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Abstract

Introduction: Disseminated intravascular coagulation or consumption coagulopathy is a well-recognized entity both in various malignant and non-malignant conditions. Most data in paediatric solid tumours are isolated case reports, while there is sparse data in paediatric acute leukaemia.

Objective: The study was conducted to analyze the incidence of DIC in our population of paediatric solid tumours.

Design: All records of children <15 years of age registered in the Paediatric Oncology department of our institute with a diagnosis of solid tumour malignancy were retrospectively reviewed for evidence of DIC.

Results: Out of the 73 children, 4 have developed DIC, an incidence of 5.5%. The mean age of children who developed DIC was 4.6 years (Range- 2months–15 years) and the majority (2/4–50%) children were less than 1 year of age. Children with DIC had a male predominance (75%) and the majority (75%) presented in advanced stages of the disease. Of the 10 children with neuroblastoma, 2 (20%) had evidence of DIC. Statistical analysis was done to determine whether any patient characteristic had the propensity to develop DIC. The only factor that attained statistical significance was younger age.

Conclusion: Disseminated intravascular coagulation though uncommon in children should always be thought of in a child with advanced disease presenting with thrombocytopenia or clinical manifestations of bleeding tendency. An index of suspicion is important for early diagnosis and emergent treatment which eventually improves survival.

Keywords: DIC, Consumption coagulopathy, Paediatric solid tumours.

Introduction

Consumption coagulopathy (Disseminated Intravascular Coagulation or DIC) is a clinicopathological entity characterized by activation of the coagulation cascade in the systemic circulation causing decreased levels of procoagulant factors and platelets. It has been known since 19th century; initially detected in animals as widespread clot activation in circulation. But it was only in 2001 that the Scientific Subcommittee on Disseminated Intravascular Coagulation (DIC) of the International Society on Thrombosis and Hemostasis proposed a consensus statement on the definition of DIC – “An acquired syndrome characterized by the intravascular activation of coagulation with loss of localization arising from different causes. It can originate from and cause damage to the microvasculature, which if sufficiently severe, can produce organ dysfunction.” [1]

DIC is a well-recognized entity both in various malignant and non-malignant conditions like infections, vascular disorders, obstetric complications, tissue injury etc. It can occur in haematological malignancies and solid tumours. [2] Though relatively uncommon in adult general practice, a large retrospective series suggests an overall incidence of 0.1% from a general hospital. Of all the causes, malignancy constituted around 6.8%. [3] In various series in adult solid tumours, the incidence of DIC has varied between 6–15%. [4,5] It is higher in acute leukaemias with a very high incidence of around 90% in acute promyelocytic leukaemia. [6] In paediatric general practice, the incidence is around 0.4–1% with sepsis accounting for 95% of all

Corresponding author: Dr. Yamini Krishnan, MD (Paediatrics), DCH DM, Senior Consultant, Department of Paediatric Oncology, MVR Cancer Centre and Research Institute, CP13/516 BC, Vellalasseri, REC (via), Poolacode, Kozhikode, Kerala – 673601, Tel. +919446981696, Email: dryamini@mvrccri.co
cases.\(^7\) Most data in paediatric solid tumours are isolated case reports, while there is sparse data in paediatric acute lymphoblastic leukaemia which have varied between 3–5%.\(^8,9\) We wanted to investigate whether the reason for the very low reporting of DIC is actually because of the very low incidence that it becomes insignificant in the paediatric population. We reviewed our database retrospectively to analyze the incidence of DIC in our population of paediatric solid tumours.

### Materials and Methods

All medical records of children less than 15 years of age registered in the Paediatric Oncology department of MVR Cancer Centre and Research Institute with a diagnosis of solid tumour malignancy were retrospectively reviewed for evidence of DIC. The study was conducted from July 1, 2017, to June 30 2018. The Institutional Review Board of the hospital approved the study on April 19, 2018. The diagnosis of DIC was made if the child had raised coagulation factors (PT, APTT), a low platelet count and decreased fibrinogen level without any evidence of organ dysfunction or sepsis.

