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Effect of antithrombotic drugs on bone health

Background

In modern medicine thrombosis prophylaxis of various types are frequently prescribed to the old population. With increased risk of thromboembolic episodes in the old population, the geriatric population is now commonly subjected to antithrombotic drugs (anticoagulants and antiplatelet drugs). Since every prescribed medication is associated with some beneficial and some non-beneficial effects, this review focuses on the effect of antithrombotic drugs on bone health in geriatric populations.

Osteoporosis

Osteoporosis is defined as a skeletal disorder characterized by compromised bone strength leading to an increased risk of fractures. Physiological loss of bone mass with aging is approximately 0.7% per year in adults; however, it is more intense in postmenopausal women than in men of the same age. The process of bone remodeling from resorption to matrix synthesis to mineralization normally takes 8 months and is a slow but constant process. Structure and functionality of bone in old people are different from young adults as there is decreased activity of osteoblasts and decreased production of growth factors and compromised bone matrix.

Approximately 95% of osteoporosis forms are primary (type I or postmenopausal osteoporosis, and type II or senile osteoporosis) in which either an estrogen deficiency (in women, type I) is the cause or in the case of senile osteoporosis (beginning of the 7th decade of life, in both women and men) a reduced alimentary intake of vitamin D and calcium is the main cause. Only 5% are secondary in nature, e.g. endocrinopathy, metastases of solid malignancies or multiple myeloma, malassimilation syndromes, drug effects (e.g., antiepileptic drugs, heparin) and renal osteopathy.

Diagnosis of osteoporosis

According to the Governing Association on Osteology (Dachverband Osteologie, DVO) guidelines from 2017 [29], the basic diagnosis of osteoporosis includes the following measures:

a) Anamnesis and patient examination: patient history positive for low-grade trauma intensity vertebral fractures, neurological deficiency and known functional disability are major risk factors for osteoporosis. Patients who suffer from certain endocrinological diseases (e.g. Cushing's syndrome, primary hypothyroidism and diabetes), rheumatic (rheumatoid arthritis), gastroenterological (e.g. celiac disease), neurological diseases (e.g. apoplexy), patients after some operative procedures, such as gastrectomy or patients consuming certain long-term medications, such as hormone therapy, steroids and antiepileptic drugs are also at risk of developing osteoporosis.
b) Osteodensitometry: according to the World Health Organization (WHO) criteria, osteoporosis is defined as a bone mineral density (BMD) that is 2.5 standard deviations (SD) or more below the average value for young healthy women (a T-score of < −2.5 SD). The measurement of T-score is done with dual-energy X-ray absorptiometry scan (DEXA scan) [3].
c) Radiology images: a decreased bone density can sometimes also be diagnosed in plain radiographs or magnetic resonance imaging (MRI). In some cases a decreased cortical thickness and loss of bony trabeculae can be seen in the early stages in radiography.
d) Laboratory diagnostics: as no single modality can prove that a patient suffers from osteoporosis, the following battery of tests are suggested by the DVO as initial laboratory assessment parameters to diagnose the cause of osteoporosis (primary or secondary osteoporosis):
Serum calcium, sodium and phosphate, glomerular filtration rate (GFR), alkaline phosphatase, gamma glutamyl transferase, blood count, erythrocyte sedimentation rate (ESR), C-reactive protein, serum protein electrophoresis, thyroid stimulating hormone (TSH), 25-hydroxy vitamin D3, testosterone (in males), bone resorption markers, calcium clearance in 24 h urine und cadmium levels in urine.

Histopathological assessment of bone: this can be performed by examining the sample obtained during bone biopsy under an electron microscope (generalized thinning of trabeculae and irregular perforation of the trabeculae, reflecting unbalanced osteoclast-mediated bone resorption) [28] and histomorphometric analysis of the bone biopsy (e.g. tetracycline double labeling).

Antithrombotic drugs and bone health

Unfractionated heparin (UFH)

The first anticoagulant to be widely used was UFH in intravenous (i.v.) administration. Despite the rapid onset of action and inexpensiveness, UFH requires close inpatient laboratory monitoring and can cause severe uncontrolled bleeding. Other potential adverse effects include elevated liver enzymes and heparin-induced thrombocytopenia.

