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DRUG-INDUCED TOXICITY: MALIGNANT AND BENIGN CASES OF HYPERTHERMIA

Review article

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Abstract

Authors investigated the reports of fever of unknown origin (FUO) and possible causes and risk factors for its development. Underestimation of importance of fever pathogenesis causes irrational changes in the treatment regimen and antipyretic prophylaxis may be preferred. The therapeutic goal is the relevant evaluation of risk and benefits of use of fever inducing drugs.

Aim: this review aims to elucidate the problem of drug-induced hyperthermia as a diagnosis of exclusion.

Diagnosing drug fever is challenging: fever itself is insufficient for a definitive diagnosis. Clinicians must first rule out other serious conditions and gather a detailed medical history. When drug fever is suspected, adding antipyretics or antibiotics is not recommended. The key intervention is stopping the suspected medication. Yet, intentionally stopping and then restarting a drug to confirm the diagnosis is dangerous and should be avoided. If stopping the drug does not improve the patient's status, reconsider the approach. Particularly in cases where no alternative therapy exists — such as with certain antitumor or nosocomial antibiotics — abrupt discontinuation may cause more harm than the fever itself.

Keywords: drug fever, drug induced hyperthermia, fever of unknown origin, antipyretic, antipyretic prevention.

ЛЕКАРСТВЕННАЯ ТОКСИЧНОСТЬ: ЗЛОКАЧЕСТВЕННЫЕ И ДОБРОКАЧЕСТВЕННЫЕ ФОРМЫ ГИПЕРТЕРМИИ

Обзор

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Аннотация

Авторы изучили сообщения о лихорадке неизвестного происхождения (ФНО), а также возможные причины и факторы риска ее развития. Недооценка важности патогенеза лихорадки приводит к нерациональным изменениям в схеме лечения, и предпочтение может быть отдано жаропонижающей профилактике. Терапевтическая цель — адекватная оценка риска и пользы применения жаропонижающих препаратов.

Цель: прояснить проблему медикаментозной гипертермии как критерия исключения.

Диагностика лекарственной лихорадки является сложной задачей: само по себе наличие лихорадки недостаточно для постановки окончательного диагноза. Врачи должны сначала исключить другие серьезные заболевания и собрать подробную историю болезни. При подозрении на лекарственную лихорадку не рекомендуется назначать жаропонижающие или антибиотики. Ключевым вмешательством является прекращение приема предполагаемых лекарств. Тем не менее, намеренное прекращение приема препарата, а затем его повторный прием для подтверждения диагноза опасно, и этого следует избегать. Если прекращение приема препарата не улучшает состояние пациента, то необходимо пересмотреть подход. Особенно в тех случаях, когда альтернативной терапии не существует — например, при применении определенных противоопухолевых или внутрибольничных антибиотиков, — резкое прекращение лечения может причинить больше вреда, чем сама лихорадка.

Ключевые слова: лекарственная лихорадка, медикаментозная гипертермия, лихорадка неизвестного происхождения, жаропонижающее средство, жаропонижающая профилактика.

Введение

Fever is a nonspecific protective and adaptive response of the body occurring in response to pathogenic stimuli. Regarding etiology fever is divided into infectious and inflammatory (viruses, bacteria, intracellular parasites) or non-infectious (autoimmune processes, allergic diseases, tumors, metastases, pharmacotherapy, endocrinopathies). Body temperature is regulated by the interaction of the nervous, endocrine, and immune systems. The most common trigger of hyperthermia are exogenous pyrogens (bacterial and viral particles, exotoxins). These pyrogen affects the central nervous system and through the heat production increase the existing temperature considered as low. Particularly fever is a protective measure of the body but undesirable hyperthermia can be life-threatening (malignant). Fever of unknown origin (FUO) is most often caused by infectious diseases, although in some cases, it may be due to an atypical form of the disease. Over 30% cases of FUO are caused by tumor, paraneoplastic syndrome and metastases. Systemic lupus erythematosus, rheumatic fever, allergic vasculitis,

rheumatoid arthritis, and other inflammatory processes can also cause FUO. Hyperthyroidism, pheochromocytoma and other endocrine diseases, hereditary conditions, and stress can also cause persistent fever. Revealing drug-induced fever is challenging in many cases, as the etiology can be quite variable and complicated.

Thermoregulation disorders

Inner pyrogens are produced by stimulated monocytes and macrophages. Immune chemokines such as cytokines play a major role in fever development (IL1, IL6, TNF- alpha, interferons). Cytokines and interferons act on thermosensitive neurons in the preoptic area of the hypothalamus by increasing the synthesis of prostaglandins E2 and F2a from arachidonic acid.

