A Review on- Childhood Tumor

Rubal Narang[1*], Shubhangi mali[2*] Gaurav Jadhav[3*]

[1]Student at pratibhatai pawar college of pharmacy, Wadala Mahadeo shrirampur.
[2]Assistant professor at pratibhatai pawar college of pharmacy, Wadala Mahadeo shrirampur.
[3]Student at pratibhatai pawar college of pharmacy, Wadala Mahadeo shrirampur.

***Abstract*: This article reviews the current thoughts on Etiology, diagnosis and management recommendations for children with different types of tumors, The most common types of cancer diagnosed in children ages 0 to 14 years are leukemias, brain and other lymphomas and we discuss briefly in this article cancer such as: Wilms tumor, brain tumor, Myofibroblastic Tumor and Salivary gland tumor and Cardiac tumor.**

Malignant brain tumors are the leading cause of cancer death among children and the second most common type of paediatric cancer. Despite several decades of epidemiologic investigation, the Etiology of childhood brain tumors (CBT) is still largely unknown, we believe that advances in genetic and molecular biologic technology, including improved histologic subtyping of tumors, will be of huge importance in the future of epidemiologic research and will lead to a more comprehensive understanding of CBT Etiology

We describe our clinical experience of eight cases of secondary cardiac tumor. The pathology of the tumors was lymphoma (three), Wilms' tumor (two), malignant teratoma (one), neuroblastoma (one), and pleuropulmonary blastoma (one). Metastatic sites were the right atrium in Wilms' tumor and neuroblastoma, the left atrium in pleuropulmonary blastoma and malignant teratoma, and multiple sites in lymphoma, purpose of Wilms' tumor is the most common renal tumor in children. Outcomes have improved dramatically over the past few decades, but important treatment questions remain. These include the role of molecular biologic markers in stratifying patients for therapy or targeting tumors for treatment. We present a summary of these advances and outline the current treatment of Wilm's tumor

***Keywords*: cancer, Pediatric brain tumors, salivary gland tumor, IMT tumor, Wilms tumor**

INTRODUCTION:

- An abnormal growth of cells which tends proliferate in an uncontrol way and, in somecase, to metastasize.
- Cancer is not one disease; it is group of additional 100 different and distinctive diseases. The type of cancer that occur in children and adolescents are different from those that occur in adults [6]
- 'Childhood' denotes to those children aged 0 to 14, inclusively. Childhood cancers are usually very different to those seen in adult. In the childhood cancer have amutual disease process-cells grow out of controlled, develop abnormal sizes and shapes, ignore their typical boundaries inside the body, destroy their neighbour cell, and ultimately can spread to either of organ and tissue [7]
- Some of these alteration in the histological typing of tumors concerned entities predominantly or exclusively occurring within the paediatric age group, in particular the embryonal tumour [5]
- Here are several epidemiologic studies T on neoplastic disease in the adult African,8J6.20.'34 but there are very few like studies on African children.6#21 Although infectious and inflammatory disease still account for a more proportion of death and illness in the African child, there is no doubt that neoplasms do also account for a significant percentage. Environmental agent which are oncogenic in Africa are largely anonymous; even the possible role of ionizing radiation in inducing cancerla [3]
- The two most common chief tumor in childhood, medulloblastoma and neuroblastoma, arise in the nervous system. Mechanisms driving the formation of these tumors have become progressively clear and point to the trouble of normal development as an important factor [2]
- Approximately 2200 individuals under the age of 20 are diagnosed with a brain tumor apiece year (American Cancer Society (ACS), 2003). Malignant brain tumors are the leading cause of cancer death among children and the second greatest common type of paediatric cancer after leukemias (Gurney et al., 1999; National Cancer Institute (NCI), 1991). Astrocytomas version for 52% of childhood brain tumor (CBT), primitive neuroectodermal tumor.
- (PNET) or medulloblastoma/embryonal tumors account for 21%, ependymomas for 9%, and other glioma 15% (Gurney et al., 1999; National Cancer Institute (NCI), 1991). The etiology of the majority of podiatric brain tumors continues to be largely unknown despite decades of epidemiologic study.[4]
- In order to measure the impact of the new WHO classification of nervous system tumor on the epidemiology and histological typing of podiatric brain tumor, we re-examined all primary CNS childhood tumors occurring in patient in their first 17 years of life that were seen in our neuropathology department over a period of 17 years, and compared our information with those of other international cancer registers and hospital series [5]
- A fundamental step in caring for these children is to estimate the current burden of childhood cancer in India and to understand how the incidence and outcome of the disease varies athwart the country. In this situation, this study aims to describe the epidemiology of childhood tumor in India. This will serve as a reference source for clinician, epidemiologist researchers, and health administrators. It should also be the stimulus for further research on the etiology of childhood cancer.[8]

