SAWA UNIVERSITY

Biological Radiation Hazards

Effects Of Radiation On Central Nervous System

Sawa University — جامعـــــة ســـــــاوة الاهلية

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3. Stage — المرحلـــــــــــة 3

الجانب النظري — محاضرة رقم 7

Theoretical — Lecture No. …7

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Biological Mechanism of Radiation Damage

CNS damage results from both **direct** and **indirect** effects of radiation:

- **Direct Effect:** Destruction of DNA in neurons and glial cells.
- **Indirect Effects:**
  - **Free Radical Formation:** Radiation interacts with water (the cell's main component), generating harmful molecules (free radicals) that damage cellular components.
  - **Endothelial Damage (Vascular Damage):** Injury to the inner lining of blood vessels is considered the **main mechanism** of radiation-induced Cerebral Vasculopathy, leading to ischemia, hemorrhage, and changes in permeability.

2. Clinical and Radiographic Manifestations (Radiographic Pathology)

The effects of radiation depend on the **total dose**, **fractionation schedule**, and the **patient's age** (children are more vulnerable due to the developing brain).
Radiographic changes are classified temporally:

Temporal Classification	Timeframe	Common Clinical Manifestations	Radiographic Findings (CT/MRI)
Acute Response	During treatment up to a few weeks	Nausea, vomiting, headache, somnolence, temporary memory loss.	**Acute Edema:** Increased T2/FLAIR signal in the white matter. Often transient and steroid-responsive.
Early Delayed Effect	1 to 6 months post-treatment	Drowsiness Syndrome, mild cognitive slowing.	**Transient Demyelination:** Diffuse or focal increase in T2/FLAIR signal in the white matter.
Late Delayed Effect	6 months to years	**1. Radionecrosis. 2. Cognitive Decline. 3. Stroke-like Syndromes.**	**1. Radionecrosis:** A **ring-enhancing mass** with surrounding edema and mass effect, **mimicking tumor recurrence**. Often requires advanced MRI (MRS/Perfusion) for differentiation. **2. Diffuse White Matter Injury (Leukoencephalopathy):** Hypoattenuation on CT and high T2/FLAIR signal in the periventricular white matter. **3. Radiation-Induced Cerebral Vasculopathy:** Vessel narrowing (Moyamoya pattern), cavernous malformations, capillary telangiectasias, hemorrhage.

3. Role of the Radiology Technologist in Evaluation

- **Differentiating Radionecrosis from Tumor Recurrence:**
  - **Advanced MRI Techniques:**
    - **Perfusion Imaging:** Radionecrosis typically shows **low relative Cerebral Blood Volume (rCBV)**, whereas tumor recurrence often shows **high rCBV** (due to neovascularization).
    - **Magnetic Resonance Spectroscopy (MRS):** Necrosis shows **decreased NAA/Cr** and **increased Lactate**, while tumor recurrence shows **decreased NAA/Cr** and **increased Choline/Creatine (Cho/Cr)**.
- **Assessing Spinal Cord Effects:**
  - **Delayed Myelopathy:** May occur in the spinal cord irradiated with high doses. Appears as a T2 signal change in the cord (swelling or atrophy).
  - **Bone Marrow Changes:** Fatty replacement of bone marrow in irradiated vertebrae (high T1 signal on MRI) is a common sign.

4. Management of Radiation Neurotoxicity

As radiology technologists, knowing the available treatment options helps you understand the patient's course:

- **Treatment of Cerebral Edema and Acute Necrosis:**
  - **Corticosteroids:** Such as Dexamethasone, are the first line of defense to reduce edema surrounding necrosis or acute white matter changes, thereby improving neurological symptoms.
- **Treatment of Chronic Radionecrosis:**
  - **Bevacizumab:** This monoclonal antibody targets Vascular Endothelial Growth Factor (VEGF) and is used to treat radionecrosis unresponsive to steroids. It works by reducing vascular permeability and fluid leakage.
  - **Hyperbaric Oxygen Therapy (HBOT):** Thought to improve perfusion in irradiated tissue and reduce edema, though evidence is variable.
  - **Surgical Resection or Stereotactic Radiosurgery (SRS):** Employed for cases resistant to medical therapy and those causing significant mass effect.

5. The Role of Positron Emission Tomography/Computed Tomography (PET/CT)

In difficult cases where advanced MRI alone cannot distinguish between necrosis and tumor recurrence, PET/CT can be utilized:
.Common Use (FDG-PET): Tumor recurrence typically shows increased glucose metabolism (High FDG uptake), whereas radionecrosis shows decreased metabolism or no uptake (Hypometabolic/Photopenic).
.Amino Acid PET: Using tracers like 11-C-Methionine or 18-F-FET, these often offer higher specificity; malignant tumors usually show higher and clearer uptake compared to radionecrosis.

QUIZ

- What are the two main mechanisms by which radiation causes damage to the Central Nervous System (CNS)?
- Describe the **Direct Effect** of radiation on CNS cells.
- Regarding **Indirect Effects**, how does **Free Radical Formation** lead to cellular damage?
- What key factors determine the effects of radiation, and why are children more vulnerable to CNS damage?
- What is the timeframe for the Acute Response, and what are the common radiographic findings (CT/MRI) associated with this phase?
- What is the common clinical syndrome associated with the Early Delayed Effect phase (1 to 6 months post-treatment)?
- List three clinical manifestations that occur during the Late Delayed Effect phase (6 months to years).?
- Describe the radiographic appearance (CT/MRI) of Radionecrosis, and what condition can it mimic?

Any questions?