Fever develops through the following phases or periods: prodromal period, period of rising temperature, a period of relative stability, a period of decreasing body temperature. Every period is accompanied by certain autonomic nervous system reactions.

Fever of unknown origin was operationally defined in 1961 through the Petersdorf-Beeson criteria:

1) repeated fever episodes exceeding 38.3 °C;

2) disease duration surpassing 3 weeks;

3) diagnostic uncertainty persisting after 1 week of inpatient assessment.

Empiric antibiotics are ineffective for fever of unknown origin and should be avoided except in neutropenic, immunocompromised, or critically ill patients [1] (except for malignant hyperthermia). It is a febrile reaction that coincides with the administration of medications in the absence of other conditions for its occurrence. Low-grade fevers not exceeding 38.3 °C should be excluded from the diagnosis of drug-induced fever. Fever resolution upon drug discontinuation confirms the iatrogenic origin. The patient must remain afebrile for at least 72 hours after normalization of body temperature. Fever can appear either suddenly (antitubercular chemotherapy) or delayed (vancomycin or minocycline).

Excessive functional strain on organs and systems participating in fever pathogenesis may result in pathological conditions or even fatal outcomes. A sharp rise in temperature followed by a critical decline may trigger circulatory collapse, syncope, heart failure, seizures, arrhythmias, and impaired consciousness.

Etiology of drug-induced hyperthermia involves a combination of host susceptibility factors (including augmented reactivity, sensitization history, and genetic variations) [2] and medication attributes (encompassing structural composition, immunogenic potential, pharmaceutical form, and administration route) [3].

Patient risk factors

Drug fever arising from hypersensitivity occurs when the drug molecule either acts as a complete antigen or binds to haptens (such as serum proteins) or acetylates the patient’s blood proteins, conferring antigenic properties on them. This hypersensitivity can present as fever alone or alongside other hypersensitivity signs, collectively termed systemic hypersensitivity syndrome. The syndrome encompasses leukemoid reaction, eosinophilia, lymphocytosis, erythema multiforme, and cerebral edema—a life-threatening condition associated with high mortality.

The pathogenesis of hyperpyrexia may be influenced by several host and environmental factors. Notably immature thermoregulatory capacity in newborns and female sex have been associated with increased susceptibility. Furthermore, dietary patterns constitute a modifiable risk factor: overconsumption of histamine-releasing foods, foods naturally rich in histamine, and protein-dense diets have all been linked to elevated risk of hyperpyrexia [4].

Risk factors of medications

4.1. Pharmacogenetics

A rare complication with a poor prognosis is malignant hyperthermia. This condition is characterized by high temperature (40°C or higher), muscle rigidity and autonomic changes. This pathological reaction is associated with defects in the ryanodine calcium channel receptor. Malignant hyperthermia represents a rare pharmacogenetic condition that is typically induced by volatile anesthetics and succinylcholine. It is most commonly associated with pathogenic mutations in the RYR1, CACNA1S, and STAC3 genes [5], [6].

In patients predisposed to malignant hyperthermia, drugs can cause a sharp, uncontrolled increase in oxidative metabolism, sustained skeletal muscle contraction, acidosis and fever. Combination of the ryanodine receptor disturbances and dopamine stimulation also contributes to the development of fever. These disorders are not classified as FUO but are nevertheless not all underlaid mechanisms are clear understood. Decreased dopamine level in the central nervous system disrupts the serotonergic structures in the striatum and hypothalamus responsible for thermoregulation. Exposure to volatile anesthetics (such as halothane, isoflurane, sevoflurane, desflurane, and enflurane), either alone or combined with the depolarizing muscle relaxant succinylcholine, can trigger malignant hyperthermia (MH). These agents induce excessive calcium release from the sarcoplasmic reticulum and facilitate extracellular calcium influx into muscle cells. This results in muscle contracture, glycogen breakdown, and heightened cellular metabolism—generating heat and excess lactate. Clinical consequences include acidosis, hypercapnia, tachycardia, hyperthermia, muscle rigidity, compartment syndrome, rhabdomyolysis (with elevated serum creatine kinase), hyperkalemia (risking cardiac arrhythmias or arrest), and myoglobinuria (with potential kidney failure) [7]. Although Parkinsonism looks similar in symptoms (high body temperature and muscle stiffness), it has a different cause. It can occur as hyperpyrexia when taking or stopping antiparkinsonian drugs and methyldopa.