- Childhood cancer, once regarded as an acute fatal illness, has become a chronic life-threatening disease (Koocher & Sallan, 1978). The psychosocial features evolving from the improving medical outlook of juvenile cancer are reflected in the literature. The early literature, written at a time when the child with cancer closely always died, dealt almost wholly with the problem of the seriously ill [9]

➢ **Tumors**: **Data and materials**

Figure no. 1:General picture of child

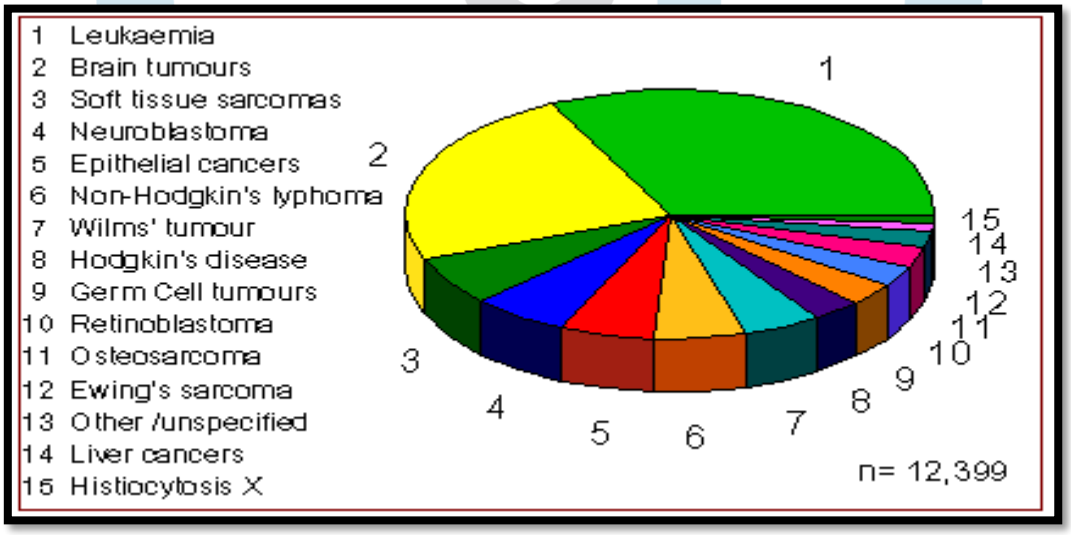

Figure no. 2: The chart above shows the proportions of different types of childhood cancers - based on UK data from The National Registry of Childhood Tumours [15]

- **Different types of cancers in childhood**

- **Leukemia**: is the maximum in common type of childhood cancer, representing about one third of all cancers in under 15-year-olds. Leukemia is a condition where besides may underdeveloped white blood cells are found in the blood and bone marrow. Four fifths of childhood leukemia's are severe lymphatic leukemias (ALL), other types comprise acute myeloid leukemia (AML) and chronic myeloid leukemia (CML).

- **Brain tumors** are the most common solid tumors in childhood, and make up around a fifth of all children's cancers. There are many diverse types of brain tumors; medulloblastoma, astrocytoma and brainstem glioma are the utmost common.
- **Neuroblastoma** (sympathetic nervous system),
- **retinoblastoma** (eye),
- **Wilms' tumor** (kidneys), and
- **hepatoblastoma** (liver) are most usually found in infants or young children. Other malignancies found in children and early adults include Lymphomas (Hodgkin's and Non-Hodgkin's Lymphoma), soft tissue sarcomas (including

rhabdomyosarcoma), bone cancer (osteosarcoma and Ewing's sarcoma), plus a number of other fewer common childhood cancers. Histiocytosis is infrequent; it is not thought to be a exact cancer, but in many respects acts like one.

The origin of most cancers remains unknown. A minority of cancers are known to be inborn (inherited). For example, roughly retinoblastomas, and Wilms' tumors are thought to be genetic. In infrequent cases the family may have a history of cancers (Li-Fraumeni Syndrome) [14].

However, most childhood cancers have no obvious genetic cause, Children with cancer are generally treated by experts.

Medical experts who have expertise in diagnosing and treating children with cancer include pediatric oncologists, pathologists, hematologists, radiotherapists, surgeons, radiographers, and others; all of whom work carefully together, often in dedicated children's cancer centers. National and International children's cancer organizations have changed in order to provide the best treatments, and are constantly engaged in research to additional understand and develop better dealings for childhood cancer. [16,17]

1. WILMS TUMOR:

Wilms' tumor (WT) or nephroblastoma is the utmost common primary malignant renal tumor in children. More than 90% of children are now probable to have an excellent outcome with current treatment modalities, Wilms' tumor is the most common renal tumor in children. Outcomes have improved intensely over the past few decades [10]