According to the various studies, long-term and high-dose treatment with heparin was responsible for loss of bone strength [8, 10]. Chronic heparin treatment reduces BMD [25]. It has been estimated that bone loss occurs after 6 months of heparin treatment with daily doses >15,000 units. The exact mechanism by which UFH induces bone loss is not completely understood but there is a postulated hypothesis that heparin causes increased bone resorption by stimulating osteoclasts and suppressing osteoblast function, leading to decreased bone mass. Other proposed mechanisms include depletion of mast cells in bone marrow and enhancement of parathyroid hormone (PTH) function [6]. As heparin was associated with osteopenia and other side effects, combined with its intravenous application, it was considered to be an antithrombotic drug with many disadvantages.

Vitamin K antagonists (warfarin/phenprocoumon)

Heparin was replaced by oral vitamin K antagonists (VKA). These drugs are highly effective and are still used as oral antithrombotic drugs. Despite their efficient nature they have been proven difficult to manage, they require frequent monitoring and dose adjustment to limit adverse consequences, increased risk of bleeding and multiple drug interactions. This contributes to the underuse of warfarin and low patient satisfaction and compliance. In addition, VKAs have a slow onset of action. When used for venous thromboembolism (VTE) treatment, bridging therapy with injected anticoagulants with a fast onset of action is required.

Studies have shown that warfarin and phenprocoumon were associated with bone loss. In a comparison between long-term warfarin users (≥1 year) and non-users, the risk of fractures was significantly higher in users compared to non-users (odds ratio, OR 1.25; 95% confidence interval, CI 1.06–1.48). The association between osteoporotic fractures and long-term warfarin use was significant in men (OR 1.63, 95% CI 1.26–2.10) but insignificant in women (OR 1.05, 95% CI 0.88–1.26) [1, 7, 22–24]. The possible mechanism of bone loss is related to the predominant vitamin K-dependent protein in bone, i.e. osteocalcin, accounting for up to 15% of noncollagenous bone. Vitamin K carboxylation makes osteocalcin more adherent to calcium and hydroxyapatite. When osteocalcin is gamma carboxylated the BMD is decreased and the fracture rate is increased. Furthermore, vitamin K supplementation reduces bone turnover and improves bone strength [18].

Low molecular weight heparins (LMWH) and indirect factor Xa inhibitor

The LMWHs were developed to overcome the drawbacks of UFH. Because of their predicable activity, they do not require monitoring and have a lower risk of heparin-induced thrombocytopenia (HIT). As these drugs are self-administered subcutaneously and do not require regular blood monitoring they can be given to outpatients. Some of the LMWHs tend to accumulate in patients with kidney impairment, hence they should be avoided or the dosage should be adjusted. In a review of the electronic databases, administration of LMWHs for 3–6 months did not increase the relative risk (RR) of all fractures in comparison to UFH, VKA or placebo (pooled RR: 0.58, 95% CI: 0.23–1.43, $I^2 = 12.5\%$), the administration of LMWHs among cancer patients at 6–12 months was also insignificant (RR: 1.08, 95% CI: 0.31–3.75, $I^2 = 4.4\%$), LMWH for 3–24 months decreased mean BMD by 2.8–4.8% (depending on the BMD site) compared to mean BMD decreases of 1.2–2.5% with oral VKA [8]. In an in vitro study the indirect factor Xa inhibitor fondaparinux showed no effect on osteoblast differentiation or function and is predicted to be bone neutral [12].

New oral anticoagulants (apixaban, enoxaban, dabigatran, rivaroxaban)

New oral anticoagulants (NOAC) are novel direct-acting medications, selective for a specific coagulation factor, either thrombin (IIa) or activated factor X (Xa). The advantages of NOAC include less safety issues (e.g., a lower incidence of major bleeding), convenience of use, minor drug and food interactions, a wide therapeutic window, and no laboratory monitoring [13]. Except for dabigatran, for which the antidote idarucizumab is readily available, all other NOAC do not yet have an approved antidote.