4.2. Psychostimulants

Peripheral dopamine blockade in muscles causes increased skeletal muscle contraction due to a genetic predisposition caused by a defect in the ryanodine receptor. In addition to antipsychotics, this reaction is also caused by metoclopramide, desipramine, lithium salts, reserpine, amoxapine, fluoxetine and psychostimulants such as cocaine and amphetamine. The pathophysiology of hyperthermia secondary to psychotropic substance use involves multifaceted neurotransmitter dysregulation, primarily affecting serotonin, noradrenaline, and dopamine systems. These mechanisms fall into three interrelated pathways:

1. Augmented Thermogenesis. Psychostimulant agents provoke hyperkinetic states, elevating metabolic heat production. Noradrenaline-mediated beta-3 adrenergic receptor activation induces mitochondrial uncoupling, culminating in non-shivering thermogenesis.

2. Impaired Thermolytic Mechanisms. Noradrenergic stimulation of peripheral vascular alpha adrenergic receptors leads to vasoconstriction, diminishing cutaneous heat dissipation. Inadequate sweating further compromises thermoregulation.

3. Central Thermoregulatory Dysfunction. Hypothalamic serotonin pathways are pivotal in temperature homeostasis. Psychotropic substance–induced serotonin surges result in receptor hyperactivation, disrupting hypothalamic thermoregulatory set points.

4.3. Route of administration

The parenteral route of drug delivery constitutes a risk factor because it may lead to phlebitis — particularly when irritating agents [8] or hyperosmolar solutions are administered [9]. Fever can happen due to the release of destroyed microorganisms, such as spirochetes, or tumor cells during the use of antibiotics or cytostatics, respectively. Pyrogens are released from the cells, leading to hyperpyrexia. In addition to their direct effects on thermoregulatory centers, medications can also increase temperature through indirect effects on the autonomic nervous system, reducing heat loss and increasing heat production (atropine-like effects, sympathomimetics). Atropine-like agents cause dry skin and decreased sweating.

Drugs as pyrogens

5.1. Antipsychotics and anticonvulsants

Fever is a frequently reported adverse event in the early stages of clozapine therapy. The lack of standardized criteria to determine whether fever represents a drug-induced effect complicates clinical decision-making. Although the underlying mechanisms are not fully understood, data indicate that clozapine-associated fever may result from a systemic inflammatory reaction linked to the drug's effects on immune regulation. Fever is a frequently reported adverse event in the early stages of clozapine therapy. The lack of standardized criteria to determine whether fever represents a drug-induced effect complicates clinical decision-making. Although the underlying mechanisms are not fully understood, data indicate that clozapine-associated fever may result from a systemic inflammatory reaction linked to the drug's effects on immune regulation [10], [11], [12].

5.1.1. Postoperative fever

Halogenated inhalation anesthetics, lidocaine, dexmedotimidine, suxamethonium, Propofol, opiods, and are known the medications most commonly associated with drug-induced fever in post operative fever. The onset of fever varied, occurring either immediately after surgery or several days later, and could be intermittent, remittent, or continuous. Many clinical cases illustrate the complex interaction among drug-induced fever and nervous system functioning. Thermoregulatory dysfunction secondary to spinal cord injury may explain compromised thermoregulatory mechanisms [14], [15], [16], and [17], [18], [19].

Proton pump inhibitors are known as pyrogen for development of FUO [20], [21], [22], [23], [24].

5.2. Pain-killers and anti-inflammatory drugs

Morphine is a commonly prescribed opioid for managing moderate-to-severe cancer-related pain. Nevertheless, it is associated with a range of adverse effects, among which morphine-induced fever stands out as an exceptionally rare and inadequately characterized clinical manifestation [25].

Drug fever often goes misdiagnosed, particularly in cases where concurrent infection is present. Painkillers and anti-inflammatory drugs are frequently used in conditions accompanied with increased body temperature although they are capable to induce fever in some predisposed patients. Many other—the counterdrugs as NSAIDs—may cause fever (ibuprofen) in spite of being antipyretics [26], [27], [28], [29]. NSAIDs are also prone to cause local toxicity due to high osmotic activity [30], [31], [32].

Antiinfectional and other chemotherapy drugs are common reasons of neutropenic and allergic fever [33], [34], [35] and [36], [37].