The various descriptive parameters were retrieved from the electronic medical records. Bleeding episodes were graded according to WHO criteria: Grade – 1 Mild mucocutaneous bleeding not requiring any intervention, Grade 2 – Major bleeding episodes including malaena, hematuria, hemoptysis, retinal bleed with visual impairment. Grade 3 – Any Grade 2 bleeding with RBC transfusion requirement. Details of treatment received including blood component support and chemotherapy schedules were recorded from the patient records. DIC was graded according to CTCAE v5 – Grade 2 Laboratory findings with no bleeding, Grade 3 Laboratory findings with bleeding, Grade 4 Life-threatening DIC Grade 5 – Death.

Statistical Analysis was done using the statistical software for social sciences (SPSS). X2 test and t-test was done for determining statistical significance. A p value of less than 0.05 was taken as significant.

### Results

73 records of children registered at our Centre from July 1, 2017, to June 30, 2018, were reviewed for evidence of consumption coagulopathy. Patient characteristics are shown in Table 1. The mean age group was 7 years (Range: 2 months – 15 years), with the majority of children in the age group of 5–15 years (57.6%). Male children constituted 46.5% (34/73) and females 53.5% (39/73). The majority of children had brain tumours (17.9%) followed by neuroblastoma (13.7%). 47 (64.4%) children had early–stage disease while rest were either metastatic or relapsed and refractory. Out of the 73 children, 4 children had developed DIC, an incidence of 5.5%. The mean age of the children who developed DIC was 4.6 years (Range: 2 months –15 years) and the majority (2/4– 50%) children were less than 1 year of age. Children with DIC had a male predominance (75%) and the majority (75%) presented in advanced stages of the disease. Out of the 10 children with neuroblastoma, 2 (20%) had evidence of DIC. Details of the 4 children are described in Table 2.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Number (N)</th>
<th>Percentage (%)</th>
<th>DIC (N) %</th>
<th>p value</th>
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<tr>
<td>Age</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 1 year</td>
<td>6</td>
<td>8.2</td>
<td>2</td>
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<tr>
<td>1–5 years</td>
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<td>4</td>
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<td>5–15 years</td>
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<td>4</td>
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<tr>
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<tr>
<td>Male</td>
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<tr>
<td>Female</td>
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<td>53.5</td>
<td>1</td>
<td>2.4</td>
</tr>
<tr>
<td>Diagnosis</td>
<td></td>
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<tr>
<td>Brain tumours</td>
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<td>0</td>
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<td>Neuroblastoma</td>
<td>10</td>
<td>13.7</td>
<td>2</td>
<td>20</td>
</tr>
<tr>
<td>Wims tumour</td>
<td>7</td>
<td>9.6</td>
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<td>0</td>
</tr>
<tr>
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<td>9.6</td>
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<td>0</td>
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<td>LCH</td>
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<td>Germ cell tumour</td>
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<tr>
<td>Hepatoblastoma</td>
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<tr>
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<td>Adrenocortical ca</td>
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<td>0</td>
</tr>
<tr>
<td>Ca parotid</td>
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<td>1.4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Ca thyroid</td>
<td>1</td>
<td>1.4</td>
<td>0</td>
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</table>

Table 1 – Patient characteristics
Consumption coagulopathy in paediatric solid tumours, Yamini Krishnan, et. al.