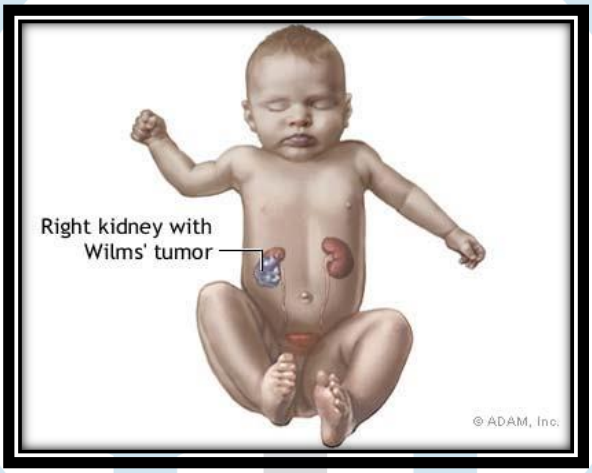

Fig1.1 Wilms tumor in child [21]

- **Epidemiology:**

In children under 15 years of age, the annual frequency rate of WT is about 7e10 cases per million, and this accounts for 6-7% of all infant cancers, more than 80% of cases are diagnosed earlier 5 years of age, with a median age of 3.5 years [10]

- **Etiology:**

There are a number of recognized syndromes associated with an increased predisposition near developing WT, Aniridia is found in 1.1% of WT patients and is attributed to a PAX6 gene deviation. This is located next to the WT1 gene on chromosome 11p13, which contains genes accountable for development of the kidney, genitourinary tract and eyes

WT1 encrypts transcription factors involved with gene regulation during renal and gonadal development Recently, a previously uncharacterized tumor suppressor gene then known as the 'Wilms tumor gene on the X chromosome', or WTX, was found to be deactivated in up to one third of WT cases [10]

- **Pathology:**

Histology is the most vital prognostic indicator for WT. The majority of WT patients have tumor with FH. Several tumor forms are associated with an increased risk of tumor reappearance, or resistance to standard WT chemotherapy**,** two other renal tumors, rhabdoid tumor of the kidney and clear cell sarcoma of the kidney, were once considered to be WT. They were acknowledged to have a higher risk of recurrence and therefore receive different therapy.

Classic WT includes three histological cell forms in varying proportions: (1) blastemal; (2) epithelial; and (3) stromal components mostly Stage I if diagnosed early

Nephrogenic rests (NRs) are precursor lesions that are found in 25e40% of kidneys with WT, Most NRs do not grow into malignancy. Rather, they can undergo maturation, sclerosis, or disappear completely

If NRs are present on one kidney, they are also probable to be present on the contralateral kidney, and frequent investigation of the contralateral kidney is recommended after surgery to monitor for metachronous tumors [10]

Table 1: Stages of Wilms tumor

Stage I	**Tumor kept to kidney and completely resected; no capsular breach, tumor spillage or renal sinus extension**
Stage II	Extracapsular penetration (including iatrogenic via biopsy prior to resection) or renal sinus postponement with vascular involvement; complete resection with negative margins and no lymph node involvement
Stage III	Non-hematogenous spread elsewhere the kidney (abdominal lymph nodes, transacted renal vein, IVC tumor thrombus); macroscopic/microscopic residual tumor after resection; peritoneal spillage during resection
Stage IV	Hematogenous metastases (lung, liver, bone, brain) or extra-abdominal lymph node spread
Stage V	Bilateral renal involvement at diagnosis

- **Treatment**

Surgery maintains an significant role in treatment, although the improved prognosis for this malignancy during the 20th century is attributed primarily to developments in chemotherapy. Overall survival rates reach 90% with current treatment regimens. **[10]**
Chemotherapy: The clinical development of children with WT was altered in the 1960s with the application of the chemotherapeutic agents dactinomycin (AMD) and vincristine (VCR). Survival was dramatically improved uniform for patients with high-stage tumors compared to prior treatment with surgery and radiation therapy alone.
Stratification of treatment is primarily based on stage and tumor histology, although the biologic features of the tumor are also a consideration [10]

2. SALIVARY GLAND TUMOR:

The salivary glands contain of the parotid gland, the submandibular gland, the sublingual gland and frequent other small glands throughout the oral cavity [19]
There is an increased incidence of another malignant neoplasms in survivors of childhood cancers. The most common second malignancies are acute leukaemia, bone and soft tissue tumors, and carcinoma of the skin, breast, and thyroid. Although, ionizing radiation has been demonstrated to rise the risk of developing a salivary gland neoplasm, there are few reports of salivary gland neoplasms occurring in patients treated for cancer in childhood [11]