There are relatively few studies about bone health and NOACs in existence. An in vitro study of rivaroxaban showed that it causes reduction in osteoblast function [26]. Hence, it results in the formation of bone with decreased strength, On the other hand the use of rivaroxaban, showed positive results when compared to warfarin. Switching to rivaroxaban from warfarin in patients with atrial fibrillation was associated with an increased in bone formation markers and a decrease of bone resorption markers. The reduction in the function of osteoblasts was associated with a reduction in the mRNA expression of the bone marker osteocalcin, the transcription factor Runx2 and the osteogenic factor BMP-2 [20].

The use of dabigatran in comparison to warfarin in patients with a history of falling showed a lower risk of osteoporotic fractures (dabigatran vs. warfarin: 1.6 vs. 3.6 per 100 person-years, Absolute risk difference (ARD) per 100 person-years –3.15, 95% CI –2.40 to –3.45, Incidence rate ratio (IRR) 0.12, 95% CI 0.04–0.33); but some studies have shown that they fair far better in comparison to other anti-thrombotic drugs [17]. Rivaroxaban (RR: 0.78, 95% CI: 0.61–0.99, *P* value for interaction (*P*) = 0.04) and apixaban (RR:

0.70, 95% CI: 0.55–0.90, $P = 0.01$) showed a lower fracture risk when compared to warfarin and dabigatran (RR: 1.01, 95% CI: 0.69–1.48) and enoxaban (RR: 0.88. 95%CI:0.74–1.04) [9].

Further clinical assessments and studies to determine the mechanism of action of these drugs and their effects on bone metabolism should be pursued. The possible mechanisms of bone loss with NOACs can be: 1) decreased osteoblasts function and 2) decreased osteocalcin function.

Acetylsalicylic acid

Acetylsalicylic acid (ASA) is an irreversible cyclooxygenase inhibitor and an antiplatelet drug. Its most common use is for primary prevention of cardiovascular events in older patients without prior history of atherosclerotic cardiovascular disease. Side effects of chronic consumption of ASA are easy bruising and abnormal bleeding, gastritis, peptic ulcers and rarely upper gastrointestinal bleeding, and reduced kidney and liver function. In a recent analysis, ASA use was associated with a lower cardiovascular mortality (RR: 0.92; 95% CI: 0.83–1.01), myocardial infarct mortality (RR: 0.82; 95% CI: 0.71–0.94) and stroke mortality (RR: 0.94; 95% CI: 0.86–1.02), but also a higher risk of mortality due to a major bleeding (RR: 1.47; 95% CI: 1.31–1.65) and intracranial hemorrhage (RR: 1.33; 95% CI: 1.13–1.58) was seen [2].

The use of ASA has never been directly associated with osteoporosis but a few reports have confirmed that low dose ASA therapy is associated with low 25-hydroxy-vitamin D (25-OH-D) levels and delayed bone healing. The ASA inhibits the synthesis of prostaglandin E2 essential in bone remodeling. With respect to the fact that ASA can cause renal damage and hepatotoxicity, theoretically this constellation can indirectly also cause osteoporosis [4, 14, 16, 19].

Some studies suggested that ASA actually improves bone health. In vitro studies showed that ASA could enhance the survival of bone marrow mesenchymal stem cells, the progenitors of osteoblasts, and stimulate the differentiation of pre-osteoblasts. It also inhibited the nuclear factor kappa-B (NFκB) pathway and decreased the expression of receptor activator of NFκB ligand, thus suppressing the formation of osteoclasts [5]. When prescribed as more than one defined daily dose/day (1 DDD/day) ASA was associated with increased risk for any fractures (OR 1.17, 95% CI 1.02–1.34). Low-dose aspirin at 0.5 or less DDD/day (OR 1.10, 95% CI 1.01–1.20) and between 0.51 and 1 DDD/day (OR 1.17, 95% CI 1.08–1.27) [5].

