# RADIOLOGICAL ACCIDENT DUE TO DIRECT EXPOSURE TO COBALT 60 SOURCE. FOLLOW-UP AFTER 16 YEARS

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# ABSTRACT

On November 16; 1995, an accident occurred at the radiotherapy department of the Goyeneche Hospital of Arequipa, Peru, that affected a maintenance man staff of 38 years of age (electrician). He was called to repair a radiotherapy machine Theratron with a Cobalt-60 source. The machine had suffered a malfunction in the source transport system, so he proceeded to re-introduce the source into the holder with the steel rod used for these cases. He then noticed that the flange plunger was broken, so he used a leaded apron and common gloves and held the source with his right hand for about five seconds to return it to its correct position. This case report is a summary of the monitoring of biological effects and results of medical studies after 16 years.

# 1. DESCRIPTION OF THE ACCIDENT

In November 1995 a radiological overexposure occurred in the radiotherapy service of the Goyeneche General Hospital in Arequipa Perú.

A 38-year old electrician was called to repair a problem in the Theratron 60 Cobalt machine. The source transport system had failed and the source did not return to the safe position. He first tried to do it with the steel rod provided for the purpose but it did not work. He then found out that flange plunger had broken. He put on a leaded apron and simple gloves, and held the source that was out of place with his hand and pushed it into position holding the source for approximately 5 seconds.

# 2. DOSIMETRY

The activity at the time of the accident was 1500 Ci and the estimated dose rate was 300 Gy/min. at 1 cm. distant from the source. Considering that the electrician pushed the source for 5 seconds, the dose received was in the order of 25 Gy.

In September 1999 (46 months after the accident), the medial finger was amputated and the bone was analyzed by ESR (Electron Spin Resonance) for the absorbed dose reconstruction, a dose of  $6.4 \pm 0.5$  Gy was estimated; this value is probably smaller than the real dose, nevertheless it can be accepted as the minimum dose received by the victim in the hand.

In addition, biological dosimetry with application of a correction factor, leads to the estimated dose of  $20\pm5$  Gy. For the irradiated region this can be inferred by fluorescence in situ hybridization (FISH), i.e., retrospective biological dosimetry performed in peripheral blood lymphocytes collected from the victim four years after the accident.

The compatibility of the results obtained by the physical and biological dosimetry methods reinforce the validity of each one, although other correction factors should be applied so as to consider many aspects, such as sampling time and elimination of damaged cells, among others. Although a long time had elapsed between the accident and the dosimetric analysis, a clear ESR signal due to irradiation in bone was measured. According to histological data and the medical history, blood flow in the finger was interrupted, compromising the bone turnover and remodeling.

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# 3. CONSEQUENCES OF THE ACCIDENT

The patient did not present prodromal symptoms like nausea or vomiting, but on the eighth day from the accident he started with progressive pain in the right hand joints. In the subsequent days the pain distributed all over the hand with increasing local temperature and pain that increased daily. On the third week after exposure he presented color changes in the hand. At day 23 he developed ampoules in the index middle and the small fingers extending to the palm with confluent superficial ulcerations.

At day 30, ulceration of the index and middle fingers is evidenced accompanied by great edema. At day 35 after the accident he is admitted to the radiotherapy department of the National Cancer Institute (Instituto Nacional de Enfermedades Neoplásicas, INEN) and started in topic and analgesic treatment (Fig. 1).

Hematological studies were done on day one and a study of bone marrow from the sternal bone and the iliac crest was performed. The result showed low cellularity in the sternum bone marrow sample. In January 1996 an X ray of the affected hand showed normal bone configuration. On February 21, 1996 it showed notable destructive changes and marked osteopenia. Three months after the accident the healing process continued and scarred atrophic skin was seen in the three first fingers (Fig. 2).





FIG. 1. Index finger of the right hand, loss of epitelial, and granulation signs (54 days after the accident).

FIG. 2. Three months after the accident, healing after further treatment, with atrophic skin. Index finger still with granulation.

Four months after the accident one can see edema of the first three fingers and ampoules at the level of the distal phalange of the second finger as well as necrosis of the central interphalange joint of the index (Fig. 3).

Due to the increasing area of necrosis, functional impotence of the index and adduction of the small finger and bone necrosis, an amputation of the second finger was performed at the level of the metacarpal phalange joint and a free skin graft was placed. This was on May 7, 1996 six months after the accident (Figs. 4, 5, 6).



FIG. 3. New manifestation of radiodermitis (four months after the accident)



FIG. 4. Five months after the acciernt, increased necrosis and retraction of the index finger, bone necrosis. An amputation is decided.



FIG. 5. Metacarpal phalange amputation of the index.



FIG. 6. Surgical sample of the amputated finger. Only the proximal phalange preserves its architecture. Necrosis of medial and distal phalange.

The graft was lost in March 1997 with evidence of necrosis at the level of fingers 1 and 3, no apparent change occurred up to November 1998 except for greater atrophy of fingers 1 and 3 (Figs.7, 8). In July 1999 radiographic signs of further necrosis and osteoporosis and total immobilization of the third finger made the hand non-functional. A further interphalange (proximal and medial) amputation was performed and no bleeding was observed during the procedure.



FIG. 7. New period of chronic radiodermitis in the first and third finger (19 months after the accident).



FIG. 8. Greater atrophy of first and third fingers, with areas of ulceration and necrosis of the progression and retraction of the third finger (31 months after the accident).

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Retraction of the proximal phalange of the third finger was seen progressively from January 2001 till January 2002 .as well as rigidity of the first finger (Fig. 9).

In May 2006 an amputation of the proximal phalange of the third finger was performed with torpid healing that lasted two months. In June 2007 a microsurgical procedure was done to open the thumb with external fixation so as to obtain a forceps movement of it. Physical therapy was performed with relatively good success as 30 degrees movement was obtained (Fig. 10).





proximal phalange of the third finger.

FIG. 9. Progression of the retraction of the FIG. 10. Results after surgery and rehabilitation to achieve pick up things.

In June 2009 the patient developed cellulites in the small finger that were treated with antibiotics and required three months to cure. In September 2010 diabetes mellitus, type II, was diagnosed and the fingers of the right hand showed diffuse osteopenia. Bone marrow studies show until today mild hypoplasia at the external bone.

In a study of human karyotype performed in March 2011 an exchange of sister cromatids in the normal range was found but there was no evidence of a neoplasia in the clinical and radiological studies performed. The last follow up on April 11, 2012 showed areas of radiodermitis in the right thumb which are compatible with the cyclic history of the radiation cutaneous syndrome.

#### 4. CONCLUSIONS

There are very few reports of radiological accidents followed clinically for a long term, which allows us to assess the deterministic and stochastic effects. The ignorance of the risks of radiation exposure is the triggering factor of this radiological accident. The cyclic history of the expected delayed effects of ionizing radiation on tissues allow us to observe the alternation of critical periods and clinically silent periods. We did not observe in the patient a development of malignant tumors or alterations in the study of sister chromatids.

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