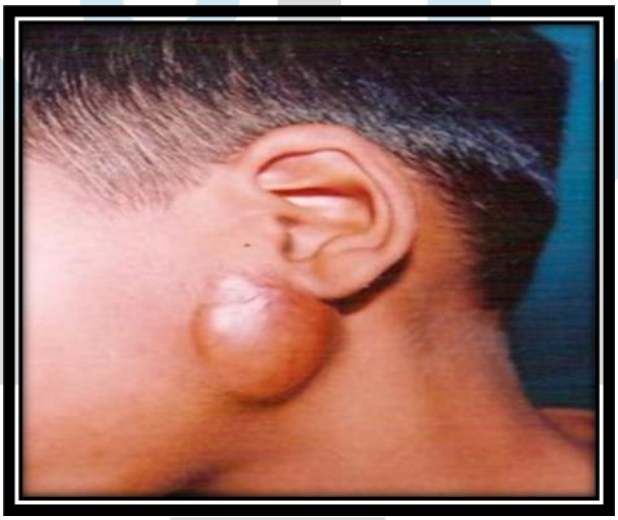

Fig 2.1: tumor on salivari gland in child[20]

Children who survive childhood malignancies are known to have an increased risk of second neoplasms. Genetic factors, chemotherapy, and ionizing radiation have been concerned in the development of these tumors. The risk of a second malignancy in a patient treated for childhood tumor reaches approximately 12% at 20 years, with leukaemia's, bone and soft tissue tumors, besides carcinomas of the breast, skin, and thyroid gland being the most common another malignant neoplasm.[11]

- **SIGNS AND SYMPTOMS**

Duration of symptom ranged from 2 months to 8 years, with an average of 1.6 years. The main presenting manifestation in all cases was the appearance of a mass. Thirty-three tumors presented in the parotid and 5 in the submaxillary gland. The mass was generally firm to hard, mobile, and non-tender. The size varied after 2 to 10 cm, with a mean size of 3.2 cm (Table 3). Three had multinodular tumor, while 2 had intra-oral presentation of their parotid tumor [18]

- **Treatment**: One permanent nerve palsy followed x-ray therapy for mucoepidermoid carcinoma of parotid gland.[18], The best treatment for podiatric salivary gland tumors was surgical excision. Since the anatomic variance of the facial nerve in children,

caution is essential during surgery. Facial nerve monitoring during parotid tumor surgery is a standard requirement now, but in the current study all operation were performed by experienced surgeons deprived of facial nerve monitoring.[19]

3. INFLAMMATORY MIOFIBROSTIC TUMOR: [IMT]

Inflammatory myofibroblast tumor was first described in the lungs in 1937, and since then has been reported at various sites such as mesentery (one of the most common extrapulmonary sites) and cardioesophageal region. It mainly is a tumor of young adult and children, and the lung is the maximum frequent site of location

Histopathological, IMT is a benign solid tumor composed mainly of spindle-shaped cells and has a chronic inflammatory component consisting of plasma cells, lymphocytes, and occasional histiocytes. The variation of terms used to describe this entity includes inflammatory pseudotumor, pseudo sarcomatous myofibroblastic proliferation, plasma cell granuloma, xanthomatous pseudotumor, inflammatory myofibrohistiocytic proliferation, inflammatory fibrosarcoma, and, recently, IMT.1-11 In fact, this is a factual tumor, and all these nomenclatures define the same histopathologic entity ,The radiologic appearance of IMT located in the thorax cannot be differentiated from other causes of coin lesions such as congenital malformations or malignancy. Chest roentgenogram and CT generally are reliable in accurately defining the lesion, but usually offer diminutive differential diagnostic support. However, a coin lesion in a child carries a high possibility of being a benign lesion, most probably IMT [12]

Table 3.1 Clinical, Radiologic, and Surgical Features of Children with IMT [12]

Case No	Age, sex	Presenting Symptoms	Radiologic Findings	Location	Treatment
1	8 yr, F	Dysphagia, weight loss	UGI series: distal oesophageal stenosis	Cardioesophageal junction	Proximal esophagogastrostomy plus esophagogastric anastomosis
2	10 yr, M	Minor respiratory symptoms	Plain chest x-ray and chest CT: Posterior mediastinal mass	, Right lower lung lobe	Total excision
3	6 yr, M	Abdominal pain, weight loss, fever, malaise	USG, CT: Heterogeneous mass in the left hepatic lobe, calcification	Left hepatic lobe	Total excision (Lateral segmentectomy)
4	9 yr, M	Abdominal pain, vomiting, weight loss, fever	USG, CT and upper GI series: Multilobulated soft tissue mass with calcification, in the left upper quadrant, causing intestinal obstruction	Proximal small bowel mesentery	Total excision with adjacent jejunal segment
5	12 yr, M	Abdominal pain, growth retardation	USG, CT: Hepatosplenomegaly, bilobulated and densely calcified intrapelvic mass	Distal small bowel mesentery	Total excision
6	11 yr, M	Weight loss, loss of appetite	USG, CT: Solid, well demarcated mass in the pelvis	Antimesenteric border of the descending colon	Total excision
7	8 yr, F	Abdominal distension, hematemesis	USG, CT: Giant infiltrating mass between the stomach and spleen with amorphous calcification, another mass in the porta hepatis, dilatation of intrahepatic bile ducts	Between the stomach and spleen, and porta hepatis	Total excision 1 splenectomy 1 partial gastrectomy 1 partial excision of the portal mass and percutaneous biliary drainage

4. BRAIN TUMOR:

Brain tumors are classified under several different schemes according to cell geomorphology and the degree of malignant behaviour. The brain is composed of two main types of cells, neurons and glia, which both arise in early development from the primitive neuroectoderm

Glial cells can be subdivided into four major types: astrocytes, oligodendrocytes, ependymal cells, and microglia (Mischel and Vinters, 2001). The popular of primary brain tumors arises from glial cells and are broadly categorized as gliomas, but are usually broken down into more specific subtypes, such as astrocytoma, oligodendroglioma, and ependymoma (Preston-Martin et al., in press).

