% deficit.

The spectacular success of paediatric oncology is mainly due to timely diagnosis, optimisation of multi-agent chemotherapy regimens, combined-modality treatment and supportive care. Most of these achievements have been the result of painstaking, large-scale enrolment and treatment of children with cancer in cooperative multicentric clinical trials. This was made possible through the creation of an extensive socioeconomic infrastructure via cooperative groups in the USA and Europe. More than 95% of children with cancer in the USA
are treated at institutions that are participating in clinical trials sponsored by the National Cancer Institute (NCI). In contrast, only 10% of adolescents and 2% of young adults are entered on to clinical trials of the paediatric or adult cooperative groups (Figure 4). The ‘adolescent and young adult gap’ spares no geographic or ethnic regions and probably has many causes [2, 48]. A perception of poor adolescent compliance to complex protocols, avoidance of adding the burden of a trial to a teenager struggling to cope with cancer, false belief that a trial is not needed because of excellent prognosis, lack of information about trial participation possibilities, lack of access to trials and exclusion criteria commonly seen in paediatric or adult trials are some of these causes [49]. Accrual is the lifeblood of clinical trials and lack of it will hinder the development of more effective or less toxic treatment strategies. The more common occurrence of carcinomas, tumours refractory to treatment once spread beyond the primary site, and more aggressive course of a number of malignancies with increasing age (ALL, embryonal tumours, CNS tumours, sarcomas) may be secondary factors contributing to the widening gap in outcome improvement rates.

Late toxicity

Late toxic effects of aggressive treatment are dreaded and have been well described in the medical literature. Surgery, chemotherapy and radiotherapy have all been incriminated for severe late effects, the frequency of which increases exponentially upon combination of the above [50, 51]. They are probably not as important anywhere as in the 15–30 age group. The threat for toxic effects is often combined with risk-taking behaviour and unhealthy lifestyle of cancer survivors, which augments normal tissue injury [52]. Aggressive surgery is frequently a cause of late effects interfering with a patient’s quality of life. Retroperitoneal lymphadenectomy causes ejaculatory dysfunction in up to 20–30% of youths with metastatic germ cell tumours, despite the improvement seen with application of nerve-sparing techniques [53]. Mutilating surgery in the limbs, head and neck or torso causes disfigurement with resultant functional disabilities. Remarkable progress has been made in the field with the advent of limb-sparing surgery and adjustable endoprostheses.

Loss of fertility is frequently a dreaded consequence of chemotherapy in adolescents and young adults. The frequency, degree and duration of infertility depend on the dose and type of drugs administered, the patient’s age and the existing malignancy. Infertility rates range from 20% to 90% for men and 15% to 75% for females [54, 55]. Testicular, ovarian and hypothalamic–pituitary irradiation are likely to cause permanent sterility, whereas Leydig cell function is more variably affected [56]. Sperm banking, female germ cell banking and hormone replacement are therapy issues as important as those of antineoplastic treatment of the patient’s quality of life [57]. Despite lack of evidence supporting latent germ cell damage [58], fears for teratogenesis or cancer in offspring of long-term survivors of adolescent cancer are common and parents have to be reassured.

Doxorubicin-induced cardiac injury is more common with cumulative administered doses in excess of 550 mg/m² or combination of anthracycline-containing chemotherapy with mediastinal radiotherapy, in which case an increased risk of 4–10-fold has been described in the literature [59]. Radiation therapy and chemotherapeutic agents such as bleomycin, high-dose cyclophosphamide, nitrosoureas, busulphan may damage the lungs, causing pulmonary fibrosis [60, 61]. CNS radiation causes cerebral atrophy, demyelination, leukoencephalopathy and neurocognitive defects with a frequency of 30–70% depending on young age, dose and combination with neurotoxic agents (ifosfamide, methotrexate, cytarabine) [62, 63]. Less common toxic effects include peripheral neuropathy, otoxicity, Raynaud’s phenomenon, bladder constricton, intestinal fibrosis/obstruction, hepatotoxicity, xerostomia/dental problems, post-radiotherapy neuropathic pain, femoral head necrosis.

Undoubtedly the most dreaded complication of antineoplastic treatment is the occurrence of a second tumour. The occurrence of a second tumour in a patient cured from his primary malignancy may be due to several factors. A chance occurrence, host susceptibility factors, genomic lesions and common environmental influences may all contribute. Still, there is irrefutable evidence from case–control and prospective studies that antineoplastic treatment increases the relative risk of secondary malignancies. Adult survivors of Hodgkin’s disease treated with MOPP-like regimens with or without radiotherapy have a relative risk of 16–66 for leukaemia, 3–35 for non-Hodgkin’s lymphoma and 3–13 for solid tumours [64, 65]. An overall cumulative risk of 20% for any second cancer has been reported at 25 years post-treatment. Causal links have been established for radiation therapy with...
sarcomas, thyroid cancer, breast cancer, lung cancer and leukemias. Cytotoxic agents commonly incriminated for carcinogenesis are alkylators and nitrosoureas [18, 50]. Cumulative drug dose, treatment duration, radiotherapy dose and field volume as well as combined/sequential chemoradiotherapy are determinants of the risk. Minimising late toxicity and carcinogenicity while maximising efficacy is one of the toughest challenges that juvenile oncology has to meet so as to offer teenagers and youths a safe prospect for cure.

Conclusions

In conclusion, a renewed focus on clinical research, coordinated support, safe and effective management of youngsters with juvenile cancer is called on by many aspects of adolescent and young adulthood malignancies and its patients. Even if medical aspects of individual diseases do not differ across age groups, distinct tumour biology and epidemiology, need for skilled intensive treatment, lack of clinical research, lack of survival improvement comparable to paediatric patients, requirement for intensive psychosocial-supportive care and occurrence of late toxic effects are there. They are more than good enough reasons for the oncologic community to take up the call and lead the way for a multidisciplinary effort on optimising care and outcome for young patients full of hope and expectations.

References