<table>
<thead>
<tr>
<th>SI No</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
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<tbody>
<tr>
<td>Age / Sex</td>
<td>3 months / Male</td>
<td>3 years / Female</td>
<td>2 months / Male</td>
<td>15 years / Male</td>
</tr>
<tr>
<td>Pathology</td>
<td>Neuroblastoma</td>
<td>Neuroblastoma</td>
<td>KHE</td>
<td>Alveolar RMS</td>
</tr>
<tr>
<td>Site of Primary</td>
<td>Cervical mass</td>
<td>Adrenal</td>
<td>Right Thigh</td>
<td>Left Leg</td>
</tr>
<tr>
<td>Stage of Disease</td>
<td>IVS</td>
<td>III</td>
<td>I</td>
<td>IV</td>
</tr>
<tr>
<td>Metastatic/ Non-metastatic</td>
<td>Metastatic</td>
<td>Non Metastatic</td>
<td>Non Metastatic</td>
<td>Metastatic</td>
</tr>
<tr>
<td>Site of Metastasis</td>
<td>Liver</td>
<td>Bone marrow</td>
<td>Nil</td>
<td>Bone marrow, lymph nodes, bone, lung</td>
</tr>
<tr>
<td>Laboratory Parameter</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Platelet count</td>
<td>72,000</td>
<td>9,000</td>
<td>7000</td>
<td>90,000</td>
</tr>
<tr>
<td>PTI / INR</td>
<td>&gt;70/high</td>
<td>33.2/3.16</td>
<td>15.2/1.38</td>
<td>19.5/1.52</td>
</tr>
<tr>
<td>APTT</td>
<td>&gt;120/high</td>
<td>51.6</td>
<td>44</td>
<td>40</td>
</tr>
<tr>
<td>Fibrinogen /LDH</td>
<td>10mg/dl</td>
<td>50</td>
<td>62</td>
<td>90/1858</td>
</tr>
<tr>
<td>Timing of DIC</td>
<td>At diagnosis</td>
<td>On treatment</td>
<td>At diagnosis</td>
<td>At diagnosis</td>
</tr>
<tr>
<td>Type of DIC Grade</td>
<td>Overt Grade–5</td>
<td>Overt Grade–3</td>
<td>Non–overt Grade–2</td>
<td>Non–overt Grade–2</td>
</tr>
<tr>
<td>Signs of bleeding Grade</td>
<td>Malaena Grade–3</td>
<td>Cutaneous and mucosal bleed Grade–3</td>
<td>Nil Grade–0</td>
<td>Nil Grade–0</td>
</tr>
<tr>
<td>Chemotherapy Given</td>
<td>Carboplatin Etoposide</td>
<td>Vincristine adriamycin cyclophosphamide</td>
<td>Vincristine steroids propranolol</td>
<td>Supportive care</td>
</tr>
<tr>
<td>Transfusion</td>
<td>Platelets FFP Cryoprecipitate</td>
<td>Platelets Cryoprecipitate</td>
<td>FFP Platelets Cryoprecipitate</td>
<td>Nil</td>
</tr>
<tr>
<td>Grade</td>
<td>Expired</td>
<td>Developed chronic DIC</td>
<td>Recovered</td>
<td>Expired</td>
</tr>
</tbody>
</table>

Table 2– Patient characteristics of children who presented with DIC

**Patient 1**

3–month–old baby presented to us in a moribund state with breathlessness and melaena. Clinically child had a massive hepatomegaly and cervical mass with Horner’s syndrome. The biopsy was suggestive of neuroblastoma with bone marrow positivity (<10%) with a final diagnosis of neuroblastoma Stage IVS. Blood investigations revealed DIC and treatment was initiated on day 2 of admission with carboplatin and etoposide. But the child expired of disease on day 10 of admission.

**Patient 2**

A 3–year–old child who was diagnosed as Neuroblastoma Stage III and initiated on chemotherapy presented to us with extensive skin and mucosal bleeds; 3 weeks after the second cycle of chemotherapy. Blood investigations were suggestive of DIC and supportive treatment was given. The child improved from the acute DIC and later investigations revealed chronic non–overt DIC.

**Patient 3**

A 2–month–old child presented to us with rapidly enlarging vascular lesion in the thigh extending up to the foot. Blood investigations revealed consumption coagulopathy: final diagnosis of Kaposiform Hemangioendothelioma (KHE) with Kasabach Merritt syndrome. He was started on vincristine, steroids and propranolol. DIC got corrected in 3 weeks and the child remains to be in remission after 1 year.