5.3. Calcium-phosphorus agents

Ion imbalance also invests into adverse drug reaction as fever. Bisphosphonates (zolendronate) are widely applied in osteoporosis to affect bone resorption. Some studies show that higher levels of vitamin D (specifically 25(OH)D > 35 ng/mL) are tied to a lower risk of fever after bisphosphonate treatment. This suggests that boosting vitamin D beforehand — especially before the first zolendronate dose — might help prevent fever. If future studies confirm this link, doctors may routinely recommend a short vitamin D supplement course prior to zolendronate infusion. Certain blood markers were linked to a higher chance of fever: neutrophils, monocytes and ferritin. At the same time, other markers — hemoglobin, lymphocytes, calcium, and high density lipoproteins — were connected to a lower risk of fever, and these relationships reached statistical significance [38], [39], [40].

5.4. Misoprostol

The widespread use of misoprostol in medication abortion and postpartum hemorrhage management is accompanied by a recognized adverse effect: drug-induced fever. This phenomenon often poses diagnostic and therapeutic challenges, increasing the likelihood of unnecessary antibiotic administration and thereby contributing to broader issues of antimicrobial stewardship. Higher misoprostol doses and advanced gestational age were significantly correlated with increased fever incidence [41], [42].

Therapeutic Approach: fever management

The therapeutic approach to excessive motor activity mandates initiation with high-dose benzodiazepines administered orally or intravenously in cooperative patients for suppression of motor activity; in the event of a critical clinical presentation, prompt endotracheal intubation shall be undertaken to permit deep sedation with high-dose benzodiazepines and concomitant neuromuscular blockade for the purpose of reducing muscular heat generation; in instances of refractory agitation persisting

during preparation for intubation and benzodiazepine loading, droperidol and/or ketamine shall be utilized as rescue pharmacotherapy to achieve adequate control of agitation [43].

Pregnant patients at risk for malignant hyperthermia require careful anesthesia planning for non-emergent procedures. Triggering agents must be excluded, and safe alternatives — such as local anesthesia, nerve blocks, epidural or spinal techniques— should be selected.

During labor, continuous epidural analgesia offers optimal management. If cesarean delivery becomes necessary and no epidural is in place, neuraxial methods (spinal, epidural, or combined spinal-epidural) are the preferred choice, provided contraindications are absent.

Notably, in pediatric cardiac surgery involving cardiopulmonary bypass, a single pre-operative intravenous paracetamol dose effectively lowers mean body temperature over the initial 24-hour postoperative period [44].

Discontinuing the suspected drug is the indispensable first step in managing DiHS/DRESS; while systemic corticosteroids retain their position as first-line therapy, the therapeutic landscape is evolving — robust and growing evidence now supports the incorporation of steroid-sparing strategies, such as cyclosporine, immunoglobulins, interleukin-5 axis inhibitors, and Janus kinase inhibitors, broadening the range of effective treatment options available to clinicians [45].

Although dantrolene (an RYR1 antagonist) remains the cornerstone of pharmacotherapy for malignant hyperthermia, its adverse effects underscore the need to investigate alternative treatment strategies. Recent research highlights the involvement of the endocannabinoid system in regulating muscle calcium homeostasis, pointing to its potential therapeutic relevance in malignant hyperthermia.

The endocannabinoid system includes endogenous ligands (such as anandamide), cannabinoid receptors (including CB1), and can influence calcium dynamics. Activation of CB1 receptors suppresses PKA-mediated phosphorylation of RYR1 and L-type calcium channels, thereby decreasing myoplasmic calcium levels and muscle contractility—a mechanism that may counteract the pathophysiological processes underlying fever.

Moreover, blocking or desensitizing the transient receptor potential vanilloid 1 (TRPV1) calcium channel reduces calcium efflux from the sarcoplasmic reticulum. Preclinical data indicate that CB1 receptor agonism can lower body temperature and mitigate cardiovascular stress, which aligns with the therapeutic objectives in MH management [46], [47], [48].

Conclusion

Diagnosing drug fever is challenging: fever itself is insufficient for a definitive diagnosis. Clinicians must first rule out other serious conditions and gather a detailed medical history. When drug fever is suspected, adding antipyretics or antibiotics is not recommended. The key intervention is stopping the suspected medication. Yet, intentionally stopping and then restarting a drug to confirm the diagnosis is dangerous and should be avoided. If stopping the drug does not improve the patient's status, reconsider the approach. Particularly in cases where no alternative therapy exists—such as with certain antitumor or nosocomial antibiotics—abrupt discontinuation may cause more harm than the fever itself.

Конфликт интересов

Не указан.

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Conflict of Interest

None declared.

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