Fig 4.1: brain tumor in child [23]

- **General developmental and its consideration of brain tumor:**

Principles Initial stages of development, such as gestation and early childhood, are marked by particular susceptibility to toxic insults. These stages are characterized by extensive amounts of intricately coordinated cell growth and differentiation.

The ultimate health effect of slightly kind of prenatal toxic insult varies substantially with the specific timing of fatal development occurring in utero. This is true for agents producing fatal and teratogenic endpoints as well as neoplastic transformation, and is true for all organs including the central nervous system (CNS)

As the scientific community studies more about the specific processes and detail of neurologic cellular development, it may become possible to approximate the timing of the specific toxic insult that leads to the development of histologically distinct brain tumor. Incidence rates with clear age-related histologic patter suggest that different agents may be causing different type of cancers. Theoretically, if specific cells are most vulnerable at certain time during development and those cell are also differentially susceptible to various toxin, we may someday be able to know which compound is most likely responsible for a given tumor.

1. **Environmental exposures**: The following section will summarize the epidemiologic finding to date regarding the many environmental exposure that have been hypothesized to contribute to the incidence of CBT
2. **Ionizing radiation**: Although many environmental exposure are hypothesized to contribute to the development of brain tumor, only ionizing radiation has a proven etiologic role
3. In children, radiation for early childhood cancer has been associated with the later development of CNS tumor (Meadows et al., 1985; Neglia et al., 1991).

Exposure to radiation during gestation has been related to increased incidence of CBT since 1958. Several more recent studies have also found elevated risks of brain and CNS tumor in children whose mothers underwent diagnostic prenatal radiation [4]

4. **N-nitroso compounds:** Another environmental exposure that has received much attention as a potential risk factor for CBT is N-nitroso compound (NOC). NOC have been found to be carcinogenic in 40 animal types, and in the form of N-nitrosourea induce brain tumor when contact occurs trans placentally (Bunin, 2000; Lijinsky, 1992). [4]

Dietary NOC, primarily nitrosamine, are found in foods that contain nitrite or have been exposed to nitrogen oxide, such as nitrite-cured and smoked meat and fish, cheese, and beer, in epidemiologic studies that include whole dietary surveys, maternal consumption of cured meats is the factor most carefully related to increased risk of CBT (Bunin et al., 1993; Kuijten et al., 1990; Preston-Martin et al., 1982) [4]

5. Pesticides:

Pesticides are universally present in our environment and are suspected of being related to various health problem. Numerous epidemiologic investigations have attempted to assess whether pesticide exposure increases the risk of cancer development, with the majority reporting positive associations (Zahm and Ward, 1998). In such studies, the hazards of all cancers consistently tend to be higher in studies of exposed children than in exposed adult

Although these compounds are not considered accepted risk factors for the development of CBT, their experimentally verified ability to access the foetus and potentially cause DNA damage suggests that other pesticide compounds may be able to do so as well. [4]

6. Tobacco

Some of the chemical component of tobacco smoke have been established to be carcinogenic in animals and in human, such as polycyclic aromatic hydrocarbons (PAHs) and nitrosamines (Autrup, 1993) [4]

7. **Electromagnetic frequencies**:

Exposure to electromagnetic fields is a potential risk factor for CBT that has also been evaluated in multiple epidemiologic studies. This association was first hypothesized in 1979 in Denver when it was informed that children who resided close to high current power lines had an increased risk of dying from leukaemia or brain cancer (Wertheimer and Leeper, 1979). [4]

8. **Infectious agents**:

Some cancer investigators believe that approximately 15% of all human malignancies can be attributed to bacterial, viral, or parasitic infections (Anderson et al., 2000). These infections and associated cancers include various types of papilloma viruses in cervical cancer, hepatitis B and C in hepatocellular carcinoma, HTLV- 1 in T-cell leukaemia/lymphoma; Epstein –Barr virus in Burkitt lymphoma, Helicobacter pylori in gastric cancer, HHV-8 in Kaposi sarcoma, and Schistosoma haematobium in bladder cancer (Anderson et al., 2000; Hall and Peckham, 1997) [4]

9. **Medications**:

The use of medications throughout pregnancy and early childhood has also been investigated for a possible epidemiologic relationship through CBT. A large retrospective cohort study in Tennessee assessed in utero exposure to metronidazole and the effect on rates of childhood cancers in children under 5 years of age (Thapa et al., 1998). The trade name for metronidazole is Flagyl, and it is an antibiotic used to treat bacterial vaginosis and trichomoniasis among other conditions.

