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Perspective

Effect of Thalidomide Drug in Women during Pregnancy

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DESCRIPTION

Thalidomide is a drug that alters the immune reaction of the body and reduces the body's capability to produce new blood vessels. Women who are pregnant or who might get pregnant while taking thalidomide shouldn't take it. Thalidomide use during pregnancy can result in the death of the unborn child or serious birth abnormalities (physical issues that are present in the newborn at birth).The majority of organ systems develop during the embryonic stage, often known as the "critical phase," whereas the neonatal phase, which persists from week eight through delivery, is when the body systems mature and are developed. Toxins like thalidomide have the potential to severely impair important developmental stages in the embryo during the window of vulnerability.

In October 1957, the German pharmaceutical company Chemie Grunenthal launched mass-producing thalidomide. It was promoted as Contergan, a perfectly safe, non-addictive medication that may be administered to pregnant women as a moderate sedative or treatment for morning sickness. However as time passed on and the medication's acceptability increased, concerns about public health and safety also began to surface. In 1961, physician independently discovered a significant frequency of serious birth malformations in children delivered to mothers who had taken the medication. This was the first sign that something might be happening. Phocomelia, which refers to a significant shortening or absence of limb structures, was one of the most frequently seen malformations. Other abnormalities were noticed as well, such as the absence or malformation of the thumbs or ears, hip dislocation, heart issues, and abnormal intestines. Women who took thalidomide gave birth to more than 7,000 affected children, and even one tablet was sufficient to cause a kid to have abnormalities in all four limbs. The children who have been born when the drug was thought to be safe are still suffering the repercussions of the medical epidemic fifty years later.

Thalidomide's embryotoxic ability to get around a fundamentally essential embryonic defence mechanism, which is in charge of keeping toxic substances out of embryonic cells and escorting tagged toxicants out of the cell, is one of the methods by which it causes birth abnormalities. Efflux transporters situated in the cytoplasmic membrane conduct these essential cellular homeostasis tasks. Efflux transporters are ATP-Binding Cassette (ABC) family of proteins members that utilize ATP hydrolysis to provide energy for the translocation of hazardous substances out of the cell through primary active transportation. The ability of these proteins to recognize and interact with the given chemical is entirely dependent upon the transport system, despite the fact that that it is generally quite efficient. However, because thalidomide is not detected by the transporters, binding is prevented, permitting the drug to stay within the cell. The Apical Ectodermal Ridge (AER), which is in responsible of limb bud growth, and the Zone of Polarising Activity (ZPA), which is in charge of establishing the anterior-posterior axis in the limb bud, are both capable of experiencing oxidative stress once thalidomide evades the efflux transport system. Thalidomide blocks angiogenesis at the molecular level by intercalating or inserting itself into DNA's Guanine-Cytosine (G-C) rich regions. The regulatory regions of the angiogenesis-inducing proteins Fibroblast Growth Factor-2 (FGF2) and Insulin Growth Factor-1 (IGF-1) are inhibited by thalidomide through this complexation. Another recognized theory for why embryonic limb buds stop developing is intercalation. This is as a result that interruption of the immature and quickly developing blood vessel networks can lead to limb truncation. Limb bud creation during embryonic development is highly angiogenic.

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