**Patient 4**

A 15–year–old boy was investigated for a leg swelling with extensive inguinal adenopathy. Staging investigation with PET CT showed an extensive involvement of bone marrow, nodes and lung metastasis. The biopsy was suggestive of alveolar RMS and blood investigations revealed non–overt DIC. Poor prognosis was explained to the parents and they opted for supportive care only.

Statistical analysis was done to determine whether any patient characteristic had the propensity to develop DIC. The only factor that attained statistical significance was age (p–value 0.007) but the mean age did not attain significance.

**Discussion**

In 1865 Trousseau described the association of thrombo–hemorrhagic disorders in solid tumours. Over the years it became well–established and the landmark paper by Sack
Some neoplasms are also known to secrete coagulation pathways may also be activated. A smaller study from Japan, of the 44 children with ALL is also well described in literature. Willebrand disease in children with Wilms tumour is also known complication of Kasabach–Merritt syndrome. The association of acquired von Willebrand disease in children with Wilms tumour is also known as Kasabach–Merritt syndrome.

DIC described with neuroblastoma, Wilms tumour, RMS, and sacrococcygeal teratoma etc. Consumption coagulopathy is also a known complication of KHE known as Kasabach–Merritt syndrome.

Among 805 children with acute lymphoblastic leukaemia and 195 children with acute myeloid leukaemia studied, 25 (3.1%) and 27 (13.8%) respectively have coagulopathy. In a smaller study from Japan, of the 44 children with ALL evaluated, 2 (5%) had DIC. Another study from India done in both adult and paediatric acute leukaemia, DIC was seen in 5.8% of children. Most data in paediatric solid tumours are case reports and there are no large prospective or retrospective studies. Earliest series in children is from Sills RH et al; in which the authors describe the prevalence of DIC in advanced patients with Ewing’s sarcoma and rhabdomyosarcoma. Another series in neuroblastoma has been reported by Hatae Y et al. Out of the 30 neuroblastoma children, they studied 6 (2%) had DIC and all the children had Stage IV disease. In a small study of 40 children with advanced solid tumours, no child was found to have consumption coagulopathy and the authors hence concluded that DIC in paediatric solid tumours is uncommon. In our study, 4 out of the 73 children studied had DIC; 2 with neuroblastoma and 1 child each with RMS and KHE. Our incidence is almost near the average DIC rates seen in the adult population probably because 36.6% of the children studied had advanced disease. There have been lots of case reports in paediatric DIC described with neuroblastoma, Wilms tumour, RMS, congenital mesoblastic nephroma, glioblastoma, infantile fibrosarcoma, multi-system Langerhans cell histiocytosis, sacrococcygeal teratoma etc. Consumption coagulopathy is also a known complication of KHE known as Kasabach–Merritt syndrome. The association of acquired von Willebrand disease in children with Wilms tumour is also well described in literature.

In clinical practice, DIC can be divided into overt DIC (decompensated) and non–overt DIC (non–decompensated DIC). When compensation occurs usually clinical symptoms appear along with laboratory features depending on the rapidity of depletion of platelets and coagulation factors. In the majority of solid tumours, DIC is usually a chronic slow process and laboratory findings may suggest a low normal platelet count and even a normal fibrinogen with no clinical signs or symptoms. Non–overt DIC is difficult to diagnose and an index of suspicion may be required. Aggressive treatment of the local condition in an emergent manner is needed. Some solid tumours may present with overt DIC. In our series of children, 2 children had acute overt DIC and 2 with chronic non–overt DIC. One child with neuroblastoma after treatment had ongoing non–overt chronic DIC.

Though DIC was a recognized entity, until the late 1980’s there was no universal definition or scoring system. It was in 1988 that the Japanese Ministry of Health and Welfare (JMHW) put forward the first universally accepted scoring system. By the early 2000’s the International Society on Thrombosis and Haemostasis (ISTH) published a score for overt and non–overt DIC. Although triple tests of prothrombin time (PT), platelet count and fibrinogen in the appropriate clinical setting are enough to suspect DIC in the absence of liver disease and massive transfusion, most scoring systems incorporate fibrin degradation products and D–dimer. Some scores are qualitative and some have quantitative values. The data of validation in children is limited especially in the critical care setting. Whether it is useful in paediatric malignancies is also not clear. The limitation of our study is that we only had results of PT, APTT, platelet count and fibrinogen in the absence of liver disease, sepsis and massive transfusion for diagnosis of DIC due to logistics of getting the FDP and D–dimer done. But all our children had a score of 7 or more by the JMHW scoring protocol.