In this population, it is one of the 10 most commonly prescribed drugs in pregnancy (Piper and Mitchel, 1991; Thapa et al., 1998). Flagyl has conventionally been prescribed regularly in pregnancy because untreated bacterial vaginosis and trichomoniasis have been linked to preterm labour (Carey et al., 2003). Metronidazole is a artificial compound, and it is believed to have short-lived active nitro-reduced metabolites that reason structural DNA damage, cell functional impairment, and cell death in microbes (Thapa et al., 1998). Metronidazole eagerly crosses the placenta and accesses the fetal circulation. The investigators found an insignificant increased risk for neuroblastomas in offspring exposed in utero to metronidazole with a relative risk (RR) of 2.60 (95% CI = 0.89 – 7.59). They found no association with all cancers combined (RR = 0.81, 95% CI = 0.41– 1.59) or with CNS tumors (RR = 1.23, 95% CI = 0.29 – 5.21).[4]

5. SECONDARY CARDIAC TUMOR

The incidence of cardiac metastases is rising, probably due to the increasing incidence of cancer and the fact that patients are alive longer with their disease. The improvement in diagnostic methods such as echocardiography and magnetic resonance imaging (MRI) makes it possible to detect primary cardiac metastases with or without clinical symptoms. The incidence of secondary cardiac tumors ranges up to 20% in adult. However, information on metastatic participation of the cardiovascular system in childhood is scanty. The pattern of secondary cardiac tumors in children seem different from that in adults because of the rarity of carcinomas and malignant melanoma in children and of neuroblastoma in adults. The purpose of this learning was to describe the clinical features and therapeutic experience of eight cases of cardiac metastases. These were either suspected as a result of their cardiovascular manifestations (six cases) or found incidentally by radiological studies such as computed tomography (two cases).

Patients and Method On retrospective examination of the medical record at Seoul National University Children's Hospital between January 1986 and October 1997, eight cases of secondary cardiac tumor were found. Patient consisted of five males and three females, ranging in age from 2 to 11

Cardiac rhabdomyoma is kind of tumor and generally seen in child is revealed in below figure [22]

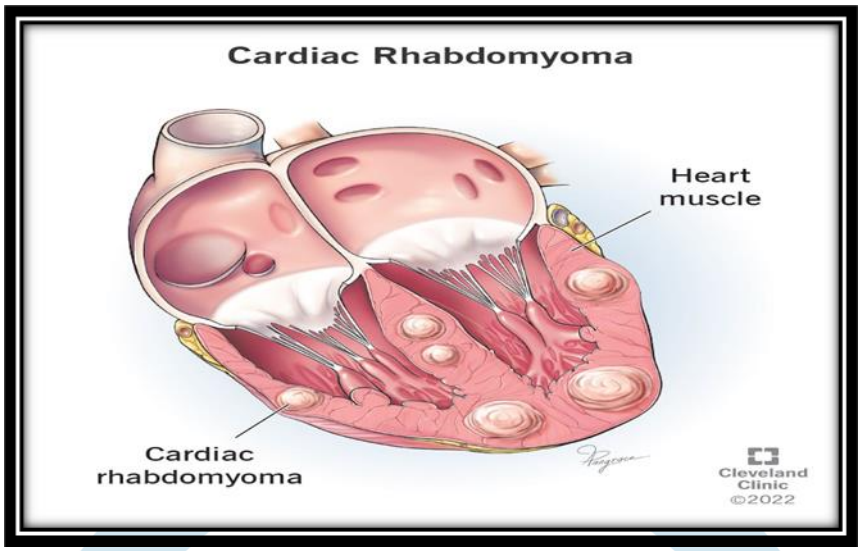

Fig. 5.1: cardiac rhabdomyoma

Table 5.1. Clinical and radiological findings of secondary cardiac tumors [13]

Case no.	Age at diagnosis	Sex	Cardiovascular symptoms	Radiological findings
1	2 years	M	Asymptomatic	MRI: right kidney mass and IVC thrombosis-like lesion extending to right atrium
2	3 years	M	Asymptomatic	CT: right kidney mass and IVC thrombosis-like lesion extending to right atrium
3	2 years 10 months	F	Dyspnea	CT: right thoracic and mediastinal mass
4	4 years 8 months	F	Right chest pain and cough	CT: huge right thoracic mass
5	4 years 11 months	M	Dyspnea	CT: right adrenal mass extending via the IVC to the right atrium
6	3 years 2 months	F	Chest pain and tachypnea, respiratory distress	Cardiomegaly
7	11 years	M	Dyspnea	MRI: thickening of right ventricle, RVOT, interventricular septum
8	3 years	M	Dyspnea on exertion and cough	Mediastinal widening, cardiomegaly

M, male; F, female; MRI, magnetic resonance imaging; IVC, inferior vena cava; CT, computed tomography; RVOT, right ventricular outflow tract.