Abstract · Zusammenfassung

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Effect of antithrombotic drugs on bone health

Abstract

With the increasing consumption of antithrombotic drugs among old people, expected as well as unexpected side effects on bone health are considerable, e.g. osteoporosis, fragility fractures, etc. This review focuses on antithrombotic drugs and their effects on bone health. The following groups were reviewed: parenteral long-term use of unfractionated heparin (UFH) is associated with osteopenia. The oral intake of vitamin K antagonists (VKA) makes them more convenient than UFH but chronic use also results in osteopenia. Limited reports of bone loss have been associated with low molecular weight heparins (LMWH) and indirect factor Xa inhibitors but in contrast to VKA and UFH they are less associated with osteopenia. There have been limited studies evaluating the effect of new oral anticoagulants (NOACs) on bones. Overall, they are considered safer than other drugs. There have been no reports about acetylsalicylic acid (ASA) and clopidogrel causing osteopenia but their metabolism by the kidneys and liver can cause reduced 25-hydroxy-vitamin D levels and can theoretically contribute to osteoporosis. Some reports suggested that high dosage clopidogrel can also negatively affect bones. After a detailed literature review long-term use of antithrombotic drugs can negatively affect the bones. Their role in bone health needs to be studied in detail and the clinical use in geriatric patients should be prudent.

Keywords

Osteoporosis · NOAC · Warfarin · Geriatrics · Trauma

Auswirkung von Antithrombotika auf den Knochen

Zusammenfassung

Mit dem zunehmenden Verbrauch antithrombotischer Arzneimittel bei geriatrischen Patienten sind deren Nebenwirkungen auf die Knochen beträchtlich, z. B. Osteoporose, Fragilitätsfrakturen etc. Diese Übersicht konzentriert sich auf Antithrombotika und deren Auswirkungen auf die Knochengesundheit. Folgende im folgenden beschriebenen Gruppen wurden berücksichtigt. Die parenterale Langzeitanwendung von unfraktioniertem Heparin (UFH) ist mit Osteopenie verbunden. Die orale Einnehme von Vitamin-K-Antagonisten (VKA) macht sie komfortabler als UFH, langfristig ist die Anwendung aber ebenfalls mit einer verstärkten Neigung zur Osteopenie verbunden. Niedermolekulare Heparine (LMWH) und indirekter Faktor-Xa-Inhibitor wurden nur in begrenztem Umfang mit Knochenschwund in Verbindung gebracht, insgesamt sind sie jedoch im Gegensatz zu VKA und UFH wohl geringer mit einer Osteopenie assoziiert. Zu den neuen oralen Antikoagulanzien (NOAK) wurden begrenzte Studien durchgeführt, um deren Wirkung auf die Knochen zu bewerten. Insgesamt gelten sie als sicherer als andere Medikamente. Bezüglich Acetylsalicylsäure (ASS) und Clopedogrel gibt es keine Berichte darüber, dass diese Osteopenie verursachen, jedoch kann ihr Metabolismus durch Nieren und Leber einen verringerten Spiegel an 25-Hydroxy-Vitamin D zur Folge haben und daher theoretisch eine Osteoporose begünstigen. Nach einer ausführlichen Literaturrecherche kann die Langzeitanwendung von Antithrombotika die Knochen negativ beeinflussen. Ihre Rolle für die Knochengesundheit muss eingehend untersucht werden und die klinische Anwendung bei geriatrischen Patienten sollte umsichtig erfolgen.

Schlüsselwörter

Osteoporose · NOAK · Phenprocoumon · Geriatrie · Trauma

Clopidogrel

Clopidogrel is an irreversible purinergic receptor P2Y12 inhibitor. Its use and side effects are similar to ASA. The P2Y12 receptor is expressed on platelets and on bone cells. Clopidogrel inhibits the function of the bone-forming osteoblasts. An in vivo study in mice treated with clopidogrel showed reduced bone mass and strength of approximately 20% [28]. It induced severe osteoporosis in the animals. In a cohort study, it showed biphasic pattern: at higher doses, e.g. 0.40–0.99DDD (hazard ratio, HR: 1.17, 95%CI: 1.11–1.24) or at more than or equal to 1DDD (HR: 1.30, 95%CI: 1.21–1.39) it was associated with increased fracture risk and at lower doses e.g. <0.10DDD (HR: 0.82, 95%CI: 0.78–0.86) or 0.10–0.39DDD (HR: 0.87, 95%CI: 0.83–0.92) it was associated with decreased fracture risk [21].

Anticoagulants and bone healing

Current knowledge on the effect of several drugs on bone healing is characterized by inconclusive and controversial results from several animal models, together with absence of univocal clinical data. Some antithrombotic dugs, e.g. warfarin, aspirin, LMWH and heparin are responsible for delayed bone healing [11, 15, 27] but the same cannot be said for others as there is a clear lack of clinical studies on this subject.