The pathophysiology described in a tumour related consumption coagulopathy is the production of tissue factor by the tumour cells or on the surface of the monocyte–macrophage system. Tissue factor can also be expressed by the endothelial cells. The tissue factor, in turn, activates factor VII followed by factor IX and X. It is also postulated that the tumour microemboli that enter circulation act as thromboplastins and trigger the coagulation cascade. Some neoplasms are also known to secrete Factor X activating cysteine protease and thus inducing coagulation. Coagulation pathways may also be activated by pro–inflammatory cytokines and tumour necrosis. A hypofibrinolytic state due to stimulation of a fibrinolytic inhibitor also has been described. The final consequence is the uncontrolled production of thrombin. Whether the patient develops overt or non–overt DIC depends on the compensatory responses like the ability to enhance platelet or coagulation factor production, fibrinolysis and the abilities of the clearance mechanisms. Some patients may have very little signs and symptoms, whereas other patients with these defective compensatory responses may have extensive bleeds and thrombosis. Older male patients with advanced malignancies and extensive necrosis have
been found to be of having a high risk for development of DIC in the adult population. In our series, though the majority had advanced disease at presentation, the stage at presentation did not attain significance. Younger age (<1 year) was found to have a higher propensity for DIC though it requires a larger study for confirmation.

The prognosis of patients presenting with DIC has been shown to be worse than those who had no evidence of DIC. In the study by Sallah et al, patients with advanced solid tumour malignancies who presented with DIC had a median survival of 9 months when compared to those who did not present with coagulopathy (median survival 14 months). The author further demonstrated the same in early-stage disease (median survival 16 months vs 44 months). It was concluded in the study that DIC was an independent prognostic factor for survival regardless of the stage of presentation. Literature in paediatric solid tumours also have suggested the same. Bien et al in his series of patients with a literature review of other case reports has concluded that the prognosis of RMS children with bone marrow involvement and DIC is poor and an index of suspicion and early initiation of chemotherapy can probably improve survival. But in his review from 1978–2010, 10 out of the 11 children with bone marrow metastasis and DIC recovered from the coagulopathy within 6 days to after 3 cycles of chemotherapy. Whether this poor survival is applicable to early stage paediatric tumours is not clear. In our study, one child with Kaposiform hemangioendothelioma had complete resolution of DIC after initiation of chemotherapy with vincristine. One of the children with Neuroblastoma had a conversion of an overt DIC to chronic DIC over the follow-up period. In a child with Neuroblastoma IV S who presented in a moribund state, the child did not survive the episode in spite of initiation of chemotherapy. The child with metastatic RMS was given only supportive care and succumbed to his illness.

An index of suspicion is important in the early diagnosis of coagulopathy. Most protocols advise a baseline coagulation profile in children before initiation of treatment. Urgent initiation of treatment of the primary disorder can result in control of DIC. Other supportive therapies may be required once treatment is initiated. Although thrombocytopenia and raised coagulation factors can lead to an increase in the risk of bleeding, blood component support including platelets, plasma and cryoprecipitate is not indicated unless there is clinical evidence of bleeding. Aggressive correction of laboratory parameters is not beneficial. Though anticoagulant therapy may be theoretically beneficial in treating tumour associated DIC, in clinical practice safety and efficacy of the strategy has never been proven.

**Conclusion**

Disseminated intravascular coagulation though uncommon in children should always be thought of in a child with advanced disease presenting with thrombocytopenia or clinical manifestations of bleeding tendency. An index of suspicion is important for early diagnosis. Emergent initiation of treatment of the primary is the key to improving survival. We would recommend a baseline coagulation profile for all children presenting with malignancy.

**References**