Table 5.2. Clinical diagnosis and outcome of secondary cardiac tumors [13]

Case no.	Echocardiography	Initial treatment	Outcome	Pathology
1	Mass in RA	Chemotherapy: response (+)	Death: multiple metastases after 18 months	Wilms' tumor
2	Mass in RA nearly obstructing the inflow of RV	Surgical removal	Alive: follow-up for 7 months	Wilms' tumor
3	SVC obstruction; masses in LA, right pulmonary artery and vein	Chemotherapy: response (+)	Death: PCP after 11 weeks	Pleuropulmonary blastoma
4	Mass in LA obstructing the inflow of LV	Surgical removal	Death: PCP after 3 months	Malignant teratoma
5	Mass in RA	Chemotherapy: no response	Death: multiple metastases after 6 months	Neuroblastoma
6	Multiple tumors in RA, interatrial septum, RVOT, and posterior septal wall of LV; pericardial effusion	Surgical removal	Death: postoperative deterioration 2 days later	Lymphoma, Burkitt type
7	Mass in RVOT; thickening of free wall and septum of RV; pericardial effusion	Surgical removal	Death: immediately after operation	Lymphoma
8	Narrowing of right pulmonary artery and compression of LA and LV	Exploratory operation	Death: postoperative deterioration 1 week later	Lymphoma, Burkitt type

RV, right ventricle; SVC, superior vena cava; RA, right atrium; RVOT, right ventricular outflow tract; LA, left atrium; LV, left ventricle; PCP, *Pneumocystis carinii* pneumonia.

- **Location and Extension of Tumors**

Principal sites of tumors in our patients were the right adrenal gland in one case (case 5), the right kidney in two cases (case 1 and 2) and the mediastinum in five cases (cases 3, 4, 6, 7, and 8). Intracardiac masses were diagnosed by echocardiography in seven cases. The patient with lymphoma (case 8) showed no definite intracardiac mass; instead, a compressed left ventricle, left atrium and right pulmonary artery were seen on echocardiography. The extension and metastasis of the tumor were assessed by computed tomography (CT) in four cases and magnetic resonance imaging (MRI) in two cases

- Case 1. In this case of Wilms' tumor, MRI (coronal image) shows a huge right renal tumor with cystic portion. The tumor extends to the inferior vena cava and right atrium.
- Case 2. Apical four-chamber view echocardiogram in a child with Wilms' tumor demonstrating a large echo-dense mass in the right atrium

- **Pathology and Outcome:**

- Pathological studied revealed malignant teratoma (one), neuroblastoma (one), pleuropulmonary blastoma (one), Wilms' tumor (two), and non-Hodgkin's lymphoma (three). Initially, three patients (cases 1, 3, and 5) were treated with chemotherapy and the other five (cases 2, 4, 6, 7, and 8) with operative removal of the cardiac tumor. In each case of Wilms' tumor (case 1) and pleuropulmonary blastoma (case 3), chemotherapy resulted in the disappearance of the intracardiac mass. In the case of neuroblastoma, chemotherapy left a residual mass in the right atrium that was detected later when the operation to remove residual adrenal mass and inferior vena caval extension was performed (case 5). The initial operations were performed to relieve obstruction (cases 2, 4, 6, and 7) or to detect tumor pathology (case 8). Except in the patient with Burkitt's lymphoma (case 8), as far as possible of the cardiac mass was removed at operation. In case 8 the mass was left intact because of an undiagnosed penetration into the left atrium. Chemotherapy after the operation resulted in a significant reduction in the size of the mass [13]

Conclusions:

Paediatric brain tumors vary considerably in their histological, topographical and gender distribution throughout childhood and adolescence, reflecting different dynamics of individual tumor objects as well as a susceptibility to their occurrence during certain aeras of a child's life**,** IMT is a benign neoplasm rarely presented with malignant features such as local invasiveness, recurrence, distant metastasis, or malignant transformation. IMT can be suspected preoperatively through some hematologic abnormalities and Epithelial salivary tumor was rare, parotid gland was most common involved site, pleomorphic adenoma and mucoepidermoid carcinoma were the most common benign and malignant tumor respectively

Current procedures conducted by Pediatric oncology groups are beginning to incorporate biologic features to stratify patients for therapy. Treatment strategies continue to focus on limiting late things of treatment while maintaining an excellent survival. Patients with cardiopulmonary symptoms the dyspnoea, tachypnoea or chest pain were thought to result from their deteriorating general condition. The above article concludes about all plain tumors in childhood such as willms tumor, cardiac tumor, salivary gland tumor, brain tumor and IMT tumor will causes very harmful for child body and sometime death will occurs.

