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nature, intensity, and the character of the Pain (e.g., burning, twinging, twitching, and oppressive) may vary with the change in the location.

is pain generates spinal re exes, which in turn increases the fasciated and smooth muscle tone. Strong pain increases sympathetic tone, resulting in tachycardia and arterial hypertension. Other pain-related vegetative symptoms such as nausea and vomiting can occur, along with depression, anxiety, insomnia, irritability, and other mood and behavioral e ects [7]. ese biological mechanisms show no de ned correlation between stimulus and response. Tissue that is traumatized from in ammation or surgical interventions liberates mediators of in ammation, such as bradykinins, prostaglandins (PGs), and cytokines [8]. ese substances decrease the speci c threshold of the nociceptor neuron [9]. Consequently, the ow of a erent impulses to the spinal cord is intensi ed, thus resulting in primary hyperalgesia. For example, painful stimuli in the area of the nociceptor will be sensed as more intense than it would normally be [10]. Anti-in ammatory agents such as steroids and nonsteroidal anti-in ammatory drugs (NSAIDs) exert their analgesic e ects at this peripheral site. In addition to this "protective function," a permanent peripheral ow causes changes in the central nervous system (CNS). ere is an evidence of di erent mechanisms, including increased expression of excitatory N-methyl-D-aspartate-receptors, immediate early gene expression, and increased Ca2+ release, that contribute to this change. is is summarized as the sensitization of the spinal cord [9]. is sensitization causes a lower threshold for switching the peripheral stimulus to the second neuron in the dorsal horn. Electroencephalographic methods show ampli ed impulses, even a er removing the noxious stimulus. is extension of pain with time is referred to as long-term potentiation [11]. Moreover, the receptive area of the spinal neuron becomes enlarged, such that pain is perceived even in untraumatized, lesion-associated regions.

is extension of pain with the site is termed as secondary hyperalgesia. Long-term potentiation and hyperalgesia indicate the plasticity of the CNS during both acute and chronic pain states. As a result of the peripheral and central plasticity, pain gets intensi ed and turns more di cult to treat. ese changes contribute to the poor success rate of the treatment [12], albeit that psychological factors are also important in the transition from acute to chronic pain [13]. erefore, e ective measures to relive pain during at this acute state may prevent chronic pain development.

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occurs less frequently with transdermal opioid application than with oral administration. e dysmotility of the gastrointestinal tract caused by opioids can be treated with an opioid antagonist such as Naloxone.

ese drugs must be administered orally if this combination is not e ective. In the case of an inadequate action–side e ect relationship, a unique opioid rotation can be considered. Fast-setting free galenicals are used for the treatment of severe pain and single dose of opioids amount to approximately one-eighth to one-sixth of the daily dose. Reducing the opioids dosage is one of the most frequent mistakes in tumor pain therapy. In cases of acute, severe pain, fast-acting preparations such as oral morphine tablets or drops, fentanyl pills, or nose spray can be used. ese compounds are absorbed orally or through the mucus or skin, and characterized by their fast onset and short duration of action. Oral slow-release opioids are increasingly used in systemic pain management despite little evidence of their e cacy.

Steroids

e use of steroids is based on their antiphlogistic and anti-edematous e ects. Steroids are widely used in the case of elevated intracranial pressure, nerve plexus in ltration, spinal cord compression, and liver capsule tension. Additional desired e ects with the steroids include appetite stimulation, central antiemetic e ects, prevention of drug-induced nausea, and mood elucidation. High dose of corticosteroids are prescribed only for short term uses due to its adverse e ects in the long run. In a palliative situation, these issues are considered to be relative.

Other therapies

Prior to invasive procedures, therapy with an NMDA-receptor antagonist such as ketamine should be considered, as it has proven e cacy against opioid tolerance. NMDA-antagonist mechanisms are also believed to be involved in the action of the opioid L-methadone. Although individual reaction to it varies, a steady state can be reached only a er 4–7 days. Bisphosphonates delay osteoclast activity and are indicated in cases of osteolytic metastases. Hence, the use of nonretarded preparations should be avoided. Neuropathic pain therapy is based primarily on the use of antiepileptic drugs and certain antidepressants, with opioids administered as second-line therapy. Another possible treatment involves the topical application of lidocaine or capsaicin