Discussion

In various reports and studies across the world, it has been pointed out that some antithrombotic drugs are capable of causing osteoporosis. Be it VKA or UFH, both have been proven to have an osteopenic effect on bone and ASA has already been proven to cause low levels of vitamin D in the human body. Combined with this fact and the ability to alternate kidney and liver functions, it can also cause osteoporosis. The NOACs are relatively new to the clinical practice; hence there has been some limited clinical evaluation of their effects on bone. For NOACs, clopidrogrel, LMWH some studies relating them to bone loss have been performed. Increasing polypharmacy has only contributed to the epidemic of fragility fractures. Antithrombotic drugs are prescribed more often now than ever before. After this review of the database it can be concluded that there is a lack of evidence on this issue and there is a need to further study the effects of these drugs on bone.

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Compliance with ethical guidelines

Conflict of interest G. Dadwal, T. Schulte-Huxel and G. Kolb declare that they have no competing interests.

For this article no studies with human participants or animals were performed by any of the authors. All studies performed were in accordance with the ethical standards indicated in each case.

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Buchbesprechung

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Medizinische Einsatzteams

Prävention und optimierte Versorgung innerklinischer Notfälle, Scoringsysteme, Fallbeispiele

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Die Letalität nach operativen Eingriffen ist trotz moderner chirurgischer Techniken und anästhesiologischer Verfahren höher als erwartet und beträgt nach jüngsten Europäischen Daten 4%. Daher ist es geboten, Konzepte zu entwickeln, um die perioperative Behandlungsqualität zu steigern. Ein Ansatz dabei ist die Einführung von Medizinischen Einsatzteams (MET), deren Aufgabe, im Gegensatz zu den klassischen Reanimationsteams, die Verhinderung von lebensbedrohlichen Ereignissen ist.

Bereits im Jahr 1997 wurden diese in den Niederlanden etabliert und es konnte eine signifikante Reduktion der Letalität, Aufnahmen auf Intensivstationen sowie Reanimationen nachgewiesen werden. Seither gibt es in zahlreichen Ländern Bestrebungen vergleichbare klinische Strukturen aufzubauen. Auch in Deutschland wurde eine Empfehlung zur Etablierung von MET zur Verbesserung der Behandlungsqualität von den anästhesiologischen und den chirurgischen Fachgesellschaften erarbeitet.

Die Herausgeber des vorliegenden Werkes legen nun das erste deutschsprachige Buch zu dieser Thematik vor, in dem in drei übergeordneten Sektionen alle wesentlichen Aspekte des Themenkomplexes in strukturierter und übersichtlicher Form dargestellt werden. Zunächst werden in anschaulicher Weise die Ziele und Möglichkeiten der MET präsentiert, im zweiten Kapitel die Fragen nach der Organisation und Schulung erörtert und Antworten gegeben, wie diese neue Konzeption umgesetzt werden kann. Letzteres ist insbesondere daher von großer Wichtigkeit, da sich in der Praxis häufig gezeigt hat, dass sinnvolle Projekte aufgrund einer mangelhaften Umsetzungsstrategie scheiterten. Im abschließenden Kapitel veranschaulichen Fallbeispiele aus unterschiedlichen Fachgebieten praxisnah, welchen Betrag MET zur optimierten Versorgung der Patienten leisten können.

Das Werk „Medizinische Einsatzteams" ist informationsreich und dabei exzellent geschrieben. Die Konzeption folgt einem interdisziplinären sowie auch interprofessionellen Ansatz und richtet sich damit gleichermaßen an Pflege- und Notfalldienstkräfte als auch Ärzt*innen unterschiedlicher Fachdisziplinen. Damit ist es den Herausgebern gelungen sowohl den umfangreichen Stoff einer breiten Leserschaft zugänglich zu machen als auch das Interesse zu wecken, diese innovative Struktur in die Klinik zu implementieren.

Zusammenfassend ist das Buch eine unverzichtbare Lektüre für alle diejenigen, die an der klinischen Patientenversorgung beteiligt sind.

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