REFERANCES:

1. Willam M. CRist, MD And larry E. KUN, MD, COMMON SOLIDS TUMORS IN CHILDHOOD, THE NEW ENGLAND JOURNAL OF MEDICINE, Feb.14,1991, pageno.461-471
2. Matthew R. Grimmer and William A. Weiss, Childhood tumors of the nervous system as disorders of normal development, © Lippincott Williams & Wilkins, page 634-638
3. A. OLUFEMI WILLIAMS, MD, TUMORS OF CHILDHOOD IN IBADAN, NIGERIA, CHILDHOOD TUMORS IN NIGERIA Williams, Cancer 36370-378, 1975
4. Rachel Tobias Baldwina and Susan Preston-Martinb, Epidemiology of brain tumors in childhood—a review, Toxicology and Applied Pharmacology 199 (2004) 118 – 131
5. Christian H. Rickert Werner Paulus, Epidemiology of central nervous system tumors in childhood and adolescence based on the new WHO classification, Child's Nerv Syst (2001) 17:503–51
6. Bahadur, G. (2000). " Age definitions, childhood and adolescent cancers in relation toreproductive issues". Human Reproduction 15: 227-221. doi:10.1093/humrep/15.1.227.
7. Childhood Cancers: Basic Facts & Figures from Minnesota Department of HealthRetrieved Dec, 2012.
8. Arora RS, Eden TOB1, Kapoor G2, Epidemiology of childhood cancer in India, Indian Journal of Cancer | October–December 2009 | Volume 46 | Issue 4-page no. 264-273
9. J. E. W. M. VAN DONGEN-MELMAN and J. A. R. SANDERS-WOUD, PSYCHOSOCIAL ASPECTS OF CHILDHOOD CANCER: A REVIEW OF THE LITERATURE*, J Child Psyhol Psychmt. Vol. 27. No. 2, pp. 145-180, 1986 Printed in Great Britain.
10. Edmund Y. Ko, Michael L. Ritchey*, Current management of Wilms' tumor in children, Journal of Paediatric Urology (2009) 5, 56e65
11. W. Stites Whatley, MD, Jerome W. Thompson, MD, MBA, and Baskar Rao, MD, Memphis, Tennessee, Salivary Gland Tumors in Survivors of Childhood Cancer, Otolaryngology–Head and Neck Surgery, Vol 134, No 3, March 2006, page no. 385-388
12. I˙brahim Karnak, Mehmet Emin S¸enocak, Arbay O. Ciftci, Melda C¸agˇlar, Meltem Bingo¨l-Kologˇlu, F. Cahit Tanyel, and Nebil Bu¨yu¨kpamukc¸u Ankara, Turkey, Inflammatory Myofibroblastic Tumor in Children: Diagnosis and Treatment, Journal of Pediatric Surgery, Vol 36, No 6 (June), 2001: pp 908-912
13. J. Huh,1 C.I. Noh,1 Y.W. Kim,1 J.Y. Choi,1 Y.S. Yun,1 H.Y. Shin,2 H.S. Ahn,2 Y.J. Kim3, Secondary Cardiac Tumor in Children, Pediatric Cardiology © Springer-Verlag New York Inc. 1999, page no. 400-403

14. Birch JM. Li-Frameni Syndrome. Eur. J. Cancer 1994;30A:1935-41
15. Stiller CA, Allen MB, Eatock EM. Childhood Cancer in Britain: The National Registry of Childhood Tumours and Incidence Rates 1978-1987. European Journal of Cancer 1995; 31A:2028-2034
16. . Lukens JN. Progress Resulting from Clinical Trials: Solid Tumours in Childhood Cancer. Cancer 1994;74:2710-8
17. Draper G, Kroll ME, Stiller C. Childhood Cancer. In: Trends in Cancer Incidence and Mortality. Cancer Surveys 1994;19,493-51
18. EL B. CASTRO, MD,' ANDREW G. HUVOS, MD, ELLIOT w. STRONG, MD, FACS, ~ AND FRANK W. FOOTE, JR., MD~, TUMORS OF THE MAJOR SALIVARY GLANDS IN CHILDREN, SALIVARY GLANDS IN CHILDREN Custro et al, Vol. 29-page no. 312-317
19. Qi-Gen Fang a, b, Shuang Shi c, Zhen-Ning Li a, b, Xu Zhang a, b, Fa-Yu Liu a, b, Chang-Fu Sun a, b,, Epithelial salivary gland tumors in children: A twenty-five-year experience of 122 patients, International Journal of Pediatric Otorhinolaryngology, G Model PEDOT-6634; No. of Pages 3
20. https://www.researchgate.net/figure/Clinical-photograph-showing-classical-L-parotid-tumour-in-a-child_fig4_325683542
21. https://www.physio-pedia.com/images/5/5f/Wilms_Tumor.jpg
22. https://images.app.goo.gl/aW1VMntch3jY4xhZ7
23. https://images.app.goo.gl/VL3B2p3Ps1HFW8mB8