cancer, the extension of the renal capsule would cause visceral pain that is accompanied by the enlargement of the tumor itself and urinary tract obstruction. Neuropathic pain occurs if the kidney cancer invades the retroperitoneal nerves. Somatic pain control is challenging as it easily

analyzed the urological cancer cases in patients who were referred to the palliative care department for the management of pain and evaluated the measures taken, with a focus on clinical practice. A er a detailed medical examination by interviewing and obtaining physical ndings for each patient, the palliative care team initiated the pain management therapy. A er the medical examination, we examined the patients' computed tomographic and magnetic resonance imaging ndings. A dermatome or osteotome was used to obtain samples for pathophysiological diagnosis. We administered treatment on the basis of the patient's pharmacological regimen and supportive care knowledge. Physiotherapy was an integral part of treatment and used the NRS for pain assessment. A decrease of ≥ 2 points was considered representative of signi cant amelioration.

Demographic data (e.g., age and sex), primary cancer lesion, stage (determined by the American Joint Committee on Cancer/ International Union against Cancer TNM classi cation and stage grouping), and metastasis etc were analyzed using the computerized database and the medical records.

We have evaluated the prior use of analgesics (opioid analgesics, non-opioid analgesics, and analgesic adjuvants) at the time of referral to the palliative care department, diagnosis of pain by the palliative care department, and initial interventions for cancer pain, including pharmacotherapies and non-pharmacological therapies.

e study was limited to 48 treated cases referred to the palliative care department by the urological division of our hospital (male/ female: 33/15). e median age of the patients was 60.31 ± 12.78 years (range: 30-82 years). e most common urological cancer was bladder cancer, which was found in 16 patients (33.3%), followed by renal cell carcinoma in 10 patients (20.8%), TCC of the renal pelvis in 7 patients (14.6%), and prostate cancer in 6 patients (12.5%). e recurrence rate was 45.8%, and 43.1% of the patients had clinical stage IV cancer. Forty-three patients had metastasis (Table 1). e most common site of metastasis was the bone, which was found in 29 patients (60.4%), followed by lung metastasis in 19 patients (39.5%), brain metastasis in 15 patients (31.2%), dissemination in 11 patients (22.9%), and lymph node metastasis in 11 patients (22.9%; Table 2). Forty- ve of 48 patients (93.7%) received preemptive analgesia at the urology department before intervention by the palliative care department. With regard to route of administration, 39 (86.7%) of 45 patients received analgesics orally, and the remaining 6 patients (13.3%) received analgesics parenterally. Analgesics used prior to referral to the palliative care department included opioids for moderate to severe pain (morphine, fentanyl, and oxycodone; 38/48: 79.1%), non-opioid analgesics (NSAIDs and acetaminophen; 43/48: 89.5%), and analgesic adjuvants (15/48: 31.2%). Among the 38 patients who received preemptive opioids, 29 (76.3%), 6 (15.7%), and 4 patients (10.5%) were treated with oxycodone, fentanyl, and morphine preparations, respectively. NSAIDs were used in 38 (88.3%) of the 43 patients who had received preemptive non-opioid analgesia, and 50% of the prescribed NSAIDs were not selective COX-2 inhibitors. Among the 15 patients who received preemptive adjuvant analgesic therapy, anticonvulsants were most commonly used in 10 patients (66.6%; Table 3). In the diagnosis of urological cancer-related pain in the patients who were referred to the palliative care department, somatic pain was the most common and found in 38 patients (79.0%), followed by neurogenic pain in 31 patients (64.5%) and visceral pain in 13 patients (27.0%; Table 4). Multiple causes of pain were diagnosed in 22 of the 48 patients. Initial interventions for cancer pain included increased quantity of oxycodone (22.9%), introduction of oxycodone (12.5%), introduction of anticonvulsants (10.4%), and change in the type of NSAID (10.4%; Table 5). Most of the changes in the type of NSAID used consisted of replacing a selective COX-2 inhibitor with another NSAID. Other interventions included physical supportive therapies such as the use of an orthopedic corset (10.4%), consultation with a psychiatrist (8.3%), administration of epidural blocks and other nerve blocks (6.3%), radiotherapy, acupuncture, and moxibustion (2.1% each). At the time of intervention in the palliative care department, disturbance of consciousness/delirium was observed in 50% of the patients who were receiving morphine. In addition, disturban morphine. In addition,